The Canadian Veterinary Journal
La Revue vétérinaire canadienne

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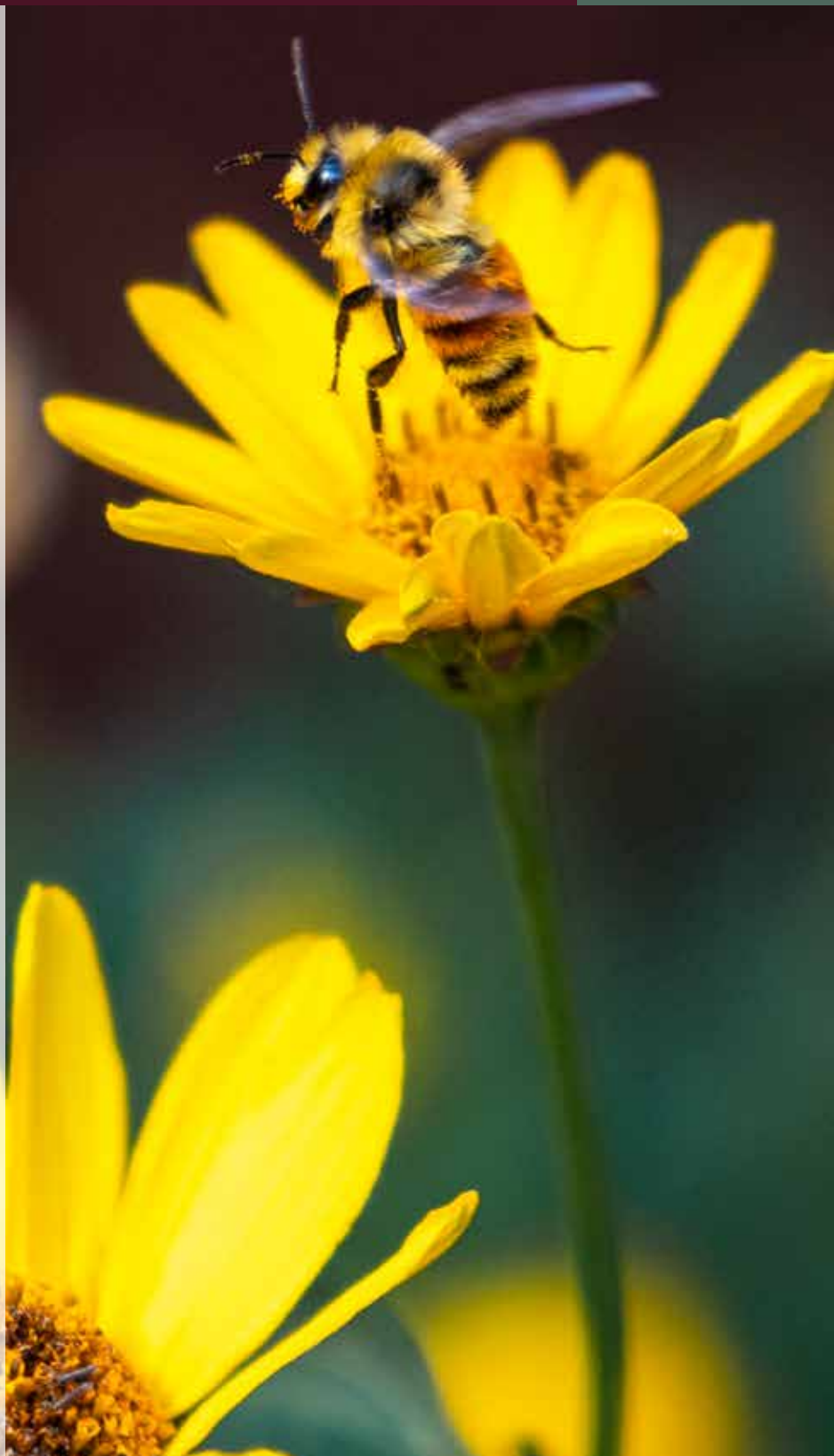

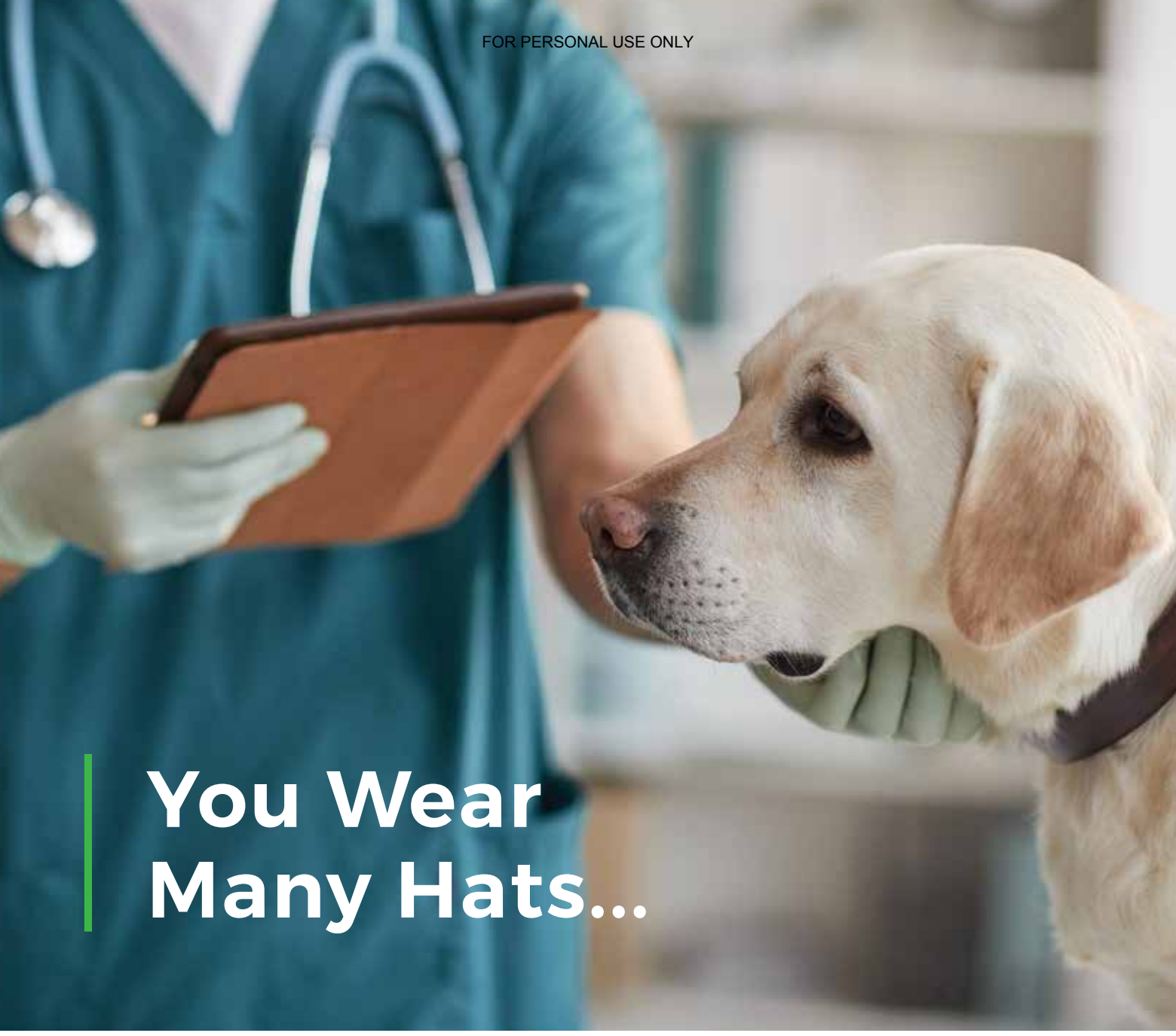

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1. Toutain CE et al Parasit Vect 2018 11 412 (v1.0) - vanillin and yeast flavor (p.2) The flavoring (yeast and vanillin) was specifically chosen for cats
2. Chappell K Parasites & Vectors 2021; 14:127 (v1.0) - 99.5 % success in administartion (p.2) Owners successfully administered 99 .5 % of tablets to their cats
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339 rue Booth Street
Ottawa, Ontario K1R 7K1
Telephone: 613-236-1162
Fax: 613-236-9681
Email: kgray@cvma-acmv.org
Website/Site Web: www.canadianveterinarians.net
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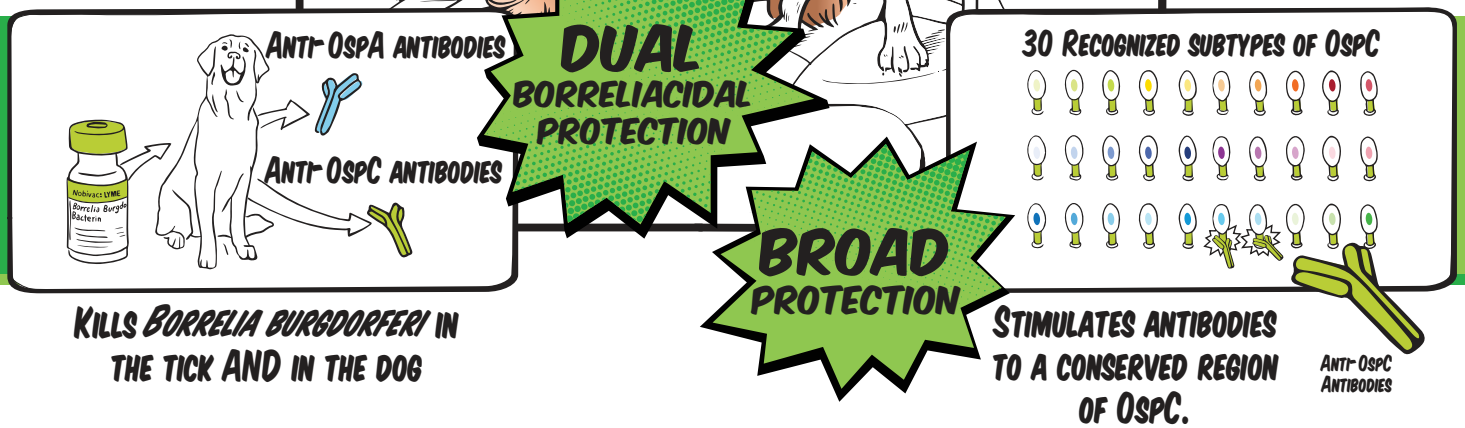

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1. LaFleur RL, *et al*. Bacterin that induces anti-OspA and anti-OspC borreliacidal antibodies provides a high level of protection against canine Lyme disease. *Clin Vaccine Immunol* 2009; 16(2):253-9.

President's Message
Le mot du président

Welcome, Doctors of Veterinary Medicine 2024! Are you ready?

Bienvenue aux nouveaux médecins vétérinaires de 2024! Êtes-vous prêts?

I've always been a bit taken with how fast the time goes. A year seems to hardly register, and for the Class of 2024, your incredible journey of the past 4 years is about to end! Convocation is just a few weeks away and your biggest journey yet will soon begin. Despite the inevitable anxiety that comes with this BIG change, I am beyond excited for you and confident that you CAN be a difference-maker in our profession, but it will require you to challenge yourself beyond anything you have ever seen or accomplished yet.

Are you ready to embark on this journey, ready to forge a path to become an "expert" in your field, and ready to become a respected member of both our profession and the communities you will serve?

Over the past year, I have tried to speak not only on subjects that are personally important to me, but also on those that I believe are crucial for the future success of our profession, for all of you and your colleagues yet to come. I encourage you to remember *why* you chose veterinary medicine, to consider how our choices in practice will *impact access to care* for the society we are intended to serve, to consider the *giving of oneself* each day for the benefit of others, and to *find a mentor* who can help you navigate the years ahead with much success.

Over the past few years, I have had countless conversations with colleagues from around the globe, the many thought-leaders in our profession, and have shared in deeply considered discussions about the value our profession collectively brings to society and the challenges and many opportunities that lie ahead. Common threads that emerge in our conversations have included advances in technology, impacts of a pandemic on education and career preparedness for students, One Health and our role in global health, the cost of veterinary medicine, and the serious issues surrounding access to care, to name only a few.

As you graduate, you should be optimistic and excited. You should have high expectations for yourself and our profession and expect to be rewarded (both financially and emotionally), much like many of us have been before you.

J'ai toujours été fasciné par la vitesse à laquelle le temps passe et les années se suivent, et pour la promotion de 2024, cela signifie que c'est bientôt la fin de votre formidable parcours des dernières années! La cérémonie de remise des diplômes aura lieu dans quelques semaines et vous allez bientôt entamer votre plus grand voyage. Malgré l'anxiété inévitable qui accompagne ce GRAND changement, je suis très enthousiaste pour vous et convaincu que vous POUVEZ faire une différence dans notre profession, mais vous devrez vous lancer des défis au-delà de tout ce que vous avez vu ou accompli jusqu'à présent.

Êtes-vous prêts à vous lancer dans cette aventure, à vous frayer un chemin pour devenir des « experts » dans votre domaine, et à devenir des membres respectés à la fois de notre profession et des communautés que vous servirez?

Au cours de la dernière année, j'ai essayé d'aborder des sujets qui sont non seulement importants pour moi, mais qui me semblent aussi essentiels pour la réussite future de notre profession, pour vous, la relève, et ceux qui viendront après vous. Je vous encourage à vous rappeler *pourquoi* vous avez choisi la médecine vétérinaire, à réfléchir à l'impact de nos choix en pratique sur l'*accès aux soins* pour les gens que nous servons, à *vous investir* chaque jour au bénéfice des autres, et à *trouver un mentor* qui vous aidera à franchir les années à venir avec succès.

J'ai eu ces dernières années d'innombrables conversations avec des collègues du monde entier et beaucoup de leaders d'opinion de notre profession, et j'ai participé à des discussions approfondies sur la valeur que notre profession apporte collectivement à la société et sur les défis et les nombreuses opportunités qui se présentent à nous. Certains points communs ressortent de ces échanges : les progrès technologiques, l'impact d'une pandémie sur l'éducation et la préparation à la carrière des étudiants, l'approche « Une seule santé » et notre rôle dans la santé à l'échelle mondiale, ainsi que le coût des soins vétérinaires et les problèmes liés à l'accès aux soins, pour n'en citer que quelques-uns.

Alors que vous vous apprêtez à recevoir votre diplôme, vous devriez être optimistes et enthousiastes. Vous devriez aussi avoir de grandes attentes envers vous-mêmes et notre profession, et

The first 5 years of your career will follow a steep trajectory of increasing knowledge, skills, comfort, and confidence. You will experience setbacks, changes in jobs, and unexpected losses — all of which are okay and completely normal. You are about to embark on another journey; convocation marks the end of one journey and the beginning of a much bigger one.

Malcolm Gladwell's *Outliers* spoke of the "10 000-hour rule," based on research that suggests it takes 10 000 hours of study and practice to become an expert in something. The truth is, it may take 20 000 hours or more, and it is not always a straight path where achieving these expert skills depends on only a few necessary steps. Becoming an "expert" takes knowledge, skill, and deliberate practice, and it requires a lot of time and effort to achieve. You will need to be self-aware in seeking feedback and refining your practice over time. Your education has prepared you well with a with a base of knowledge and skill that you will need to build on after you walk across the stage this month to continue your journey.

You are off to a great start. Your first 5 years in practice will be formative and a crucial part of your career success. There will be times when you will feel overwhelmed, underappreciated, and tired as you adjust to your chosen career path. Know that our profession is there to support you, and the reward of trust and respect from society will continue to both motivate you and protect your well-being. It will require you to step well outside of your comfort zone, and there will be times when you will need to allow yourself to be incredibly uncomfortable and to make mistakes.

I recall learning a lesson 20 years ago that continues to hold true. I had interviewed for my first job and was confident and ready (with mountains still to learn) and trying to maximize my salary in Year 1. I still remember the words from my first employer, Dr. Gary Morgan of West Prince Veterinary Services, as he sat across a kitchen table from me, which I think are as true today as they were then: "There is no such thing as a free lunch." Gary, a colleague and mentor, was correct. The truth was, no matter how ready and ambitious I was, I still had so incredibly much to learn. It is as true in 2024 as it was in 2004 that skill, ability, professional growth, and earned income all go hand in hand, and it takes time and effort to achieve. Be patient. Be devoted. Be successful.

Class of 2024, as you cross the stage in the next few weeks, hold your heads high with pride. You will continue to accomplish great things as you work towards becoming experts in your field! ■

Dr. Trevor Lawson

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espérer être récompensés (financièrement et émotionnellement) comme beaucoup d'entre nous l'ont été avant vous.

Les cinq premières années de votre carrière seront marquées par une progression rapide de vos connaissances, de vos compétences, de votre aisance et de votre confiance en vous. Vous connaîtrez des revers, des changements d'emploi et des pertes inattendues – tout cela est normal. Vous êtes sur le point d'entamer un nouveau voyage, et la collation des grades marque la fin d'un cheminement et le début d'un autre, beaucoup plus important.

Le livre *Outliers* de Malcolm Gladwell parle de la « règle des 10 000 heures », selon laquelle il faudrait 10 000 heures d'étude et de pratique pour devenir un expert dans un domaine. En réalité, cela peut prendre 20 000 heures ou plus, et la trajectoire vers l'expertise n'est pas toujours linéaire avec seulement quelques étapes à franchir. Pour être un « expert », il faut approfondir ses connaissances, développer ses compétences et s'exercer délibérément, ce qui demande beaucoup de temps et d'efforts. Vous devrez solliciter de la rétroaction et perfectionner votre pratique au fil du temps. Votre formation vous a permis d'acquérir une base de connaissances et de compétences sur laquelle vous pourrez vous appuyer une fois que vous aurez obtenu votre diplôme pour poursuivre votre parcours.

Vous partez du bon pied. Vos cinq premières années d'exercice seront formatrices et joueront un rôle crucial dans la réussite de votre carrière. Il y aura des moments où vous vous sentirez dépassés, sous-estimés et épuisés pendant cette période d'adaptation à la carrière que vous avez choisie. Sachez que vos collègues sont là pour vous soutenir et que la confiance et le respect de la société continueront à vous motiver et à protéger votre bien-être. Il vous faudra sortir de votre zone de confort et, à certains moments, vous devrez vous donner le droit d'être très inconfortables et de faire des erreurs.

J'ai appris il y a vingt ans une leçon qui est encore d'actualité. Je passais une entrevue pour mon premier emploi. J'étais confiant et prêt (tout en ayant encore beaucoup à apprendre) et je voulais maximiser mon salaire dès la première année. Je me souviens encore des paroles de celui qui était assis devant moi à une table de cuisine et allait devenir mon premier employeur, le Dr Gary Morgan de West Prince Veterinary Services, qui me semblent aussi justes aujourd'hui qu'elles l'étaient à l'époque : « Il n'y a rien de gratuit dans la vie ». Mon futur collègue et mentor avait raison. Même si j'étais prêt et ambitieux, j'avais encore énormément de choses à apprendre. Il est toujours vrai en 2024, comme c'était le cas en 2004, que les compétences, les aptitudes, la croissance professionnelle et le salaire vont de pair, et qu'il faut du temps et des efforts pour atteindre ses objectifs. Soyez patients et dévoués, et vous réussirez.

Futurs diplômés de 2024, lorsque vous monterez sur la scène dans les prochaines semaines, ayez la tête haute et soyez fiers. Vous accomplirez de grandes choses en vous employant à devenir des experts dans votre domaine! ■

Dr Trevor Lawson

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Veterinary Medical Ethics
Déontologie vétérinaire

Ethical question of the month — February 2024

While discussing vaccine protocols with the owner of a new puppy, you learn that the owner and head trainer at a local dog education center has also been discussing vaccines with dog owners. Specifically, they have advised that rabies vaccines can cause both behavioral and learning problems in dogs. The trainer also offers advice on how to create or alter a rabies vaccination certificate to allow a dog to cross the Canada-USA border and for kennel admission where vaccination is required. You attempt to educate the owner about the lack of validity of the vaccination concerns and the risks of falsifying documents for a designated reportable disease. You do not know the trainer personally, but you are aware this person is highly regarded in the community. **You are concerned about backlash, especially on social media, if you take action. What do you do under such circumstances?**

Question de déontologie du mois — Février 2024

Alors que vous discutez des protocoles de vaccination avec un client qui a un nouveau chiot, vous apprenez que la propriétaire et principale éducatrice canine d'un centre canin local parle aussi des vaccins avec ses clients. Elle aurait dit que les vaccins contre la rage peuvent causer des problèmes de comportement et d'apprentissage chez les chiens. Elle donne également des conseils sur la manière de créer ou de modifier un certificat de vaccination contre la rage pour qu'un chien puisse franchir la frontière entre le Canada et les États-Unis ou soit admis dans un chenil où la vaccination est exigée. Vous expliquez à votre client que les préoccupations relatives à la vaccination ne sont pas fondées et que la falsification de documents relatifs à une maladie à déclaration obligatoire comporte des risques. Vous ne connaissez pas l'éducatrice canine personnellement, mais vous savez que cette personne est très appréciée dans la communauté. **Vous craignez des réactions négatives, notamment sur les médias sociaux, si vous intervenez. Que faites-vous dans de telles circonstances?**

Ethicists' commentary on confronting misinformation in the animal-care community

This case raises the issue of how a veterinarian should handle strongly held but controversial and scientifically unfounded views about animal health among clients and their non-veterinary advisors.

The questioner (who we assume to be the veterinarian in the case) has discussed vaccine protocols with the owner of a new puppy, but the client is resistant to the vet's advice regarding the rabies vaccine. The client has been advised by the head trainer at a local dog education facility that the vaccine should be avoided because it can cause behavioral and learning problems in dogs. Without this vaccination, the dog cannot cross the Canada-USA border or be kenneled while the owner is away, but the trainer has also offered advice on how to overcome these problems by falsifying vaccination certificates.

The veterinarian has tried to educate the puppy's owner both about the absence of scientific evidence for the alleged side effects of the vaccine and about the risks of falsifying vaccination documents. What more can and should the veterinarian do, specifically in relation to the trainer, who seems to be highly regarded?

One way forward would be for the veterinarian to directly contact and try to convince the trainer to change how they advise owners, though — given what is known about vaccine opposition — this may not be fruitful. The veterinarian could also report the trainer to the relevant authorities. A client of a "dog education center" might reasonably expect accurate and sensible advice on the care of their dog. Part of this professional obligation is to avoid straying outside one's area of professional competence. A dog trainer providing professional advice on vaccines is thus committing a breach of professional ethics. If the trainer or the training center is a member of the Canadian Association of Professional Dog Trainers or a similar organization, it may be possible to submit a complaint to that body. In addition, a trainer who provides professional advice on vaccine protocols may find themselves at odds with provincial legislation regarding unauthorized veterinary practice. On this basis, the veterinarian could also submit a complaint to their own regulatory authority, which in turn may decide to take the trainer to court.

We acknowledge that the veterinarian is concerned about potential blowback associated with approaching or reporting the trainer. Whistleblowers can suffer real harm from taking such action. A reasonable worry in the current case is a social media "storm" from the trainer's loyal clientele that could lead to

reputational and economic damage for the veterinarian, though this risk may be mitigated if the veterinarian could get other colleagues involved.

Given these risks, we cannot provide firm ethical guidance regarding whether the veterinarian should actually challenge the trainer. At a minimum, the veterinarian fulfills their key duty by not shying away from confronting the client about the lack of evidence for the factual claims made and by pointing to the risks of falsifying vaccination documents. To go further than this is laudable, but not strictly required.

Drs. Clare Palmer, Peter Sandøe, and Dan Weary

Ethical question of the month — May 2024

Another general practitioner in your area has recently begun to accept referrals for specialized orthopedic procedures. One of your clients has a dog that you diagnosed with a cruciate tear, and you referred them to a Board-certified surgeon. You later find out that the client has self-referred to a general practitioner and has requested you send that practitioner the dog's medical records. After you send the records, the receiving veterinarian requests that you send an additional referral from your practice. **What is the implication of sending this referral? Does it imply a recommendation and a trusting relationship between your veterinary clinic and the receiving veterinarian, or is it simply related to the transfer of patient files?**

Question de déontologie du mois — Mai 2024

Un autre vétérinaire généraliste de votre région a récemment commencé à accepter des cas référés pour des interventions orthopédiques habituellement effectuées par des spécialistes. Le chien d'un de vos clients a une rupture du ligament croisé et vous l'avez orienté vers un spécialiste en chirurgie. Vous apprenez par la suite que le client a plutôt décidé de consulter un vétérinaire généraliste, à qui il vous demande de transmettre le dossier médical de son chien. Vous envoyez le dossier, et le vétérinaire destinataire vous demande ensuite de lui faire parvenir une requête issue par votre clinique. **Qu'implique l'envoi de cette requête? La requête laisse-t-elle supposer une recommandation et l'existence d'une relation de confiance entre votre clinique et ce vétérinaire, ou concerne-t-elle seulement la transmission du dossier du patient?**

Responses to the case presented are welcome. Please limit your reply to approximately 50 words and forward along with your name and address to: **Ethical Choices, c/o Canadian Veterinary Medical Association, Attn: Journals Department, 339 Booth Street, Ottawa, Ontario K1R 7K1; email (bettinadvm@gmail.com). A longer response may appear as a Letter to the Editor.**

Suggested ethical questions of the month are also welcome! All ethical questions or scenarios in the ethics column are based on actual events, which are changed, including names, locations, species, *etc.,* to protect the confidentiality of the parties involved.

Les réponses au cas présenté sont les bienvenues. Veuillez limiter votre réponse à environ 50 mots et nous la faire parvenir avec vos nom et adresse **par la poste (Choix déontologiques, Association canadienne des médecins vétérinaires, À l'attention de : Journals Department, 339 rue Booth, Ottawa, Ontario, K1R 7K1) ou par courriel (bettinadvm@gmail.com). Les réponses plus longues pourraient être publiées dans le courrier des lecteurs.**

Les propositions de questions déontologiques sont toujours bienvenues! Toutes les questions et situations présentées dans cette chronique s'inspirent d'événements réels dont nous modifions certains éléments, comme les noms, les endroits ou les espèces, pour protéger l'anonymat des personnes en cause.

Letters to the Editor Courrier des lecteurs

N. Ole Nielsen's Commentary: Imagining veterinary medicine and education in 2040 — A comment

Dear Editors,

Dr. Nielsen's commentary on veterinary specialization (1) raises some oft- and some less-discussed issues.

The often-discussed issue of changing career paths within the veterinary profession is NOT addressed. Does the dairy practitioner who moves to the city for one of many reasons (changing farm practices, quality of life, family, or other) require retraining? They may choose federal abattoir meat safety or research or small-animal practice. However, their student training prepared them for none of these specialties. Do they now cease to earn income for 2 y in order to undergo 2 y of elective courses and clinical externship training? Or, do we allow veterinarians, unlike our medical colleagues, to change specialties without additional education and training?

Less often discussed, and to my knowledge, not discussed, is the issue of delegating our "technical" client educational sessions and straightforward techniques to assistants. Currently, successfully delegated duties include pregnancy testing and artificial insemination in cattle, and there are nurse practitioners in human medicine. We have Animal Health Technologists (AHTs), but how much is really delegated to them?

The AHTs currently do the sedation and general anesthesia of our patients — arguably the most dangerous procedure for which we are responsible — with a veterinarian being legally required to be in the building at the time. However, how much diabetic education and monitoring (as in human medicine), incision suturing, cat castrating, and so on, do AHTs actually do? The medical profession has been very reluctant to encourage and empower nurse practitioners for fear of loss of control. Is the same true of our veterinary profession?

There appears to be a shortage of AHTs across Canada. Is this because they are inadequately paid or insufficiently appreciated or have better job opportunities? Or is it because insufficient numbers are being trained?

As for foreign graduates gaining full qualifications to practice in Canada, where is the discussion? Which countries? How much monitoring? What student programs are open to them? How much financial assistance is available? Which examinations? What is the success rate, what is the client complaint rate, and what is the retention rate 5 y after registration? And how do these compare with Canadian veterinary graduates?

What about part-time education within veterinary schools? "Part-time" being evening or weekend courses, online courses, or intensive short courses? Is completion adequate or are examinations required for additional accreditation?

Details matter — ask any negotiator.

Dr. Nielsen cites lovely examples of students entering fields of ecology, One Health, small-animal practice, dairy practice, and research — in a company, a corporate veterinary practice, a corporate dairy practice, and a university research institute. Is he, by omission, suggesting that small-business veterinary practice is obsolete, either in the countryside or within metropolitan centers?

Where and by whom are these issues being discussed and decided? Where and when are they being reported in *The Canadian Veterinary Journal?*

Submitted by Lea Stogdale, DVM, DACVIM, Aesops Veterinary Care, Winnipeg, Manitoba.

Response from N. Ole Nielsen

The option of changing career paths in veterinary medicine has been used as a compelling reason for making the DVM a generalist degree that enabled the holder to be licensed in various fields of practice upon graduation. Over time, the enormous changes that have occurred in society have made continuation of this policy and its inflexibility a root cause for limiting the profession's potential to better serve society and its own members. The present problems of the profession are arguably an unnecessary and unacceptably high price to pay for this privilege when it is possible to opt for the flexibility of a policy of designated licensure. The advantages of a generalist medical education would be retained, and strengthened by a rigorous and mandatory curriculum focused on comparative medicine that all DVM students would be required to master, along with clinical training in the field in which they wish to be licensed to practice. Information technology, sophisticated CE programs, and emergence of private practices with clinical competence comparable to university veterinary teaching hospitals would provide the resources that would make a licensed career change a reasonable process. Furthermore, the pending huge impact of

AI on education and practice will surely make the process even more effective.

Dr. Stogdale raises a number of other issues that I agree deserve more attention from the profession. Perhaps the new McEachran Institute might be a means to address some of them.

The hypothetical practice scenarios I described were pruned from 7 to 4, upon the advice of the Editor, to address article word limits. The original manuscript also included "community" (general), "wildlife," and "equine" practices.

A more detailed description of the rationale behind my views regarding veterinary education and practice is chronicled on the McEachran Institute website (2).

Dr. Nielsen, CM, is a retired veterinary pathologist and former dean of both the Western College of Veterinary Medicine and the Ontario Veterinary College.

Response from *The CVJ*'s Co-Editors-in-Chief

Dr. Stogdale issues a welcome challenge to *The CVJ*. Your Editors encourage and will actively facilitate more discussion, commentary, and professional dialogue about contemporary issues and visionary perspectives on topics in veterinary education, training/retraining, practice, *etc.* In our view, *The CVJ* is a primary voice and vehicle for information-sharing for Canadian veterinarians. We already provide opportunities to share information on various topics in regular feature columns, including Veterinary Wellness, Veterinary Medical Ethics, and One Health. It is noteworthy that the Food Animal Matters column in the February issue includes a discussion on roles of registered veterinary technicians in food animal practice. Regardless, there is no doubt that we could do more! Therefore, we welcome and encourage submission of relevant articles, opinion pieces, letters to the Editors, *etc.* Our Instructions for Authors (3) describe the kinds of articles and formatting requirements. If you need assistance before, during, or after submission, please reach out to us; we are always willing to provide assistance upon request.

John Kastelic, DVM, PhD, Dipl. ACT (jpkastel@ucalgary.ca)
Tim Ogilvie, DVM, MSc, LLD, DACVIM (ogilvie@upei.ca)

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2. McEachran Institute [homepage on the Internet]. Available from: https://www.mceachraninstitute.ca Last accessed March 8, 2024.
3. Canadian Veterinary Medical Association [Internet]. The Canadian Veterinary Journal — Instructions for Authors [updated 2024]. Available from: https://www.canadianveterinarians.net/journals-and-classified-ads/the-canadian-veterinary-journal/instructions-for-authors Last accessed March 8, 2024.

Quiz Corner
Test éclair

1. A 4-day-old colt requires veterinary attention for fever and a swollen umbilicus. The umbilicus has been ligated with umbilical tape (Figure 1, below).

Transabdominal ultrasound imaging of the umbilical remnants reveals an enlarged umbilical stump and enlarged right umbilical artery. Physical examination of the foal also reveals moderate warm effusion of the right stifle and notable lameness in the right hind limb.

Figure 1.
Image courtesy of Nora Grenager, VMD, DACVIM.
Image de Nora Grenager, D.M.V., DACVIM.

Which one of the following approaches should be included in an appropriate management plan?

A. Remove umbilical remnant; aspirate and flush stifle under general anesthesia; administer systemic antimicrobials.
B. Administer oral antibiotics; dip umbilicus in chlorhexidine q8h; apply topical diclofenac on stifle.
C. Apply rubber band on umbilical stump; administer enteral NSAIDs q12h; give tetanus antitoxin; radiograph leg for fracture.
D. Obtain magnetic resonance imaging of right hind limb; gently manually reduce umbilical hernia q8h.
E. Cauterize umbilical stump with silver nitrate; administer intra-articular amikacin into affected joint.

1. Un poulain de 4 jours a besoin de soins vétérinaires car il fait de la fièvre et présente une enflure du moignon ombilical. Le cordon ombilical a été ligaturé avec un ruban ombilical (figure 1).

L'échographie transabdominale de la région ombilicale révèle une distension du moignon ombilical et de l'artère ombilicale droite. L'examen physique du poulain permet également de constater un épanchement chaud modéré dans le grasset droit et une boiterie marquée du membre postérieur droit.

Laquelle des approches suivantes devrait faire partie d'un plan de prise en charge appropriée?

A. Retirer le moignon ombilical; ponctionner et rincer le grasset sous anesthésie générale; administrer des antimicrobiens systémiques.
B. Administrer des antibiotiques par voie orale; tremper le moignon ombilical dans de la chlorhexidine toutes les 8 heures; appliquer du diclofénac topique sur le grasset.
C. Placer un élastique sur le moignon ombilical; administrer des AINS par voie entérale toutes les 12 heures; administrer l'antitoxine tétanique; faire une radiographie de la patte pour vérifier qu'il n'y a pas de fracture.
D. Réaliser une imagerie par résonance magnétique du membre postérieur droit; réduire manuellement et délicatement la hernie ombilicale toutes les 8 heures.
E. Cautériser le moignon ombilical avec du nitrate d'argent; administrer de l'amikacine intra-articulaire dans l'articulation affectée.

2. A 2-year-old spayed female standard poodle dog is examined because of an acute onset of vomiting, diarrhea, and collapse.

On physical examination, the dog is weak, with pale mucous membranes and poor pulse quality. Results from the physical examination are shown below.

2. Une femelle caniche standard stérilisée de 2 ans est examinée en raison de vomissements, de diarrhée et d'un collapsus.

À l'examen physique, la chienne est faible, ses muqueuses sont pâles et son pouls est de mauvaise qualité. Les résultats de l'examen physique sont résumés ci-dessous.

Parameter/Paramètre	Value/Résultat	Reference/Valeurs de référence
Temperature/Température	37.2°C (99°F)	37.5–39.2°C (99.5–102.5°F)
Heart rate/Fréquence cardiaque	56 bpm	60–120 bpm
Respiratory rate/Fréquence respiratoire	25 brpm/rpm	15–34 brpm/rpm

Results from hematology and serum biochemistry testing are shown below in the tables, and an electrocardiogram is shown below in Figure 1.

Les résultats de l'hématologie et de la biochimie sont présentés dans les tableaux ci-dessous, et l'électrocardiogramme est illustré à la figure 1.

Canine hematology/Hématologie canine

Test/Paramètre	Value/Résultats	Reference/Valeurs de référence
RBC/Érythrocytes	4.5	5.39–8.70 M/μL
Hematocrit/Hématocrite	32	38.3–56.5%
Hemoglobin/Hémoglobine	11.8	13.4–20.7 g/dL
MCV/VGM	71	59–76 fL
MCH/TCMH	26.1	21.9–26.1 pg
MCHC/CCMH	36.8	32.6–39.2 g/dL
Reticulocytes/Réticulocytes	34	10–110 K/μL
WBC/Leucocytes	10.9	4.9–17.6 K/μL
Neutrophils/Neutrophiles	4.2	2.94–12.67 K/μL
Lymphocytes	4.2	0.4–2.9 K/μL
Monocytes	0.4	0.13–1.15 K/μL
Eosinophils/Éosinophiles	2.1	0.07–1.49 K/μL
Platelets/Plaquettes	325	143–448 K/μL

Canine blood chemistry/Biochimie canine

Test/Paramètre	Value/Résultat	Reference/Valeurs de référence
Glucose	60	63–114 mg/dL
Creatinine/Créatinine	2.5	0.5–1.5 mg/dL
BUN/Urée	75	9–31 mg/dL
Phosphorus/Phosphore	7.0	2.5–6.1 mg/dL
Calcium	11.9	8.4–11.8 mg/dL
Sodium	132	142–152 mmol/L
Potassium	8.5	4.0–5.4 mmol/L
Chloride/Chlorure	95	108–119 mmol/L
TCO_2 (bicarbonate)	12	17–24 mmol/L
Total protein/Protéines totales	6.0	5.5–7.5 g/dL
Albumin/Albumine	2.8	2.7–3.9 g/dL
Globulin/Globulines	3.2	2.4–4.0 g/dL
ALT	100	18–121 U/L
AST	13	13–55 U/L
ALP	112	5–160 U/L
GGT	6	0–13 U/L
Bilirubin — total/Bilirubine totale	0.1	0.0–0.3 mg/dL
Cholesterol/Cholestérol	110	131–345 mg/dL
Amylase	1000	337–1469 U/L
Creatine kinase/Créatine kinase	500	10–200 U/L
Lipase	205	0–250 U/L

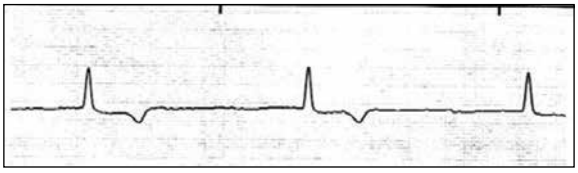

Figure 1.
Image courtesy of Terri DeFrancesco, DVM, DACVIM, DACVECC.
Image de Terri DeFrancesco, D.M.V., DACVIM, DACVECC.

What is the rhythm disturbance and what is the most likely cause of this abnormality?

A. Sick sinus syndrome; hypoglycemia
B. Second-degree AV block; hyponatremia
C. Third-degree AV block; hypovolemia
D. Ventricular premature contraction; hypercalcemia
E. Atrial standstill; hyperkalemia

De quel trouble du rythme cardiaque s'agit-il et quelle est la cause la plus probable de cette anomalie?

A. Dysfonction du nœud sinusal; hypoglycémie
B. Bloc AV du deuxième degré; hyponatrémie
C. Bloc AV du troisième degré; hypovolémie
D. Contraction ventriculaire prématurée; hypercalcémie
E. Paralysie auriculaire; hyperkaliémie

(See p. 507 for answers./Voir les réponses à la page 507.)

The questions and answers are provided by Zuku Review, online veterinary test prep.

Les questions et les réponses sont gracieusement fournies par le site de préparation aux examens vétérinaires Zuku Review.

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Brief Communication Communication brève

Molecular profile and epidemiological traits of *Streptococcus suis* isolated from diseased pigs in western Canada reveal multiple-serotype infection: Implications for disease control

Matheus de O Costa, Ruwini Gamage, Jette Christensen

Abstract

Objective

Streptococcus suis is a major agent of disease in modern swine operations, linked to increased mortality, treatment costs, and secondary infections. Although it is ubiquitous in swine, only a fraction of pigs develop clinical disease. The goals of this study were to profile isolates obtained from diseased pigs in western Canada and to investigate potential associations with disease severity.

Procedure

Isolates of *S. suis* (n = 128) from 75 diagnostic submission and 63 premises were paired with epidemiological surveys completed by submitting practitioners (n = 22). Whole-genome sequencing was used to type isolates.

Results

The most prevalent serotypes identified were 1/2 (7.8%, 10/128), 2 (9.3%, 12/128), 3 (9.3%, 12/128), and 7 (7.8%, 10/128); and sequence types 28 (17%, 23/128) and 839 (14%, 19/128). There was no association between serotype or sequence type and organ source or barn location. Approximately 74% (14/19) of the premises had diseased animals colonized by >1 *S. suis* serotype, but only 1 pig was simultaneously infected with multiple serotypes and sequence types. Serotype distribution from diseased pigs in western Canada differed from that of those in other geographic regions.

Conclusion

Infection of diseased pigs by multiple serotypes should be considered when disease control strategies are implemented. No association between *S. suis* type and isolation organ was identified.

Résumé

Le profil moléculaire et les caractéristiques épidémiologiques de *Streptococcus suis* isolés de porcs malades dans l'ouest du Canada révèlent une infection à sérotypes multiples : implications pour la maitrise de la maladie

Objectif

Streptococcus suis est un agent pathogène majeur dans les exploitations porcines modernes, lié à une mortalité accrue, aux coûts de traitement et aux infections secondaires. Bien qu'elle soit omniprésente chez le porc, seule une fraction des porcs développe une maladie clinique. Les objectifs de cette étude étaient de dresser le profil des isolats obtenus à partir de porcs malades dans l'ouest du Canada et d'étudier les associations potentielles avec la gravité de la maladie.

Procédure

Des isolats de *S. suis* (n = 128) provenant de 75 soumissions pour diagnostic et de 63 sites ont été associés à des enquêtes épidémiologiques réalisées auprès des praticiens soumettant les échantillons (n = 22). Le séquençage du génome entier a été utilisé pour typer les isolats.

Large Animal Clinical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan S7N 5B4 (Costa, Gamage); Population Health Sciences, Faculty of Veterinary Medicine, Utrecht University, Yalelaan 7, Utrecht, The Netherlands (Costa); Canada West Swine Health Intelligence Network (CWSHIN) Inc., Winnipeg, Manitoba (Christensen); Epidemiologic Surveillance and Analysis Consulting (EpiSAC), Charlottetown, Prince Edward Island (Christensen).

Address all correspondence to Matheus Costa; email: matheus.costa@usask.ca

Unpublished supplementary material (Figures S1, S2, S3, S4) is available online from: www.canadianveterinarians.net

Résultats

Les sérotypes les plus répandus identifiés étaient 1/2 (7,8 %, 10/128), 2 (9,3 %, 12/128), 3 (9,3 %, 12/128) et 7 (7,8 %, 10/128); et les types de séquence 28 (17 %, 23/128) et 839 (14 %, 19/128). Il n'y avait aucune association entre le sérotype ou le type de séquence et la source d'organes ou l'emplacement de la ferme. Environ 74 % (14/19) des exploitations abritaient des animaux malades colonisés par > 1 sérotype de *S. suis*, mais 1 seul porc était infecté simultanément par plusieurs sérotypes et types de séquences. La répartition des sérotypes chez les porcs malades de l'ouest du Canada différait de celle des porcs d'autres régions géographiques.

Conclusion

L'infection des porcs malades par plusieurs sérotypes doit être envisagée lors de la mise en œuvre de stratégies de maitrise de la maladie. Aucune association entre le type de *S. suis* et l'organe d'isolement n'a été identifiée.

(Traduit par Dr Serge Messier)

Can Vet J 2024;65:429–436

Modern pork production requires refined management practices and veterinary oversight to ensure herd health and performance. *Streptococcus suis* is, from a health perspective, one of the most challenging agents the industry currently faces. This Gram-positive bacterium is associated with increased post-weaning mortality in commercial operations, often presenting as meningitis, arthritis, endocarditis, or polyserositis in pigs. It is a potential zoonotic agent leading to human meningitis and septicemia (1,2). Being ubiquitous in commercial herds worldwide and with an estimated prevalence of virtually 100%, *S. suis* inflicts substantial economic losses due to increased mortality, treatment costs, and secondary infections (3).

Serotyping has been suggested as a practical way of inferring virulence and isolate significance, given the opportunistic nature of *S. suis* in pigs (4). Therefore, it continues to be part of the veterinary and diagnostic toolkits. Serotype distributions from diseased and healthy pigs have been reported from several countries, including hog-dense areas in eastern Canada, Germany, Italy, the United States, China, and Brazil (5–10). Unsurprisingly, different geographical locations have reported different serotype distributions.

Classically, serotyping of *S. suis* has been done using slide co-agglutination (11). The advent and cost-effectiveness of molecular biological techniques led to PCR and *in silico* methods evolving as alternatives to the co-agglutination tests for serotyping (12,13). An improved PCR assay based on mismatch amplification mutation assay was recently described. This assay is an improvement from previous efforts, as it can discriminate among all known serotypes (including 1 and 14 or 2 and 1/2) (14). In parallel, *in silico* serotyping (based on whole-genome sequencing) performed similarly to the co-agglutination and PCR tests. This approach can also detect new serotypes and provides further information about isolates, including multilocus sequence type (MLST), and presence of virulence-associated genes and antimicrobial resistance genes (5,15). A visual summary of the serotyping techniques available is presented in Figure 1.

Given the economic importance and considerable control challenges associated with *S. suis,* the objectives of this study were to type *S. suis* isolated from diseased pigs in western Canada and to investigate isolate profile relationships with disease severity in affected farms.

We selected for inclusion in the study diagnostic submissions from March to October 2021, from swine for which clinical signs were suggestive of *S. suis*-associated diseases (sudden death, meningitis, arthritis, endocarditis, polyserositis) and *S. suis* was isolated. Next, each submitting veterinarian was invited to participate in an epidemiological survey and, if a completed survey was received, the corresponding isolate(s) was (were) further processed.

Diagnostic submission samples were cultured aerobically on Tryptic Soy Agar (Thermo Fisher Scientific, Waltham, Massachusetts, USA) with 5% bovine blood at 37°C for 24 to 48 h. Isolated alpha-hemolytic, round, and translucent colonies were identified using matrix-assisted laser desorption/ionization time-of-flight mass spectrometry with a threshold value ≥ 2.0 (MALDI-TOF, Biotyper microFlex LT/SH system; Bruker Daltonik GmbH, Bremen, Germany). Bacterial genomic DNA was extracted using a MagAttract HMW DNA Kit (Qiagen, Toronto, Ontario) following the manufacturer's instructions. Extracted DNA was prepared for sequencing using the Oxford Nanopore SQK-PBK004 library prep kit (Oxford Nanopore, Oxford, England). After end-repair, barcoding ligation was performed using ONT ligation kit SQK-LSK109 and native barcoding kit EXP-NBD104 as per the manufacturer's instructions. Barcoded DNA was pooled into libraries and 200 ng of each DNA library was loaded onto a FLO-MIN106 flow cell and sequenced using an ONT GridION device (Oxford Nanopore). Software (ONT MinKnow) was used to collect raw electronic signal data, which were base-called into fast5 and fastq formats using Metrichor (both from Oxford Nanopore). High-quality reads were then processed, assembled, and annotated using the comprehensive genomic analysis tool in Pathosystem Resource Integration Center (PATRIC) (16). Assembled genomes were serotyped and multilocus sequenced typed (MLST) *in silico* using a published pipeline (15). The MLST typing database used was updated on March 19, 2021. Only genomes with > 150× coverage and 90% completeness (when compared to the reference strain *S. suis* SS2-1) were included in the analyses.

Minimum inhibitory concentrations for ampicillin, penicillin, ceftiofur, and tetracycline were determined using micro-broth dilution as per Clinical & Laboratory Standards Institute (CLSI)-approved standards VET01S ED5:2020,

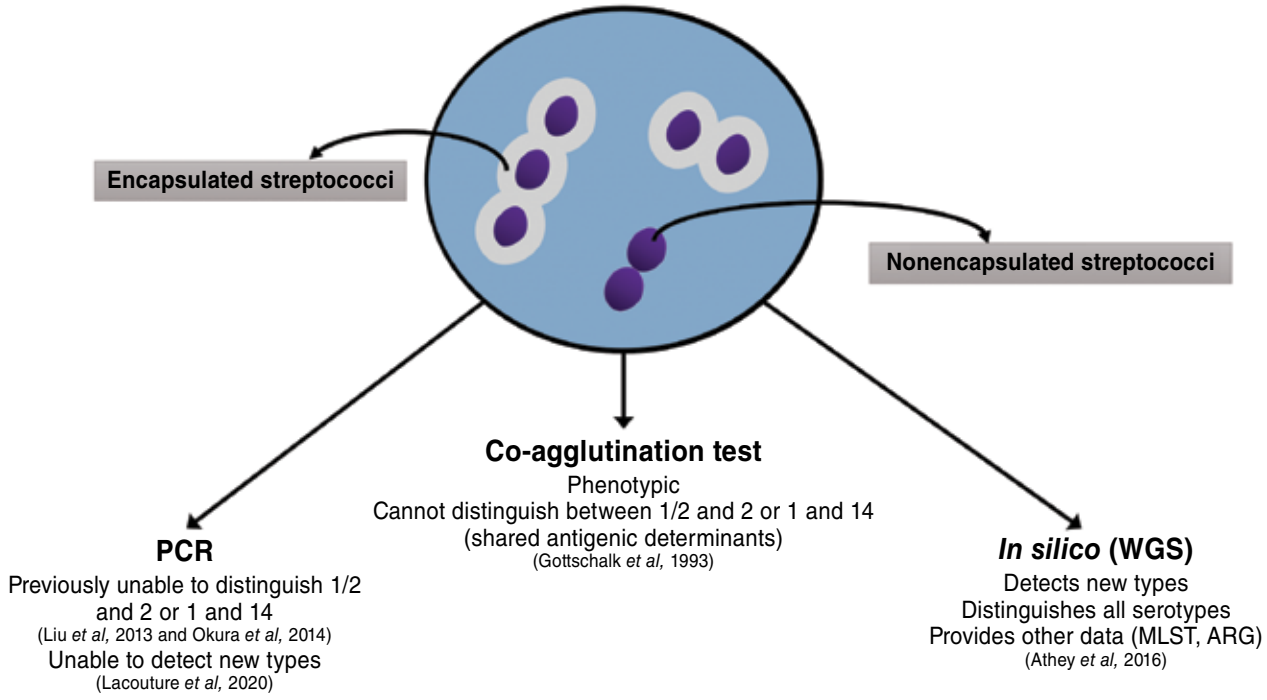

Figure 1. *Streptococcus suis* serotyping strategies used and their limitations.
ARG – Antimicrobial resistance genes; MLST – Multilocus sequence type; WGS – Whole-genome sequencing.

M100-ED31:2021. Compounds were tested across a range of 2-fold step dilutions in Mueller Hinton broth supplemented with lysed horse blood in a Sensititre auto-inoculator (Thermo Fisher Scientific). Plates were incubated aerobically for 18 to 24 h at 37°C. Controls included CLSI-recommended reference strains *Enterococcus faecalis* (ATCC 29212) and *Streptococcus pneumoniae* (ATCC 49619). Isolates were classified as resistant, susceptible, or intermediate, according to the CLSI-suggested breakpoints (17).

Epidemiological data were obtained through a voluntary survey completed by each veterinarian submitting samples to the diagnostic laboratory. This included premises (defined as a commercial operation located in a defined geographical location, with or without multiple buildings/rooms, but a single site), identification number (PID), date of submission (date), province (Alberta, Manitoba, Saskatchewan), and clinical score. The latter was recorded by the submitting practitioner according to this scale: Score 1 = normal morbidity and normal mortality, 2 = increased morbidity and normal mortality, 3 = normal morbidity and increased mortality, and 4 = increased morbidity and increased mortality. For this study, a "case" was defined as isolates from the same PID within 21 d, with 1 exception where materials were submitted on several dates from pigs with different clinical signs: MB3 (3 cases: March, April, and May). The PID was used in the analysis, but for privacy reasons it was converted to anonymous premises keys (AB1 to AB6, MB1 to MB48, SK1 to SK9). Data were grouped according to the organ source of the isolates. "Noninvasive" referred to those recovered from lung or nasal tissues, whereas "invasive" described isolates obtained from any other tissue samples (abscess, brain, heart, joint, liver, lymph node, pooled, spleen, thoracic fluid) (see Figure S1, available online from: www.canadianveterinarians.net). Cases where isolates were obtained from both tissue categories were referred to as "both."

The relationships between clinical score and the premises-level factors [location, number of cases per premises, whether $>$ 1 case was submitted (yes/no), whether $>$ 1 isolate was detected and included (yes/no), number of isolates, and number of isolates resistant or susceptible to antimicrobials between production types (conventional *versus* antibiotic-free)] were tested in contingency tables, using X^2 or Fisher exact tests (univariate analyses). The clinical scores may have been biased, given that these were subjective interpretations by the submitting practitioners. To assess this potential bias, we evaluated the effect of consistently high-scoring practitioners. Analyses were done using SAS 9.4 (SAS Institute, Cary, North Carolina, USA).

A total of 128 isolates, 75 cases, and 63 premises were included in this study. Completed surveys were received from 22 practitioners. Average genome coverage was 203×, completeness was 98.74%, GC content was 40.37%, and genome size was 2.3 Mbp.

Isolates of *S. suis* were obtained from diseased pigs raised in Alberta (7%, 9/128), Manitoba (74.3%, 95/128) and Saskatchewan (18.3%, 24/128). Samples were mostly from early nursery (42.4%, 53/128) and late nursery (20.8%, 26/128), followed by grower/feeder (13.6%, 17/128), finisher (12%, 15/128), suckling (7.2%, 9/128), gilts (2.4%, 3/128), and sows (1.6% 2/128). A total of 76% (96/128) of the isolates came from pigs reared under conventional production strategies, 20% (25/128) were from pigs raised without antibiotics, and 3.2% (4/128) were from pigs in a genetic multiplier. Approximately 1/3 of the isolates were cultured from lung

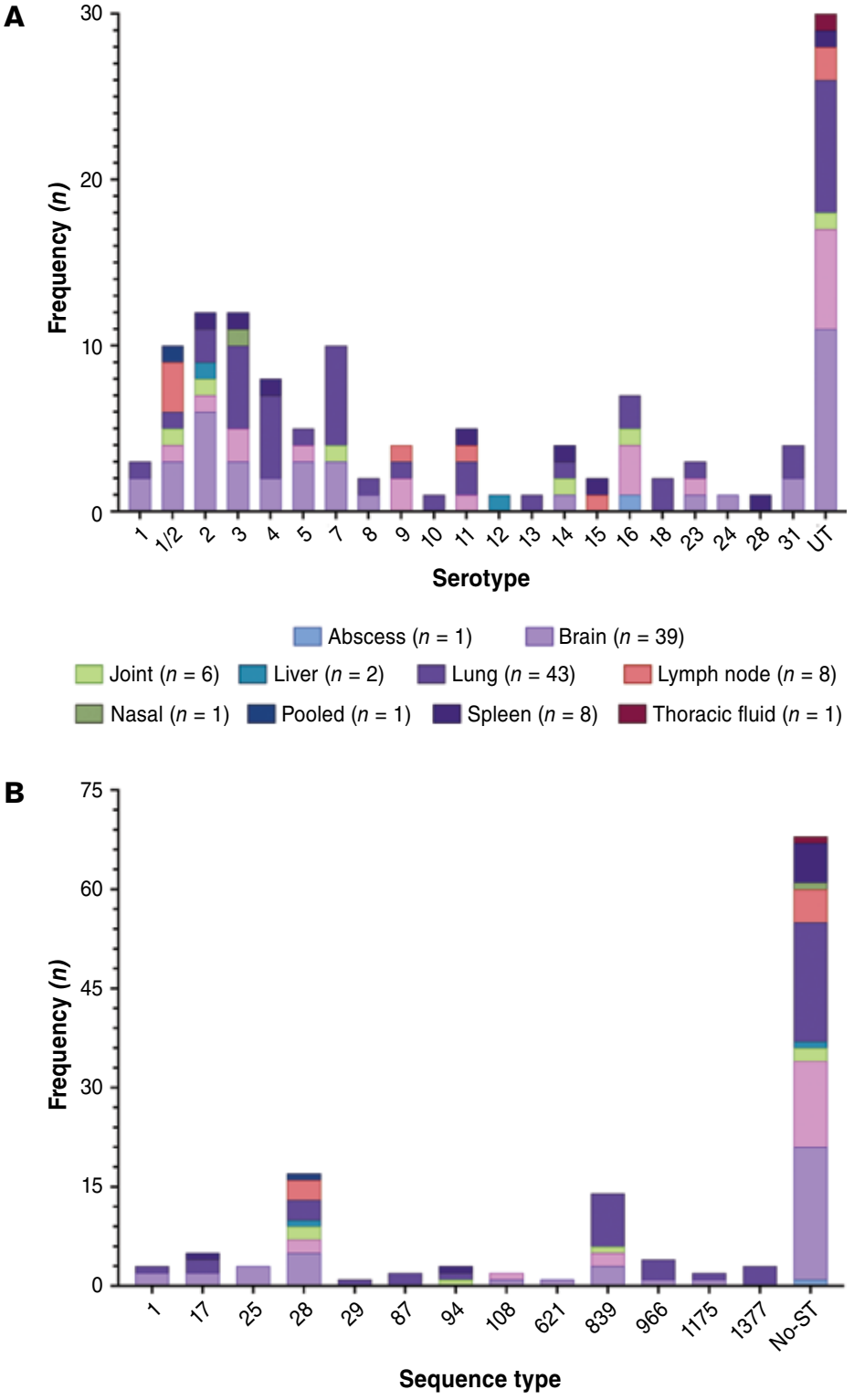

Figure 2. *Streptococcus suis* isolate ($n = 128$) distribution by organ source, according to serotype (A) and sequence type as per multilocus sequence typing (B).

No-ST – No matching sequence type found in the public multilocus sequence type database (PubMLST.org); UT – Isolates untypable by the methods used in this study.

samples (33.5%, 43/128), followed by brain (30.5%, 39/128) and heart (14.1%, 18/128) samples. Isolates from nasal samples were 1.5% of the total ($n = 2$). The pooled sample reported resulted from a mix of internal organs (liver, kidney, and spleen). A summary of isolate distribution by serotype, sequence type, and organ of origin is presented in Figure 2. The most prevalent serotypes were 1/2 (7.8%, 10/128), 2 (9.3%, 12/128), 3 (9.3%, 12/128), and 7 (7.8%, 10/128), and sequence types

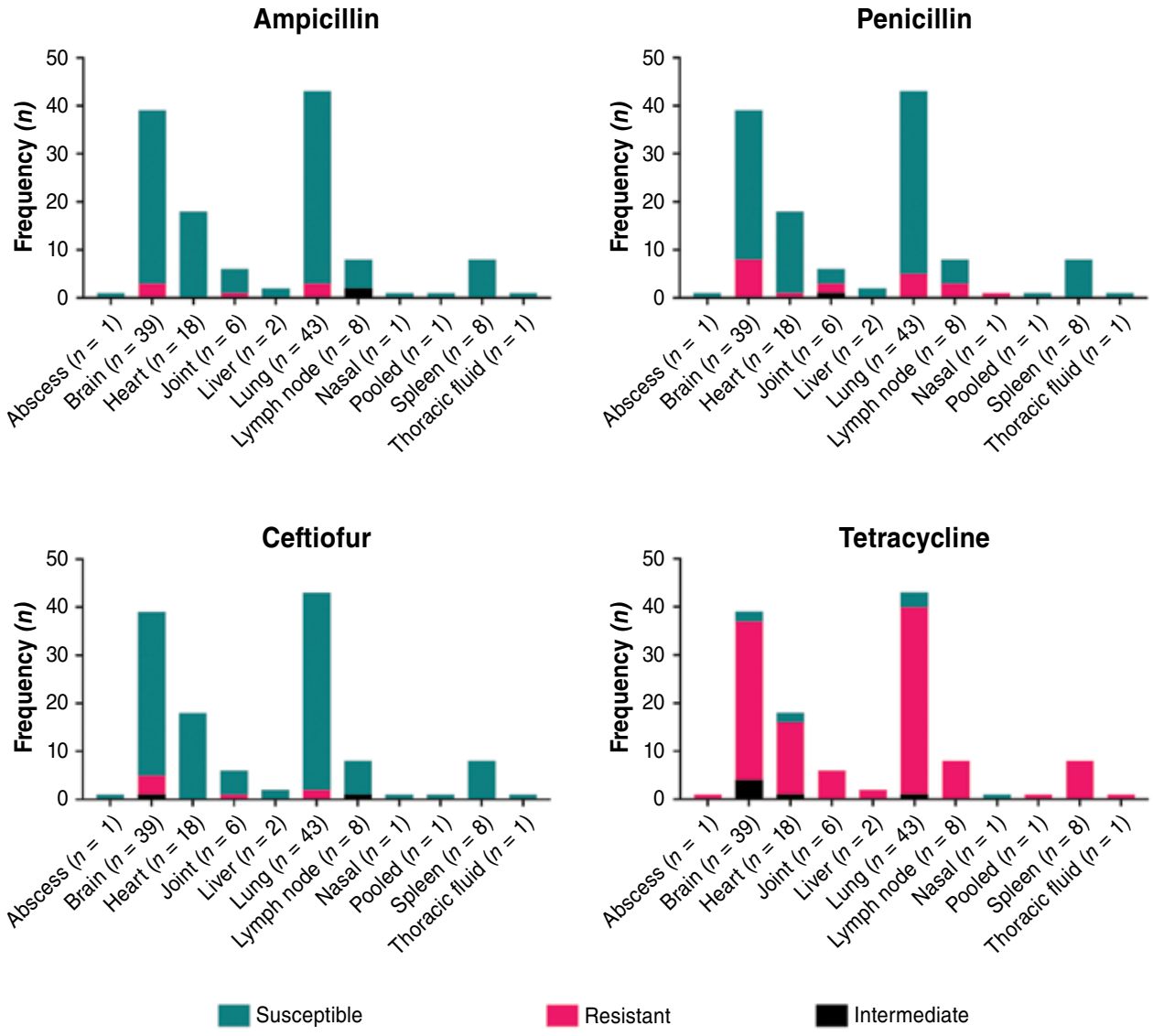

Figure 3. Antimicrobial susceptibility of *Streptococcus suis* isolates recovered from diseased pigs ($n = 128$), according to isolation site.

28 (17%, 23/128) and 839 (14%, 19/128). Isolates untypable by serotyping and MLST accounted for 23% (30/128) and 68% (88/128), respectively (see Figure S2, available online from: www.canadianveterinarians.net).

Antimicrobial susceptibility of the isolates is summarized in Figure 3 and Figure S3 (available online from: www.canadian veterinarians.net). Isolates were largely susceptible to ampicillin (92%, 119/128), penicillin (83%, 107/128), and ceftiofur (92%, 119/128), and mostly resistant to oxytetracycline (89%, 114/128). Penicillin-resistant isolates were isolated from brain (untypable serotype and sequence type, $n = 3$), joints (serotype 14, sequence type 94, $n = 1$), and lungs (serotype 10, sequence type 839, $n = 1$; untypable serotype and sequence type, $n = 2$). Intermediate resistance to penicillin was identified in 2 isolates form lymph nodes; both were untypable by serotyping and ST. There were no differences in antimicrobial susceptibility between conventional ($n = 96$) and antibiotic-free ($n = 25$) operations (see Figure S4, available online from: www.canadianveterinarians.net).

Of the 63 premises included in the study, 53 had 1 case submitted, 8 had 2 cases, and 2 had 3 cases. Nineteen cases had isolates obtained from noninvasive samples, whereas 43/75 were from invasive samples and 13/75 were recovered from both groups (lungs/nasal or other organs). The number of isolates per premises ranged from 1 to 9 (Table 1). Close to half (54%, 34/63) of the premises had 1 case and 1 isolate obtained. However, the remaining 46% (29/63) of premises had $>$ 1 isolate obtained during the study period. Of the 19 premises with 1 case and multiple isolates, 5/19 (26%) had a single serotype identified (including untypable *S. suis*) and the remaining 14/19 (74%) had $\geq$ 2 more serotypes identified. No noninvasive isolates were identified in cases with a clinical score of 4 and multiple serotypes detected (Table 2).

Table 1. Isolates and case distribution of pigs with *Streptococcus suis*, according to premises.

Number of isolates per premises	Number of premises by number of cases (column %)			Subtotal (number of nasal/lung isolates/% of row total isolates)
	1	2	3	
1	34 (64%)	—	—	34 (10/29%)
2	11 (21%)	2 (25%)	—	13 (7/26%)
3	4 (8%)	1 (13%)	1 (50%)	6 (6/33%)
4	2 (4%)	2 (25%)	—	5 (5/31%)
5	2 (4%)	3 (38%)	0 (0%)	5 (7/46%)
9	0 (0%)	0 (0%)	1 (50%)	1 (8/88%)
Subtotal	53	8	2	63 (43/33%)

Table 2. Serotype distribution of pigs with *Streptococcus suis* across 19 premises with 1 case and multiple isolates.

Premises	Province	Number of isolates	Clinical score[a]	*S. suis* serotype
MB27	Manitoba	2	1	7/8
MB19	Manitoba	2	3	UT
AB1	Alberta	2	4	3
AB5	Alberta	2	4	16/UT
AB6	Alberta	2	4	11
MB42	Manitoba	2	4	5/7
MB48	Manitoba	2	4	1/2/UT
SK4	Saskatchewan	2	4	3
SK7	Saskatchewan	2	4	1/2
SK8	Saskatchewan	2	4	2
MB18	Manitoba	2	N/A	10/UT
MB15	Manitoba	3	4	2/7
MB40	Manitoba	3	4	11/UT
MB43	Manitoba	3	4	4/16
SK6	Saskatchewan	3	4	1/2/3/UT
MB29	Manitoba	4	4	5/14/UT
MB37	Manitoba	4	4	2/7/15
SK2	Saskatchewan	5	4	14/UT
SK9	Saskatchewan	5	4	1/2/4

N/A — Not applicable.
[a] Score: 1 = normal morbidity and mortality, 2 = increased morbidity and normal mortality, 3 = normal morbidity and increased mortality, 4 = increased morbidity and mortality.

All 10 premises with 2 or 3 cases submitted had > 1 serotype identified, with at least 1 isolate identified as invasive (Table 3). In one instance (MB44), a single, diseased pig had 2 serotypes isolated from brain samples from a meningitis case: serotypes 2 and 4, sequence type 25 and sequence type 17, respectively.

From the 63 premises included in this study, 71% (45/63) recorded severe clinical presentation with increased morbidity and increased mortality (clinical score = 4; Table 4). There were no significant differences in clinical scores among provinces, number of cases per premises, or serotypes per case. The only significant factor was the number of isolates/serotypes detected (Table 4). As expected, premises with a clinical score of 4 had more isolates and serotypes recovered than premises with lower scores. Interestingly, isolates identified as invasive were more likely to be associated with a clinical score of 4 than noninvasive isolates ($P = 0.044$). Cases submitted from March to May had varied scores (ranging from 1 to 4). However, after May 2022, all cases had a clinical score of 4. This was a significant difference (75 cases; Fisher exact test, $P < 0.0001$). However, when this was assessed excluding a practice that stopped sending isolates after June 2022 (17 cases) and controlling for high-scoring practitioners, there was no significant difference (56 cases; Fisher exact test, $P = 0.24$).

The data presented here are, to the best of our knowledge, the first report profiling *S. suis* isolates associated with diseased pigs from western Canada. Serotypes 2, 3, 1/2, and 7 accounted for ~40% of the typable isolates obtained, whereas sequence type 28 and sequence type 839 were the most prevalent MLST types identified. No associations between sequence type or serotype and source organ or farm location were identified, although farms reporting a clinical score of 4 were more likely to have invasive isolates recovered than those with lower clinical scores. Furthermore, the data presented evidenced that ~74% of the premises had animals colonized by > 1 *S. suis* serotype.

Interestingly, we did not identify any non-*S. suis* serotypes in the study population. Approximately 30% of the isolates obtained in this study were untypable using the described methods, similar to those in previous studies from other geographical regions (5,18,19). When comparing serotype distribution with other Canadian reports, there were increased proportions of serotypes 3 (5% in Ontario, 7% in Quebec) and 7 (5% in Ontario), and a decreased number of serotype 9 (14% in Ontario) (5,6). In a report from the United States, 1/2 was the predominant serotype obtained from pigs (25%), followed by serotype 7 (11%) (8). Serotypes 2 and 1/2 were also the most frequently isolated types in Germany, whereas data from Spain and the Netherlands suggested that serotype 9 was more frequently identified in those countries (20,21). Based on our data, the serotype distribution present in western Canada was, as expected, different from other regions within Canada and other major pork-producing countries. Implications regarding clinical presentation, severity of disease, and control are unclear, but further studies using infection models are encouraged to help clarify these questions. Nevertheless, this prevalence difference should be considered in cases where animals or fomites are transported between countries, as the potential for introduction of new serotypes or sequence types is clear.

Further characterization of the genetic diversity within western Canadian *S. suis* was completed using MLST. The majority of isolates were identified as sequence types 28 and 839. A previous study that classified isolates based on their pathotypes suggested that MLST may be a better predictor of virulence potential than serotyping (8). This same study implicated sequence types 1, 28, 94, and 108 as pathogenic types, all of which were circulating in western Canadian herds. Sequence type 94 was identified as part of the most diverse clonal complex

Table 3. Serotype distribution of pigs with *Streptococcus suis* across the 10 premises with 2 or 3 cases, summarized by date of first submission (Date), serotypes detected at first submission (Serotype #1), days since last submission (Day 1, Day 2), and serotype(s) in second or third case (Serotype #2, Serotype #3).

	Case #1			Case #2			Case #3			
Premises	Date	Serotype #1	Isolate type	Day 1	Serotype #2	Isolate type	Day 2	Serotype #3	Isolate type[a]	Serotypes
MB3	31-Mar-21	3/UT	Noninvasive	13	18/7	Noninvasive	11	18/31	18 noninvasive, 31 invasive	3/7/18/31/UT
SK1	08-Apr-21	3	Noninvasive	111	1/2	Invasive	71	16	Invasive	1/2/3/16
MB1	04-Mar-21	9	Invasive	71	UT	Noninvasive	—	—	—	9/UT
MB2	21-May-21	UT	Noninvasive	25	23	Invasive	—	—	—	23/UT
MB4	26-Mar-21	4/UT	Invasive	84	UT	Invasive	—	—	—	4/UT
MB5	25-Mar-21	1	Invasive	35	5/24	Invasive	—	—	—	1/5/24
MB6	22-Apr-21	7	Noninvasive	48	2/16	Invasive	—	—	—	2/7/16
MB7	03-May-21	1/2	Invasive	24	UT	Invasive	—	—	—	1/2/UT
MB8	15-Apr-21	UT	Noninvasive	119	2/4/UT	Invasive	—	—	—	2/4/UT
MB9	08-Jul-21	3	Invasive	105	8	Invasive	—	—	—	3/8

[a] Isolate type grouped according to site of isolation: Invasive — Any internal organ other than lungs or nasal samples; Noninvasive — Isolate recovered from lung or nasal samples; Both — Isolates obtained from invasive and noninvasive sites.

Table 4. Clinical score distribution by number of isolates/serotypes for pigs with *Streptococcus suis*.

Number of isolates/ number of serotypes	Clinical score					Premises
	1	2	3	4	N/A	
1 isolate, 1 serotype	6	6	2	19	1	34
> 1 isolate, 1 serotype	0	0	1	5	0	6
> 1 isolate, > 1 serotype[a]	1	0	0	21	1	23
Premises subtotal	7	6	3	45	2	63

The clinical score was recorded by the submitting practitioner. Score: 1 = normal morbidity and mortality, 2 = increased morbidity and normal mortality, 3 = normal morbidity and increased mortality, 4 = increased morbidity and mortality.
N/A — Not applicable.
[a] $P = 0.003$, Fisher exact test.

(CC94) present in pigs, including the other 12 STs (including sequence type 839). However, it is important to highlight that this association was based on genotypical analysis only, and that there is a lack of animal studies (particularly in swine) investigating the true virulence potential of the sequence types described above. Other sequence types identified from clinical cases in Canada and the United States include STs 25, 145, 80, 68, 69, 72, 75, 92, 77, and 70 (22). In a report from human cases in Thailand, sequence type 1 was associated with meningitis, whereas sequence type 104 was associated with cases of sepsis without meningitis (23). As noted, there is a knowledge gap regarding the actual virulence potential of these clusters to swine based on animal trials, and how sequence types relate to supposedly highly virulent serotypes. This is largely due to a lack of reliable infection models, but this knowledge would provide guidance for further interpretation of epidemiological data.

The distribution of serotypes by premises indicated a tendency for more severe clinical presentation to be associated with invasive samples, more isolates, and more serotypes. The obvious source of bias here was that practitioners would submit more cases and samples from premises with increased mortality and morbidity. Another potential source of bias, which cannot be fully understood based on the data, is the relationship among clinical scores, season, and submission-bias, because the data included some practitioners that only scored 4 and only submitted cases between June and October. Regardless, there was 1 submission where a single pig was colonized and simultaneously infected with > 1 serotype and sequence type of *S. suis* — not a single serotype/sequence type. This has been reported (24) from tonsil swab samples, which represent a heavily contaminated site not associated with clinical disease. This finding raises concerns when producers and veterinarians choose autogenous vaccines as a control method — especially when > 2 serotypes are detected within the same pig. This first report of a scenario with mixed infection by different serotypes warrants that veterinarians request typing of multiple isolates when these are obtained from systemic sites (not lungs or tonsils) in a single pig.

Our analyses did not identify any associations between specific serotypes or sequence types and the likelihood of systemic invasion, but invasive isolates were more likely to be reported in farms experiencing increased morbidity and mortality. Vaccine protection efficacy is serotype-specific, with no or little cross-protection between serotypes. Some serotypes influence immunoglobulin isotype profile following exposure or vaccination due to the degree of opsonophagocytosis induced, which is specific for each serotype (25,26). Therefore, these data highlighted that *S. suis* diagnostics must consider that pigs host a heterogenous *S. suis* population, and that vaccines may only provide robust control of *S. suis*-associated disease in pigs when a polyvalent vaccine is developed. Antimicrobial susceptibility findings were unremarkable, following trends previously reported (5,10,18,27).

In conclusion, this is the first study to profile the *S. suis* population circulating in diseased pigs in western Canada. We inferred that serotype and sequence type distributions differed from what has been reported for other geographical areas with

substantial pork production. This has implications for regional *S. suis* control as well as for national and international animal movement — especially from nucleus and multiplier herds. No associations between serotype or sequence type and organ of isolation were identified; thus, no particular pathotypes were identified. Finally, we determined that diseased pigs could harbor a heterogeneous population of *S. suis* types simultaneously, which has implications for diagnosis and control.

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Case Report Rapport de cas

Decompressive craniectomy surgery in a dog with intracranial extradural hematoma following blunt force trauma

Ciaran C. O'Carroll, Bryan T. Welch, Meagan A. Walker, Adam T. Ogilvie, Lorrie Gaschen, Katie L. Hoddinott

Abstract — A young miniature poodle was presented following blunt force trauma to the head. The dog initially responded well to medical management before developing clinical signs associated with increased intracranial pressure 48 h post-injury that became refractory to hyperosmolar therapy. A computed tomography scan obtained 76 h post-injury showed a short, oblique, non-displaced, complete fissure in the right temporal bone and a second short, oblique, non-displaced, complete fissure in the ventral aspect of the temporal bone. A biconvex, moderately hyperattenuating, space-occupying temporoparietal lesion was visualized immediately adjacent to the area of the temporal fractures. These findings were consistent with a diagnosis of intracranial extradural hematoma. Decompressive craniectomy successfully evacuated the extradural hematoma to alleviate increased intracranial pressure. The dog's neurologic function recovered quickly postoperatively. At follow-up physical examinations at 14 and 437 d, excellent return to function was noted.

Key clinical message:
This report describes the diagnosis and surgical management of an intracranial extradural hematoma in a dog with increased intracranial pressure refractory to medical management. Furthermore, this report describes the diagnostic imaging findings used to diagnose this particular form of primary brain injury.

Résumé — Chirurgie de craniectomie décompressive chez un chien présentant un hématome extradural intracrânien à la suite d'un traumatisme contondant. Un jeune caniche miniature a été présenté à la suite d'un traumatisme contondant à la tête. Le chien a initialement bien répondu à la prise en charge médicale avant de développer des signes cliniques associés à une augmentation de la pression intracrânienne 48 heures après la blessure, qui sont devenus réfractaires au traitement hyperosmolaire. Une tomodensitométrie obtenue 76 heures après la blessure a montré une fissure complète courte, oblique, non déplacée dans l'os temporal droit et une deuxième fissure complète courte, oblique, non déplacée dans la face ventrale de l'os temporal. Une lésion temporo-pariétale biconvexe, modérément hyperatténuée et occupant de l'espace a été visualisée immédiatement à côté de la zone des fractures temporales. Ces résultats concordaient avec un diagnostic d'hématome extradural intracrânien. La craniectomie décompressive a réussi à évacuer l'hématome extradural pour atténuer l'augmentation de la pression intracrânienne. La fonction neurologique du chien s'est rétablie rapidement après l'opération. Lors des examens physiques de suivi à 14 et 437 jours, un excellent retour au fonctionnement a été noté.

Message clinique clé :
Ce rapport décrit le diagnostic et la prise en charge chirurgicale d'un hématome extradural intracrânien chez un chien présentant une augmentation de la pression intracrânienne réfractaire à la prise en charge médicale. En outre, ce rapport décrit les résultats de l'imagerie diagnostique utilisée pour diagnostiquer cette forme particulière de lésion cérébrale primaire.

(Traduit par Dr Serge Messier)

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Department of Companion Animals, Atlantic Veterinary College, University of Prince Edward Island, 550 University Avenue, Charlottetown, Prince Edward Island C1A 4P3 (O'Carroll, Welch, Walker, Ogilvie, Hoddinott); VetCT, 21 JJ Thomson Avenue, Cambridge CB3 0FA, United Kingdom (Gaschen).

Address all correspondence to Ciaran C. O'Carroll; email: cocarroll@upei.ca

Trauma is a common reason to seek veterinary medical care. Trauma represents the second-most likely cause of mortality in both juvenile and adult dogs (1). Approximately 55% of traumatic injuries in dogs are due to blunt force trauma, with motor vehicle accidents being the most common cause of severe blunt force trauma requiring intensive care unit admission (2,3). Further, ~25% of animals with severe blunt force trauma have concurrent head trauma (HT) (3).

Intracranial hemorrhage is a potential sequela in any animal that has experienced HT, but can also be caused by other conditions. Intracranial extradural hematoma (EDH) has been described in dogs as a result of trauma (4,5) or infection with *Cryptococcus* (6). Previous studies of dogs with HT suggested subdural hemorrhage is more common than extradural hemorrhage (4,7,8). To the authors' knowledge, there are few reports of surgical decompression for management of an intracranial EDH in the veterinary literature (4,5,8), with only 1 other description of the surgical technique (5).

This report describes the imaging findings for, diagnosis of, and successful decompressive craniectomy used to relieve increased intracranial pressure (ICP) caused by an intracranial EDH in a dog, with excellent return to function noted at follow-ups 14 and 437 d postoperatively. This procedure represents a viable treatment option for dogs with EDH who become refractory to medical management. This case report also supports the utility of computed tomography (CT) in critically ill animals with HT.

Case description

An intact male miniature poodle aged 4 y and 11 mo and weighing 6.2 kg was presented with blunt force HT after a steel ramp fell on him. The dog was reported to have been unresponsive for ~10 min immediately following the trauma. The owner noted hemorrhage coming from the right ear immediately after the accident. After regaining consciousness, the dog remained obtunded, vocalized intermittently, and was reported to have a right-sided head tilt with torticollis to the right.

On presentation to the primary care veterinarian, the dog was obtunded and unable to stand without support. A brief neurologic examination revealed normal pupillary light reflexes and normal withdrawal reflexes in all 4 limbs. The dog was hyperglycemic (15 mmol/L, reference: 3 to 6 mmol/L) and moderately hypokalemic (2.4 mmol/L, reference: 3.7 to 5.8 mmol/L). The attending veterinarian administered buprenorphine (0.03 mg/kg, IM) and an IV bolus of lactated Ringer's solution supplemented with 5 mEq of potassium chloride, administered over 2 h to correct the hypokalemia. The dog was also treated with mannitol (0.5 g/kg, IV) as it remained obtunded, raising suspicions of increased ICP.

The dog was referred to a veterinary teaching hospital for further evaluation and ongoing treatment. On presentation, no cardiac murmur or arrhythmia was detected on cardiac auscultation. The dog had mildly increased respiratory effort. Further examination revealed pink mucous membranes with a capillary refill time of < 2 s and a rectal temperature of 36.4°C. There was evidence of prior hemorrhage from the right ear canal noted during the examination. A truncated neurological examination was performed due to the dog's critical condition. The dog was obtunded, recumbent, vocalizing, and had a right-sided head tilt and right-sided torticollis. Menace and palpebral responses were present but diminished. Direct and consensual pupillary light reflexes were intact, and normal withdrawal reflexes were present in all 4 limbs. The limbs were intermittently in rigid extension. No other substantial injuries were noted on physical examination.

A CBC and serum biochemistry were evaluated. Mild neutrophilia (12.54×10^9/L, reference: 3 to 12×10^9/L) and thrombocytopenia (179×10^9/L, reference: 216 to 470×10^9/L) were noted on hematology. Serum biochemistry identified a mild hyperglycemia (6.7 mmol/L, reference: 3.3 to 6.1 mmol/L) and a mild increase in serum amylase (1720 U/L, reference: 200 to 1200 U/L). The dog was admitted to the intensive care unit and was placed in an oxygen cage set to provide 35 to 40% oxygen. Initial medical management included crystalloid fluid therapy with lactated Ringer's solution (2.5 mL/kg per hour, IV) and methadone (0.2 mg/kg, IV, q4h). N-acetylcysteine (Acetylcysteine Solution USP; Teligent Canada, Mississauga, Ontario) was administered (140 mg/kg IV loading dose, followed by 70 mg/kg, IV, q8h). In the initial 12 h post-admission, the heart rate ranged from 56 to 80 bpm and systolic blood pressure ranged between 131 to 139 mmHg. After this initial period, intermittent episodes of hypertension with inappropriate bradycardia (heart rate of 50 bpm and systolic blood pressure of 150 mmHg) developed, indicative of raised ICP. Treatment with a bolus of 7.2% hypertonic saline (5 mL/kg, IV) resolved the bradycardia and hypertension.

The dog initially responded well to treatment and its mentation had noticeably improved by 24 h post-admission. The dog was ambulatory with only mild ataxia, and its head tilt and torticollis had improved. However, by 48 h post-admission, the dog became dull, its head tilt and torticollis worsened, and it became nonambulatory. Increased ICP was again suspected to be the cause of the dog's deterioration due to concurrent bradycardia (68 bpm) and hypertension (154 mmHg systolic). Treatment with hyperosmolar agents was initiated. The dog received a bolus of 7.2% hypertonic saline (5 mL/kg, IV) and neurologic improvement was noted, along with resolution of the bradycardia and hypertension. Bradycardia and hypertension recurred shortly thereafter, with only a partial response to 2 further boluses of hypertonic saline. As the clinical signs became refractory to hypertonic saline, treatment with mannitol was initiated. The dog received 2 further doses of mannitol (500 mg/kg, IV) but was minimally responsive to this therapy.

A CT scan with contrast of the head and cervical spine was obtained 76 h post-trauma, to assess the cause of the increased ICP and aid in further treatment planning. Studies of the head and cervical spinal region were acquired, with the dog in sternal recumbency, using a 64-slice multidetector scanner (Aquilion 64; Canon Medical Systems, Otawara, Japan). Images were acquired pre- and post-administration of iodinated contrast media (2.5 mL/kg, IV) (Omnipaque; GE Healthcare Canada, Mississauga, Ontario) using contiguous slices of 1-millimeter thickness. The CT images showed a short, oblique, nondisplaced, complete fissure in the right temporal bone and a

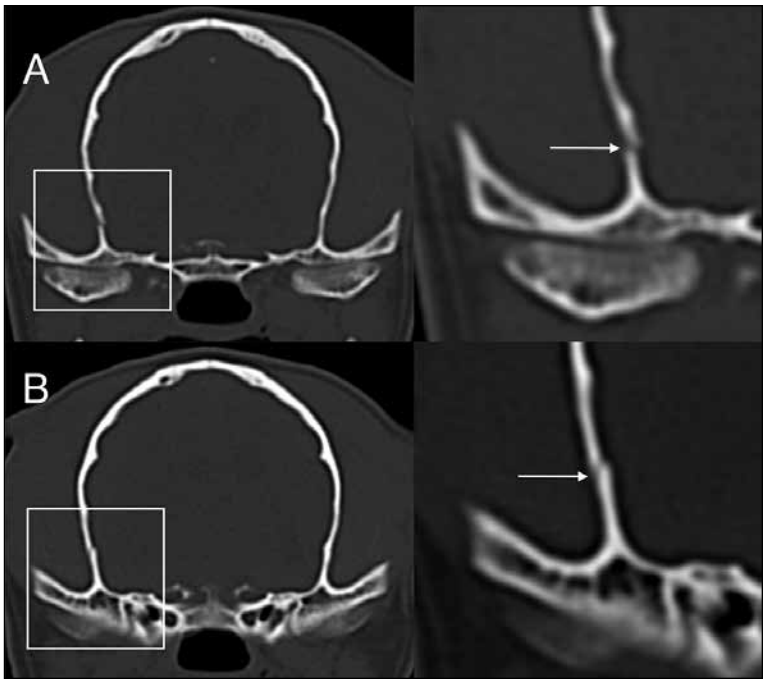

Figure 1. Computed tomographic scan of the calvarium shown in a bone window with 1-millimeter slice thickness in a dog with intracranial extradural hematoma. There are 2 non-displaced fissures in the right temporal bone: 1 cranial (A) and 1 caudal (B) (arrows in the magnified images). White boxes indicate the images magnified at right in A and B.

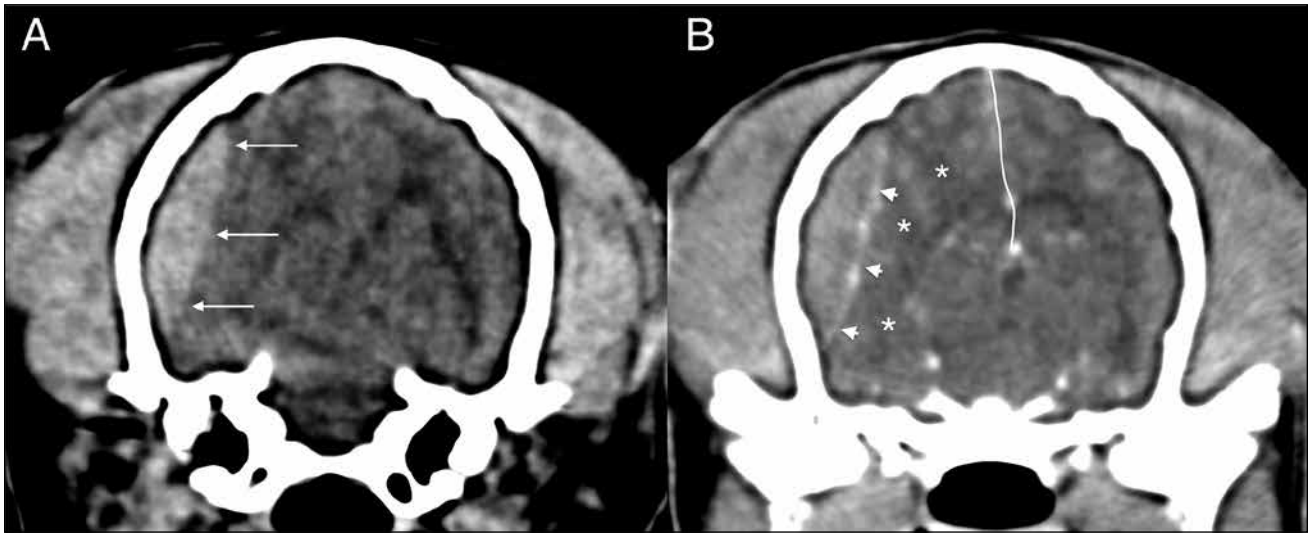

Figure 2. Pre- (A) and post- (B) contrast computed tomographic scans of the skull of a dog with intracranial extradural hematoma, in a soft tissue window with 1-millimeter slice thickness. A – There is a biconvex-shaped, mildly heterogenous hyperattenuating space-occupying temporoparietal lesion present (arrows). This is an extra-axial lesion characteristic of an extradural hemorrhage. B – Diffuse, ill-defined hypoattenuating intra-axial space-occupying lesion of the left cerebrum (asterices) with leftward shift of the *falx cerebri* (white line). The left lateral ventricle is decreased in size compared to the right. There is a hyperdense, medially displaced meningeal border present (arrowheads). Hypoattenuation of the cerebrum following trauma is prioritized to edema.

second short, oblique, non-displaced, complete fissure in the ventral aspect of the temporal bone, slightly further caudal (Figure 1). A biconvex, moderately hyperattenuating (60 to 70 Hounsfield units), space-occupying temporoparietal lesion was present immediately adjacent to the area of the temporal fractures (Figure 2). These findings were consistent with a diagnosis of EDH.

The temporal and parietal regions of the brain parenchyma medial to the space-occupying lesion had an ill-defined hypoattenuating region (Hounsfield unit values 10 < neighboring parenchyma) when compared to the corresponding structures of the left cerebral hemisphere, consistent with cerebral edema. In combination, these lesions created a mass effect that displaced the contrast-enhanced meningeal membranes medially and

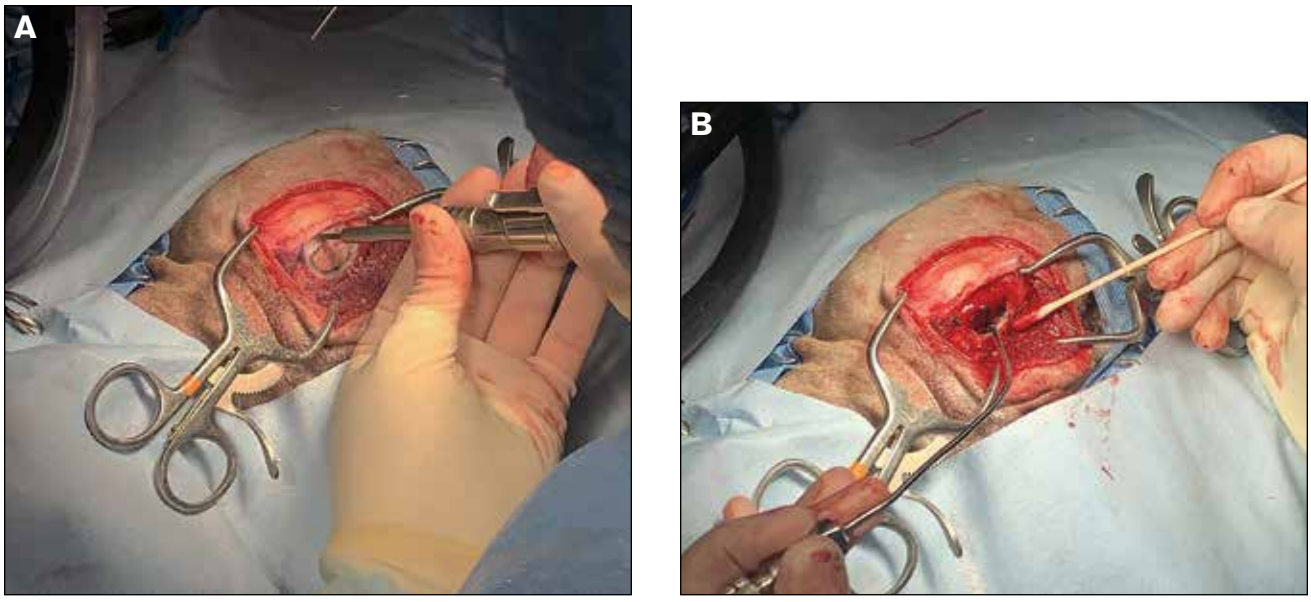

Figure 3. A – High-speed pneumatic burr for a craniectomy in a dog with with an intracranial extradural hematoma. B – Cotton-tipped applicators used for gentle removal of the epidural hematoma in a dog with with an intracranial extradural hematoma.

caused a leftward midline shift of the *falx cerebri.* There was a small amount of fluid attenuation in the ventral dependent aspect of the right tympanic bulla. It was unclear whether this represented preexisting *otitis media* or hemorrhage due to trauma. In light of the findings of dried blood on physical examination at admission and the owner-reported hemorrhage from the right ear, trauma was considered the most likely cause. Cervical spinal structures were unremarkable.

Based on the lesions noted on CT and the dog's deteriorating neurological status, a decompressive craniectomy was planned. A standard right-sided rostrotentorial approach was made. A high-speed pneumatic burr was used to make a 2-centimeter bone window in the parietal bone (Figure 3 A). The bone was gently removed until the underlying EDH was exposed. The hematoma was gently removed using moistened cotton applicators, ocular spears, and a ball probe with a gentle drip of sterile saline (Figure 3 B). A 3-ply porcine small intestinal submucosa graft (BioSIS; Vetrix, Cumming, Georgia, USA) was applied over the bony craniectomy defect, and the temporalis muscle was rotated back into position and re-apposed using 3-0 polydioxanone suture in a simple interrupted pattern, followed by routine closure of the subcutaneous tissues and skin. An esophagostomy tube and an indwelling urinary catheter were placed to aid postoperative management.

The dog recovered in the intensive care unit in an oxygen cage delivering 35 to 40% oxygen. The dog was positioned in lateral recumbency with his head maintained in an elevated position. A fentanyl infusion (5 μg/kg per hour, IV) was continued postoperatively to provide analgesia before the dog was transitioned to gabapentin (10 mg/kg, PO, q8h). Acepromazine (0.005 mg/kg, IV) and alprazolam (0.02 mg/kg, PO) were administered as required to alleviate anxiety and limit excess mobility during the recovery period. Dexamethasone (0.01 mg/kg, IV, q24h) was administered postoperatively because of its anti-inflammatory properties. Supportive care was continued and included IV crystalloid fluid therapy (lactated Ringer's solution), prophylactic IV antibiotics (cefazolin, 22 mg/kg, IV, q8h), antiemetics (maropitant citrate, 1 mg/kg, IV, q24h; ondansetron, 0.25 mg/kg, IV, q8h), and a promotility agent (metoclopramide, 2 mg/kg per day, administered as a continuous rate infusion). Antiemetic and prokinetic therapies were considered necessary as the dog had 1 episode of regurgitation during its recovery from anesthesia; this raised concerns for potential elevations of ICP if emesis were to occur, and for the risk of aspiration pneumonia due to the dog's recumbency and sedation.

The dog's neurological status improved progressively following surgery and no further clinical signs of increased ICP developed. The dog was mildly ataxic and had a mild head tilt 48 h postoperatively, but was ambulating well. The dog was eating and interacting well with its environment and caretakers. No complications were noted during the postoperative period, and the dog was discharged from the hospital 4 d postoperatively with a 5-day course of amoxicillin/clavulanic acid suspension (14 mg/kg, PO, q12h) and 14-day course of gabapentin (10 mg/kg, PO, q8h). At 14 d postoperatively, the dog had no neurological deficits and the previously identified head tilt and ataxia had resolved. The surgical site had healed well and skin sutures were removed. The esophagostomy tube had become dislodged while traveling to the recheck appointment, and the stoma was allowed to heal by secondary intention. At 437 d postoperatively, the dog returned for a follow-up physical examination. The dog's owners reported no abnormalities or abnormal behaviors at home. The dog was pain-free and mentally appropriate, with no neurological deficits identified on examination. Both follow-up examinations were done at the veterinary teaching hospital by the same clinicians initially involved in the case.

Discussion

Brain injuries following trauma are divided into primary and secondary injuries. Primary injuries, such as epidural and subdural hematomas, contusions, and lacerations, occur immediately following trauma as a direct result of the kinetic forces disrupting intracranial structures. Secondary injuries occur over an interval following injury and are thought to occur *via* release of excitatory neurotransmitters leading to neuronal uptake of calcium that promotes oxygen radical reactions. The reactive oxygen species produced as a result of this process may contribute to cellular damage, necrosis, and subsequent cerebral edema (9). Most efforts in veterinary medicine focus on recognizing and preventing secondary brain injury, as early, targeted therapy can minimize propagation of secondary brain injury (9). Treatments directed at resolving or minimizing primary injury; *e.g.,* decompressive craniectomy, are not commonly used and may be an area for further development in veterinary medicine.

Traditional medical management of traumatic brain injury (TBI) in veterinary medicine has historically consisted of oxygen therapy, elevating the head, analgesics, hyperosmolar therapy in the face of intracranial hypertension, treatment of post-traumatic seizures, and mechanical ventilation when required. Administration of N-acetylcysteine replenishes glutathione stores that become depleted following brain trauma. Glutathione has antioxidant properties that reduce the production of reactive oxygen and reactive nitrogen species (10). N-acetylcysteine has had promising results as an adjunctive therapy for TBI in clinical trials of human patients and animal models of TBI (11). Therefore, N-acetylcysteine may reduce reactive oxygen species and limit their consequences for animals with TBI.

Intracranial hemorrhages, such as that described above, are a form of primary brain injury and can be subcategorized as intracerebral and extracerebral lesions (12). Intracerebral lesions are located within the brain parenchyma, whereas extracerebral lesions are located outside the brain parenchyma and include hematomas in the intraventricular, subdural, subarachnoid, and epidural spaces. Intracranial hemorrhage can lead to rapid formation of space-occupying lesions within the intracranial space, leading to increased ICP. In addition, cerebral edema secondary to intracranial hemorrhage can lead to further increases in ICP (13). In severe cases, increased ICP can lead to fatal herniation of the brain stem through the *foramen magnum* (9).

In human patients, EDH is present in ~2% of patients with TBI (14), with reported mortality rates due to EDH ranging from 1.2 to 33% (15). Epidural hematomas are usually the result of bleeding caused by fractures of the bones of the skull. Approximately 11 to 47% of human patients with EDH are reported to experience a period of unconsciousness immediately following injury, followed by an initial period of recovery, often referred to as a "lucid interval," before ultimately experiencing a deterioration in neurological status (15). This pattern is similar to that of the dog in this report and may explain the initial improvement in neurological status followed by the eventual deterioration.

Computed tomography was crucial in the management of this case as it accurately detected and characterized a cause of increased ICP that could be managed surgically. The cause of extradural hemorrhage is well-described in humans. It can result from meningeal arterial bleeding and trauma to venous structures or diploic channels within the calvarium (16). Computed tomography can identify small fissures of the calvarium and can differentiate epidural from subdural hemorrhage. The diagnosis of extradural hemorrhage is based on the identification of a lentiform (biconvex, lens-shaped), space-occupying, hyperdense lesion along the calvarium (17). Subdural hemorrhage is characterized by a hyperdense crescent with a concave shape adjacent to the calvarium, where the internal margin parallels the margin of the adjacent brain. Both extradural and subdural lesions are extra-axial. Computed tomography can also identify intra-axial injuries, such as hemorrhage and edema, that complicate and help to explain the clinical signs and neurological examination results. Acute hemorrhage is hyperdense on CT and decreases with time. Hypodensity is indicative of acute edema. Both hyperdense hemorrhage and hypodense cerebral lesions can be present simultaneously in dogs with TBI (18). In previous veterinary studies, intracranial hemorrhage was detected reasonably frequently in dogs undergoing CT (7,18,19) or magnetic resonance imaging (4) of the head in the first 72 h after HT. The role of early CT for prognostication in dogs remains uncertain, as some studies suggested that CT has only limited value for prognostication in the interval immediately after HT (19). The Koret CT score has been developed (18) and validated (7) as a prognostic scoring system to help predict short- and long-term outcomes in dogs with HT, using CT images acquired within 72 h after injury. It is clear that further research is required in this area to better elucidate the utility of CT imaging in canine TBI. Unfortunately, in this case, the dog did not undergo imaging until ~76 h post-injury, making use of the Koret CT score more challenging.

Indications for surgical intervention in human patients with EDH have been defined by the Brain Trauma Foundation (20). Surgical intervention is based on hematoma volume and thickness, degree of midline shifting of the *falx cerebri*, and clinical assessment of the individual. Currently, decompressive craniectomy is recommended for any human patient with high-volume EDH (> 30 cm^3), regardless of their Glasgow Coma Scale scores. Brain Trauma Foundation recommendations also suggest that human patients with smaller hematomas who also have significant changes in mental status (Glasgow Coma Scale < 9) and anisocoria should undergo surgical evacuation (20). It is likely that similar guidelines could be developed for dogs.

Human patients undergoing decompressive craniectomy for EDH typically undergo cranioplasty as a subsequent procedure following craniectomy (21–23). Cranioplasty is typically performed to improve cosmesis and provide protection to the contents of the cranial vault. The excised bone flap can undergo cryopreservation (22) or can be stored in a subcutaneous pocket in the abdominal area (21), allowing it to be repositioned in the craniectomy defect as an autogenous bone graft in the future. The feasibility of similar techniques in veterinary medicine has yet to be demonstrated, though they are likely possible. Cranioplasty has been described in dogs undergoing craniectomy using various methods, including preformed PMMA

cement caps (24) and custom titanium implants (25). The unplanned nature of the intervention in this case precluded such procedures. The use of porcine small intestinal submucosa was reported for reconstruction of a cranial defect created during removal of a neoplasm (26). This technique can be performed quickly, with minimal planning, and was very effective.

In conclusion, decompressive craniectomy is uncommonly performed in dogs. However, it may prove to be beneficial in the management of dogs with clinical signs of increased ICP secondary to intracranial EDH. In addition, CT imaging is warranted in cases of TBI that decompensate in the face of traditional medical management. CVJ

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Case Report Rapport de cas

A novel acrylic orthodontic device for treatment of linguoverted mandibular canine teeth in small dogs

Daehyun Kwon, Kwangsik Jang, Yangwon Chae, Euisin Yang, Seong Soo Kang, Se Eun Kim

Abstract — Linguoverted mandibular canine teeth (LMC) is a common malocclusion in dogs. Several inclined bite-plane techniques using acrylic resin have been introduced to correct LMC in dogs. Although these techniques have suggested modifications to overcome shortcomings, there are still limitations; *e.g.,* high technical sensitivity, as the viscous acrylic resin must still be fabricated in the oral cavity. The authors developed a novel method for small-breed dogs that uses a doughy acrylic resin form to achieve an easy intraoral design and extraoral fabrication. Eight small-breed dogs were presented to evaluate and treat malocclusion causing palatal trauma. First, a Class-1 malocclusion with linguoversion of the mandibular canine teeth (6 dogs with unilateral LMC and 2 dogs with bilateral) was diagnosed based on oral examination. Dogs were treated with the new method using a doughy acrylic resin form for 6 to 7 wk and had posttreatment follow-up 1 y after the procedure. All treated canine teeth were in correct positions 1 y after the appliances were removed.

Key clinical message:
The authors believe that the new method using a doughy acrylic resin form could be a good alternative for veterinarians to use when treating LMC.

Résumé — Un nouveau dispositif orthodontique en acrylique pour le traitement des canines mandibulaires linguoverties chez les petits chiens. Les canines mandibulaires linguoverties (LMC) sont une malocclusion courante chez le chien. Plusieurs techniques de plan de morsure incliné utilisant de la résine acrylique ont été introduites pour corriger la LMC chez le chien. Bien que ces techniques aient suggéré des modifications pour surmonter les lacunes, elles présentent encore des limites; par exemple, une sensibilité technique élevée, car la résine acrylique visqueuse doit encore être fabriquée dans la cavité buccale. Les auteurs ont développé une nouvelle méthode pour les chiens de petite race qui utilise une forme pâteuse de résine acrylique pour obtenir une conception intra-orale et une fabrication extra-orale faciles. Huit chiens de petite race ont été présentés pour évaluer et traiter une malocclusion provoquant un traumatisme palatin. Tout d'abord, une malocclusion de classe 1 avec linguoversion des canines mandibulaires (6 chiens avec LMC unilatérale et 2 chiens avec bilatérale) a été diagnostiquée sur la base d'un examen oral. Les chiens ont été traités avec la nouvelle méthode en utilisant une forme pâteuse de résine acrylique pendant 6 à 7 semaines et ont fait l'objet d'un suivi post-traitement 1 an après la procédure. Toutes les canines traitées étaient dans la bonne position un an après le retrait des appareils.

Message clinique clé :
Les auteurs estiment que la nouvelle méthode utilisant une forme pâteuse de résine acrylique pourrait être une bonne alternative que les vétérinaires pourraient utiliser lors du traitement du LMC.

(Traduit par Dr Serge Messier)

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MAY Veterinary Dental Hospital, Seoul, Republic of Korea (Kwon); Department of Veterinary Surgery, College of Veterinary Medicine and BK21 FOUR Program (Kwon, Jang, Chae, Yang, Kang, Kim) and Biomaterial R&BD Center (Jang, Chae, Yang, Kang, Kim), Chonnam National University, 77, Yongbong-ro, Buk-gu, Gwangju, 61186, Republic of Korea.

Address all correspondence to Se Eun Kim; email: ksevet@jnu.ac.kr

Daehyun Kwon and Kwangsik Jang contributed equally to this work.

In the normal anatomic alignment of teeth, the maxillary incisor teeth are rostral to the mandibular incisor teeth, and the mandibular canine teeth interdigitate between the maxillary canine teeth and the third incisor (1,2). Malocclusion, a common dental problem in dogs, is designated Classes 1 to 4 (3,4). Class-1 malocclusion is the malposition of 1 or more teeth, including linguoversion of the mandibular canine teeth, mesioversion of the maxillary canine teeth, and rotated and infraerupted teeth. Symmetrical skeletal malocclusions are defined as Class 2 (mandibular brachygnathism) and Class 3 (mandibular prognathism). Skeletal asymmetry of the mandible or maxilla is defined as Class-4 malocclusion (5,6).

Lingual deviation of the mandibular canine teeth, currently termed linguoverted mandibular canine teeth (LMC), is relatively common in dogs and is also called base narrow, linguoverted, or lingually displaced mandibular canine teeth (4,7,8). The causes of this condition include persistent deciduous teeth, malpositioned tooth buds, trauma to a tooth or tooth bud during eruption, a brachygnathic mandible, and excessive anisognathism (3,9,10). Linguoverted mandibular canine teeth can cause various problems, such as gingivitis and palatal trauma, potentially leading to oronasal fistula formation and incompetent closure of the mouth (7,9,11,12).

One treatment option for LMC is orthodontic therapy, with a primary goal to provide comfortable, functional, and harmonious occlusion *via* a minimally invasive method (4,7). There are various orthodontic techniques for treating LMC, including gingivoplasty, rubber-ball therapy, and orthodontic appliances such as expansion screw devices, "W"-springs, cast metal telescoping inclined planes, temporary crown extensions, and acrylic inclined bite planes (1,2,4,7,9,12–14). In particular, acrylic inclined bite planes have been widely used for decades due to their convenience and cost advantage (4,13). However, in our experience, intraoral fabrication of inclined bite planes in a dog's oral cavity is technically challenging due to the limited space and viscosity of materials used.

As the limitations described are especially challenging in small-breed dogs, we designed a novel appliance to overcome them. The appliance, an intraorally designed but extraorally fabricated inclined plane made from an acrylic resin with a doughy consistency, was successfully used in 8 small-breed dogs. This approach was simple, inexpensive, and noninvasive, enabling production of an accurate and finely polished appliance.

Case descriptions

Selection of animals

From 2013 to 2020, 8 client-owned small-breed dogs (Table 1) from private households were presented to MAY Veterinary Dental Hospital (Seoul, Republic of Korea) with LMC. They were enrolled in the study after being designated as Class 1 among 7 classes defined by the American Society of Anesthesiologists Physical Status (ASAPS) (15).

Each dog's physical status, energy level, and appetite were good, with general physical examination results within the normal ranges. Conscious oral examinations revealed that the occlusal relationship of the 8 dogs was Class-1 malocclusion. Six dogs had unilateral lingually displaced mandibular canine teeth and the other 2 had bilateral lingually displaced mandibular canine teeth (Figure 1). Three dogs had unilateral or bilateral persistent deciduous canine teeth.

Treatment plan

The treatment plan for each dog was discussed with its owner preoperatively. The main objective was to eliminate the traumatic interlock between the crown of the mandibular canine tooth and the maxilla (16). The discussed treatment options included extraction of mandibular canine teeth or opposing incisors (interceptive orthodontics of permanent teeth), crown-height reduction combined with vital pulp therapy, ball therapy, and orthodontic treatment using a fixed or removable device; *e.g.,* inclined bite planes (8,11,17).

An oral examination was done and full-mouth intraoral radiographs were obtained to confirm there was sufficient space for the orthodontic movement of the mandibular canine teeth. All findings were recorded. Ultrasonic scaling was done on all teeth, which were then polished with fluoride-free pumice paste (Nupro Prophy Paste Fluoride-Free; Dentsply Sirona Korea, Seoul, Republic of Korea). Bilateral inclined bite planes were planned for all dogs because a unilateral inclined bite plane can cause shifting of the mandible instead of buccal movement of the canine tooth.

Treatment technique

Glycopyrrolate (0.01 mg/kg) and tramadol (2.5 mg/kg) were given SC before general anesthesia. Each dog was premedicated with butorphanol (0.1 mg/kg) and midazolam (0.2 mg/kg), IV, and anesthesia was induced with propofol (4 mg/kg), IV. Following placement of a cuffed endotracheal tube, inhalation anesthesia was maintained with 1.5 to 2% isoflurane and O_2 at 2 L/min.

First, a dough was created by mixing an acrylic resin polymer and monomer (Ortho-Jet; Lang Dental Manufacturing Company, Wheeling, Illinois, USA) (Figure 2 A, B). Then, the dough was placed in the diastema between the maxillary third incisor tooth and the canine tooth and a basic form created by manually rolling it into the proper size and shape (Figure 2 C). A spatula was used to ensure a 2-millimtere space between the form and the hard palate, facilitating self-cleaning and efficient home care (Figure 2 D). Excess acrylic was trimmed with scissors extraorally. The angles and directions of the intended inclined plane were marked by replacing the uncured acrylic in its intended position in the mouth, and by opening and closing the mandible (Figure 2 E). Curing of the primary form was accelerated by immersing it in warm water for 30 to 60 s.

Trimming with a carbide denture burr along marked lines was done extraorally (Figure 3 A). Two additional grooves were made on mesial and distal aspects of the appliance to improve the adhesion by increasing the adhesive-appliance interface (Figure 3 B). A prepared inclined bite plane was placed in the dog's mouth again to reevaluate the design, and then the appliance was removed for final polishing with a silicone point (Silicone point R2; Shofu, Kyoto, Japan) and Robinson brush (Buffalo Dental Manufacturing, Syosset, New York, USA). The finely polished surface reduces plaque adhesion and improves

Table 1. Details regarding using an acrylic orthodontic device for treatment of linguoverted mandibular canine teeth in small-breed dogs.

Dog	Breed	Age (mo)	Sex	Body weight (kg)	Description	Classification	Appliance retention time	Other treatment
1	Toy poodle	11	CM	4.2	Unilateral	Class 1	7 wk (after expansion of the space between the maxillary third incisor and the canine for 6 wk)	Extraction of deciduous maxillary and mandibular canine
2	Toy poodle	13	SF	5.1	Unilateral	Class 1	8 wk	No
3	Maltese	12	CM	3.4	Bilateral	Class 1	7 wk (after expansion of the space between the maxillary third incisor and the canine for 5 wk)	Extraction of deciduous mandibular canine
4	Pomeranian	12	CM	3.2	Unilateral	Class 1	7 wk (after expansion of the space between the maxillary third incisor and the canine for 5 wk)	Extraction of deciduous mandibular canine
5	Toy poodle	11	SF	4.5	Unilateral	Class 1	6 wk	No
6	Dachshund	36	SF	6.2	Bilateral	Class 1	8 wk (after expansion of the space between the maxillary third incisor and the canine for 6 wk)	No
7	Pomeranian	14	CM	3.6	Unilateral	Class 1	8 wk	No
8	Maltese	15	SF	3.8	Unilateral	Class 1	8 wk	No

CM — Castrated male; SF — Spayed female.

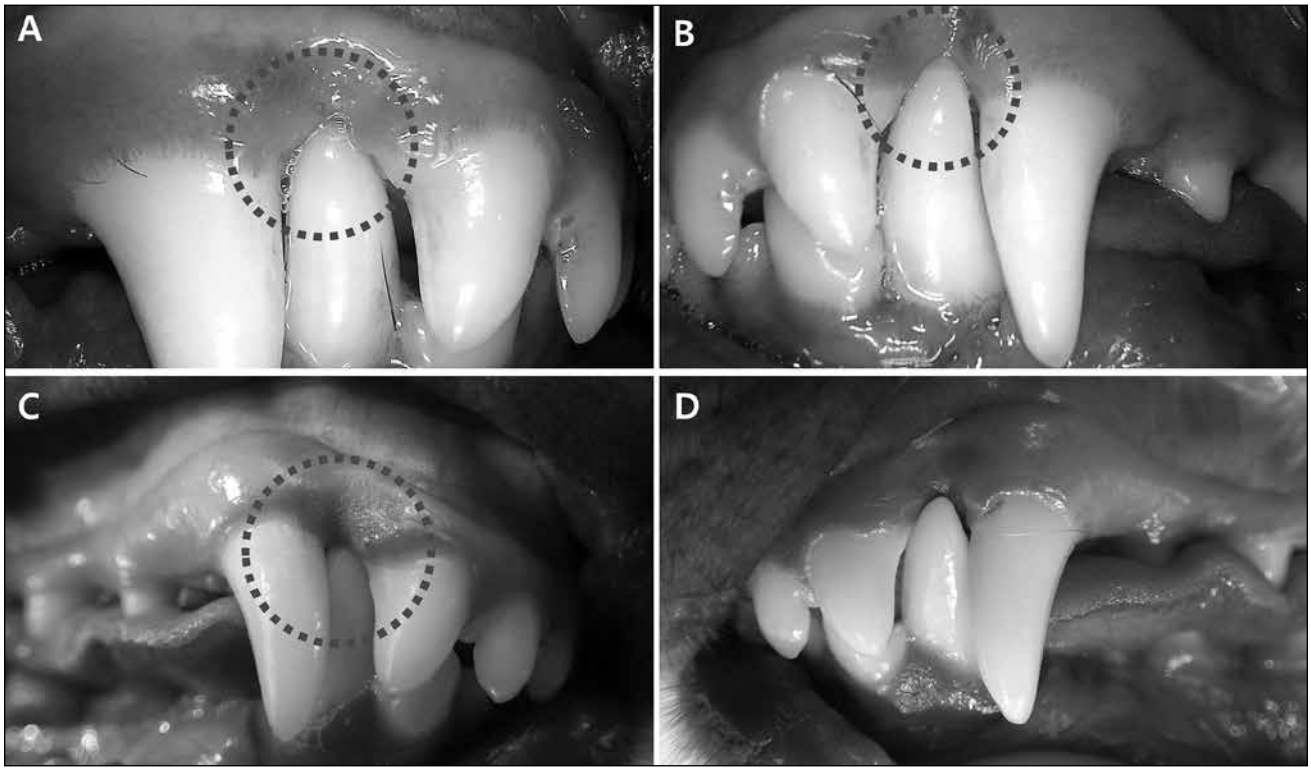

Figure 1. Examples of bilateral mesioverted canine teeth (A and B) and a unilateral mesioverted canine tooth (C and D) in dogs. The right (A) and left (B) mandibular canine teeth are linguoverted bilaterally and impinge on the gingiva between the maxillary third incisor teeth and canine teeth (dotted circle). C – The unilateral linguoverted right mandibular canine tooth has caused trauma to the maxilla (dotted circle). Note the narrow space between the right maxillary third incisor tooth and the canine tooth. D – The left mandibular canine tooth has normal occlusion.

oral hygiene (Figure 3 C, D). The maxillary third incisor tooth and the canine tooth were etched for 20 s with 37% phosphoric acid gel and rinsed for 20 s. The appliance was bonded onto the etched surface with core build-up resin (Light-Core; Bisco, Schaumburg, Illinois, USA) and cured using a blue light (Be Lite; B&L Biotech, Ansan-si, Republic of Korea) for 20 s (Figure 4).

In 4 dogs, the diastema between the maxillary third incisor tooth and the canine tooth was too small to accommodate the tipping mandibular canine. In these dogs, distal tipping mechanics, with orthodontic buttons and elastic chain on the maxillary canine tooth and the reinforced anchor teeth (fourth premolar and first molar) (18), were used. All persistent deciduous teeth were extracted at that time (Table 1). After obtaining enough

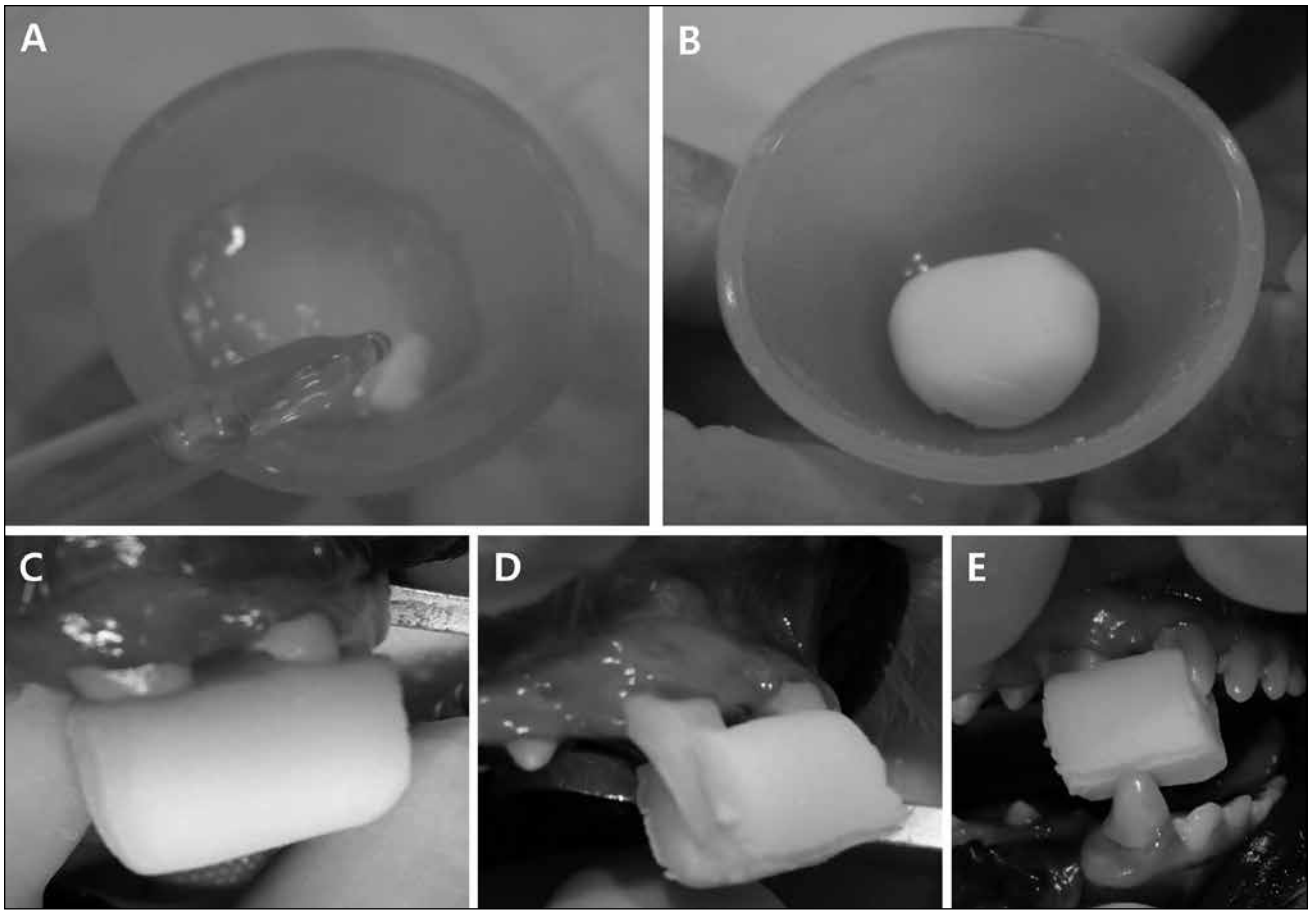

Figure 2. Design of the doughy acrylic resin form. A – A dough was created by mixing an acrylic resin polymer and monomer. B – The created dough. C – The dough was placed in the space between the maxillary third incisor tooth and the canine tooth. D – A spatula and scissors were used to create the primary dough form with the appropriate shape and size. Note the 2-millimeter space between the primary dough form and the gingiva, for maintaining sanitation. E – The direction and degree of movement of the mandibular canine tooth were marked through manual opening and closing motions.

space, the button and elastic chain were removed, and the acrylic inclined bite planes were created as described to induce buccal movement of the mandibular canine teeth.

Follow-up

Each dog's owner was instructed to rinse the oral cavity with 0.12% chlorhexidine solution daily and clean around the appliance until it was removed. In addition, daily tooth brushing, including of the appliance area, was also recommended. A regular evaluation was done weekly. Once the mandibular canine teeth had moved to the desired positions, the inclined bite plane was removed under general anesthesia (using the same anesthetic protocol described). Using a fissured diamond bur, the acrylic resin was sectioned between the teeth, and long, vertical grooves were made using the same bur in the acrylic resin, avoiding iatrogenic damage to teeth. Sections of acrylic were gently separated from the teeth with small extraction forceps applying torque in a twisting motion. Residual adhesive was removed using an ultrasonic scaler and teeth were polished with a contouring and polishing disc (3M disc; 3M Korea, Seoul, Republic of Korea).

Thereafter, each dog was temporarily extubated to assess occlusion. In all dogs, assessments revealed that all target teeth were in the desired positions (Figure 5). Each dog was reintubated, a complete oral examination was performed, and all remaining teeth were scaled and polished with fluoride-polishing pastes.

All dogs were examined 1 y after removal of appliances. Those examinations confirmed that all treated canine teeth remained in correct positions.

Discussion

Tooth movement in young animals is relatively more effective than in older animals due to active growth of tissues in the oral cavity. Furthermore, young dogs usually adapt quickly to orthodontic appliances (4). Linguoverted mandibular canine teeth in dogs is a common indication for orthodontic treatment (7,17). If this condition is not treated in a timely fashion, it can cause various problems that range from mild to severe. For example, a pseudopocket of an incompletely erupted mandibular canine tooth can cause periodontal disease, whereas the trauma from the contact between an LMC and palatal tissues can cause an oronasal fistula (5,7). In addition, this type of malocclusion may induce dysmasesis, temporomandibular joint dysfunction, and abnormal growth and development of dentition and the facial skeleton (4,19). In particular, continuous pressure on

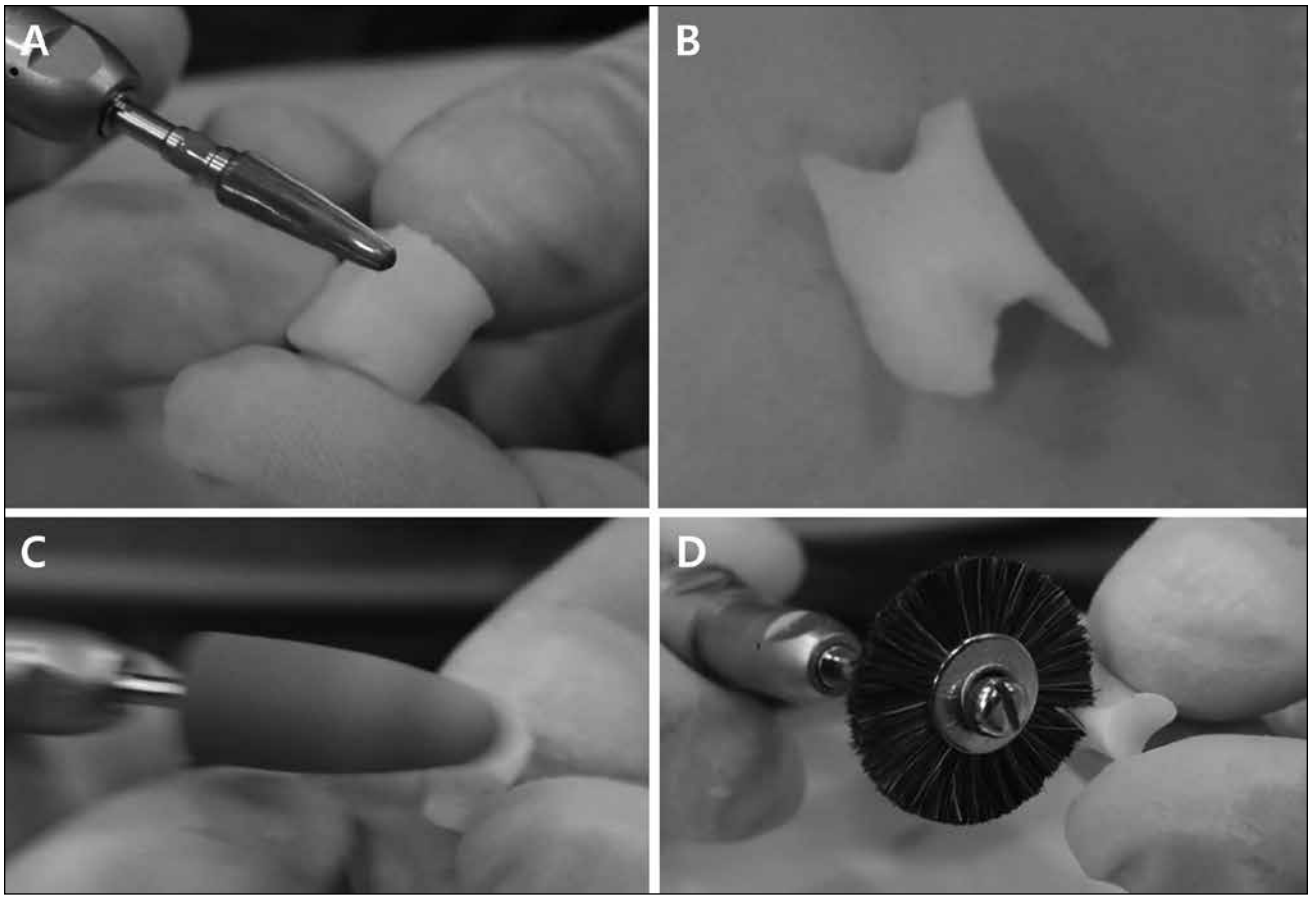

Figure 3. Sculpting the inclined bite plane. A – Trimming with a carbide denture bur was done extraorally along the marked lines. B – Two additional grooves were made on each side of the appliance to increase the adhesive strength. C and D – Polishing the appliance using a silicone point (C) and Robinson brush (D).

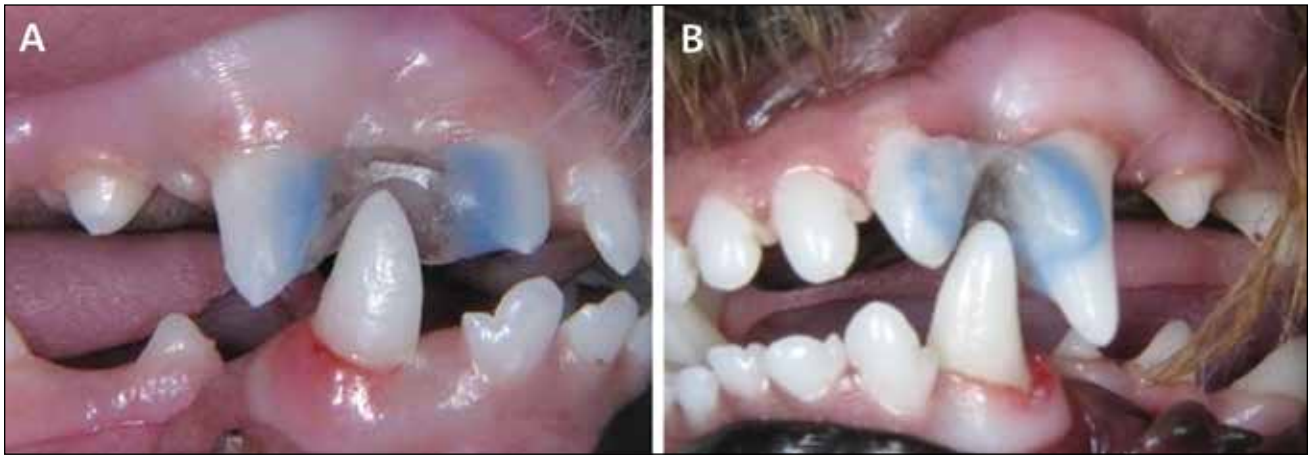

Figure 4. The maxillary third incisor tooth and the canine tooth were etched for 20 s with phosphoric acid gel. Inclined bite planes were placed onto the right (A) and left (B) mandibular etched surfaces with core build-up resin.

the tip of the crown due to malocclusion may compromise the blood supply due to intrusion, leading to pulpitis and pulp necrosis (8). Before orthodontic treatment, owners must agree not to breed the affected animals, to prevent malocclusion in the offspring (5,9).

In mild cases of linguoverted canine teeth, gingivoplasty of the gingiva between the maxillary third incisor tooth and the canine tooth can be effective (4). Ball therapy can also change the inclination of the mandibular canine teeth buccally without additional orthodontic treatment; however, not all dogs are interested in playing with balls (12). It is also difficult for owners to devote the time needed to create an environment in which animals can be interested in and play with the ball for sufficient time for therapeutic purposes (12). In addition, persistent

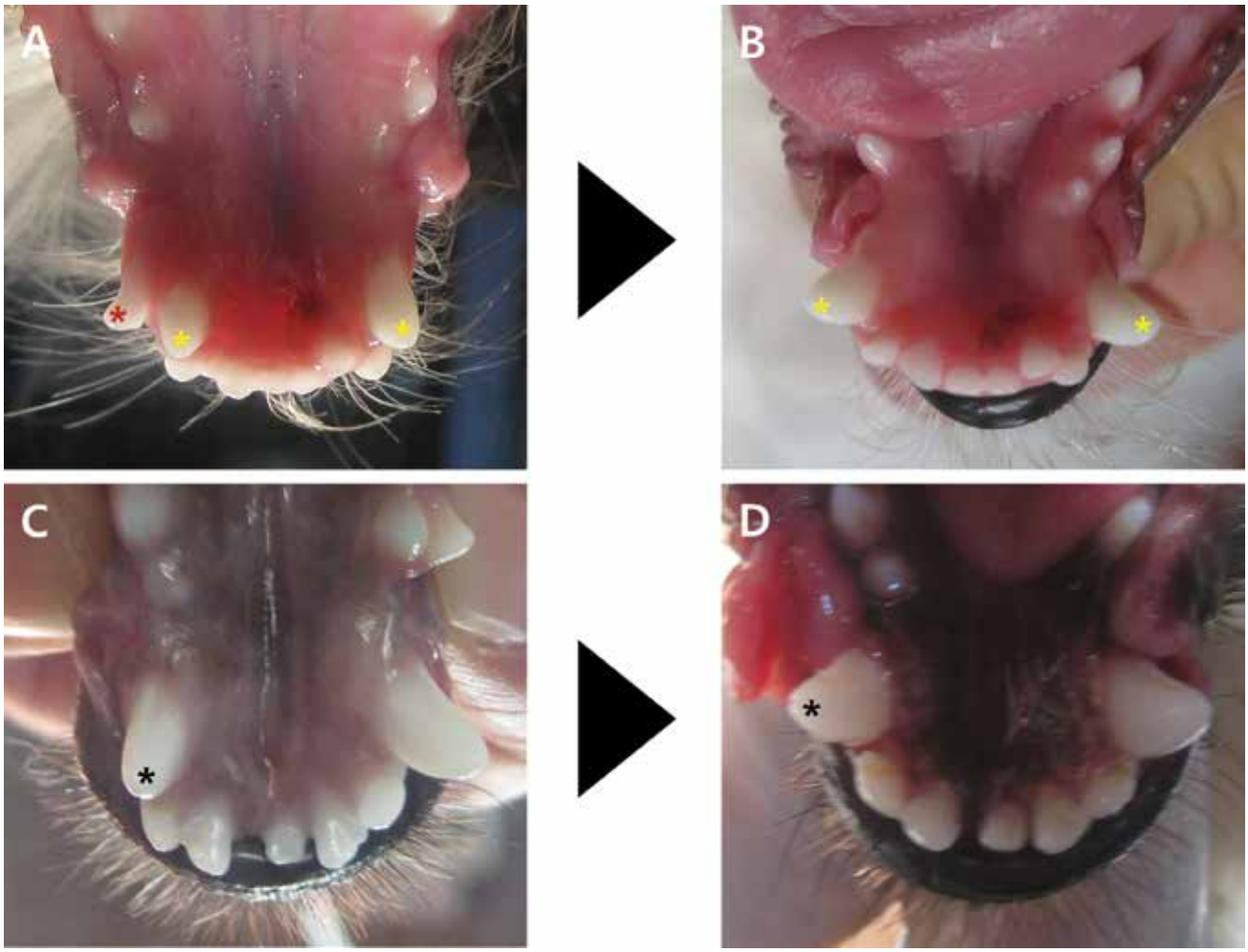

Figure 5. Examples of treatment of bilateral mandibular mesioverted canine teeth (A and B) and a unilateral mandibular mesioverted canine tooth (C and D) in dogs. A – The bilateral mandibular canine teeth were lingually displaced before the treatment (yellow asterisks). A persistent deciduous right mandibular canine tooth (red asterisk) was extracted. B – Both mandibular canine teeth were labially tipped 1 y after appliance removal (yellow asterisks). C – The right mandibular canine tooth was lingually displaced before treatment (black asterisk). D – The right mandibular canine tooth was labially tipped 1 y after appliance removal (black asterisk). The left mandibular canine tooth did not move in any direction during treatment.

deciduous teeth or inadequate size of the diastema between the maxillary third incisors and canine teeth may render ball therapy ineffective (4,12,20).

Other orthodontic treatment modalities for correction of LMC, including inclined bite planes, have been described. Several of these techniques rely on direct fabrication of the inclined bite planes in an animal's mouth, without requiring dental laboratories, thus reducing costs.

Ulbricht *et al* described a direct acrylic inclined bite plane technique (9). They applied a flowable composite directly onto the maxillary canine tooth and the third incisor tooth and then fabricated and polished it intraorally. Intraoral manipulation is technique-sensitive and may cause iatrogenic trauma to gingiva, palate, and teeth when performed by an inexperienced practitioner (*e.g.,* veterinarian). Furthermore, narrow gaps between the composite and gingiva and the unpolished surface facing the gingiva can promote food and plaque accumulation. Therefore, Furman *et al* suggested a variation to the acrylic inclined bite plane application (13). Their technique applied a flowable composite directly on the maxillary canine tooth and third incisor tooth, without etching. After curing, they removed it using a luxator to shape and polish it extraorally. The appliance was placed back on the teeth after fabrication, and the contact point plus occlusion were evaluated. Teeth were etched with 37% phosphoric acid, and the appliance was firmly bonded with light-curing adhesives. This technique had several advantages compared to that described by Ulbricht *et al* (9). The appliance could be easily designed intraorally and then fabricated and polished extraorally, reducing the possibility of trauma to oral structures. This technique also facilitated creation of a space between the appliance and mucosal surface, to facilitate home care. Before the final adhesion with light-curing adhesives, multiple adjustments and corrections can be easily made. The film thickness of the bonding agent can maximize bonding. Although this technique has many advantages, intraoral procedures involving direct application of flowable composite require operator skill, especially in small-breed dogs with small mouths and teeth. If excessive acrylic resin is applied,

considerable time and labor are required to shape the appliance properly.

Recently, Haggerty *et al* suggested an alternative method using light-cured acrylic denture base material with multiple, customized segments to construct an inclined bite plane appliance of the desired shape and size (21). This has the advantage of creating a more applicable and sophisticated device than the traditional method, using a flowable self-curing bis-acryl composite material. However, achieving the required shape and smooth finish intraorally requires considerable experience and skill, similar to the other techniques described.

The authors suggest a new method that overcomes the noted challenges of handling flowable acrylic resins, providing all the advantages of extraoral fabrication described by Furman *et al* (13). In this technique, the monomer and polymer are mixed in a dappen dish and, after achieving a doughy consistency, transferred to the target area. Unlike viscous acrylic resins used in previously described techniques, this can be easily shaped using fingertips and other tools (*e.g.,* spatula and scissors). This process can reduce the failure rate compared to the existing method using low viscosity and flowable composite, and generate a more acceptable primary form for small-breed dogs. An additional benefit of this resin for fabrication of inclined bite planes is acceleration of the curing process by dipping it in warm water (for 30 s to a few minutes, in our experience).

A core build-up resin was chosen as an adhesive for the appliance. Core build-up refers to placement of a restoration on a severely damaged tooth. Core build-up materials should have sufficient compressive strength to protect the remaining tooth from fracture and various intraoral forces. They also should have sufficient strength to prevent dislodgment during function; biocompatibility with surrounding tissues; ease of manipulation; adhesion to tooth structures, pins, and posts; and dimensional stability. Resin-based core build-up materials met most of these demands (22). Their bond-strength performance is much better than that of flowable composite resin, and their shear bond strength is somewhat greater than that of autopolymerized acrylic resin (23,24). Here, a blue-colored resin was used to enhance the contrast with the tooth surface, reducing the risk of iatrogenic damage to teeth during appliance removal.

In 4 cases, the diastema between the maxillary third incisor tooth and the maxillary canine tooth was not wide enough to accommodate the LMC. Sufficient maxillary space should be gained before the mandibular canine tooth is moved or concurrent with its movement. The acrylic inclined bite plane is not optimal for multi-directional movement such as mesiobuccal movement of the mandibular canine tooth. It also cannot provide distal movement of the maxillary canine tooth because it is attached not only to the maxillary canine tooth but also to the third incisor tooth (16). Therefore, the maxillary canine tooth was first tipped distally using the elastic chain appliance to provide enough space for the LMC to move into its correct position. An elastic chain is clinically used to correct mesioverted maxillary canine teeth (12). If the maxillary space requires only slight expansion, a cast metal telescoping inclined plane can be considered. A cast metal telescoping inclined plane is only attached to the maxillary canine tooth; therefore, it allows for slight distal tipping of the maxillary canine tooth during movement of the mandibular canine tooth through contact with the appliance and the mandibular canine tooth (16,18).

An inclined bite plane provides an intermittent force on the target tooth, as the canine cusp can only contact the bite plane when the mouth is closed. If the dog feels discomfort during biting, it will adjust its biting force and try to reduce the stress on the periodontal tissue around the target tooth. Therefore, the amount of force applied to the tooth can be "self-regulated." This self-adjustable intermittent force can minimize pain associated with orthodontic movement and can generate a light tipping force to move the crown and root of the mandibular canine tooth in an opposite direction with minimal complications (4,19). If a heavy force is applied, the apical blood supply can be compromised and sterile necrosis may occur in the compressed periodontal ligament area, resulting in delayed tooth movement and "undermining resorption" (9,25). In the case of a unilateral lingually displaced mandibular canine tooth, an inclined bite plane should be made bilaterally. An inclined bite plane on the normal side can work to prevent shifting of the mandible and reduce stress on the temporomandibular joint during mastication (8). Bilateral inclined bite planes also enable balanced maxillary growth (19).

In this study, all 8 dogs were examined 1 y after appliance removal, and all mandibular teeth were maintained in the desired positions. Five dogs were reevaluated after another 2 to 4 y, and all mandibular canine teeth were still well retained.

Home care and supervision are essential to optimize outcomes. Appliances located in the oral cavity create an environment that easily allows food remnants, debris, and plaque to accumulate. Therefore, owners need instructions regarding proper home oral-hygiene techniques to minimize periodontal disease during treatment (13). Daily brushing and oral rinsing with 0.12% chlorhexidine around the appliances can control gingival inflammation. Furthermore, these dogs should be restricted from playing with chew toys or chewing hard materials during orthodontic treatment (9).

As mentioned, the previously introduced acrylic inclined bite plane techniques require advanced skill due to the viscous consistency of the materials and limited space for intraoral shaping and polishing of the appliances. In contrast, the method proposed in this study used doughy acrylic resin that required less technical skill and experience to create an intraoral initial form while allowing the convenient, extraoral fabrication of an accurate and smooth appliance. This resulted in enhanced oral hygiene and reduced patient discomfort. The main limitation of this study was the small number of dogs involved. It was also limited to small-breed dogs, which does not imply the appliance would not be effective if used in a large-breed dog. Further studies are needed to evaluate the effectiveness of this method on a larger cohort, ideally including also medium- and large-breed dogs.

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Case Report Rapport de cas

Combined cholecystotomy, retrograde hydropulsion, and choledochal stenting to treat extrahepatic biliary tract obstruction in 3 cats

Joséphine Roels, Mark Kim, Jean-Luc Cadoré, Céline Pouzot-Nevoret, Thibaut Cachon

Abstract — Extrahepatic biliary tract obstruction (EHBO) is uncommonly encountered in cats. Surgical treatment aims to decompress the biliary tract and insure bile duct patency. In veterinary medicine, cholecystotomy is not widely used in practice. The objective was to describe the use of cholecystotomy, retrograde hydropulsion of choleliths, and choledochal stenting to remove choleliths from the extrahepatic biliary tract back in the gallbladder.

Three adult domestic shorthair cats were presented with anorexia, lethargy, and vomiting. Serum biochemistry revealed hyperbilirubinemia and increased hepatic enzymes. Abdominal ultrasonography showed evidence of EHBO requiring surgical intervention. Choleliths were localized in the proximal and middle portions of the common bile duct (CBD) in the first case, in the distal portion of the CBD and within the major duodenal papilla in the second case, and in the middle and distal portions of the CBD in the third case. Cholecystotomy was followed by retrograde hydropulsion of the choleliths into the gallbladder, after which choledochal stenting was performed. Complications were defined as major when requiring additional medical or surgical treatment, or minor when not.

Three major complications were reported. In 2 cases, severe anemia requiring blood transfusion occurred 24 h postoperatively; in 1 case, EHBO recurrence was encountered 41 d postoperatively. All cats were discharged within 4 d following surgery. Two cats were still alive at 12 and 14 mo after surgery, respectively. In the last case, owners refused revision surgery and the cat was euthanized.

Key clinical message:
Cholecystotomy combined with retrograde hydropulsion of choleliths permitted removal of choleliths and decompression of the biliary tract in 3 cats. Major complications included severe anemia and EHBO recurrence.

Résumé — Cholécystotomie combinée, hydropulsion rétrograde et pose de stent cholédocien pour traiter l'obstruction des voies biliaires extra-hépatiques chez 3 chats. Les obstructions biliaires extra-hépatiques (OBEH) sont peu fréquentes chez le chat. Le traitement chirurgical vise à lever l'obstruction et s'assurer de la perméabilité des voies biliaires. En médecine vétérinaire, la cholécystotomie est une technique peu pratiquée. L'objectif de ce rapport de cas était de décrire l'utilisation de la cholécystotomie, de l'hydropulsion rétrograde des cholélithes et d'une prothèse endoluminale cholédoquale (PEC) pour repousser les cholélithes présents dans les voies biliaires extrahépatiques dans la vésicule biliaire (VB).

Trois chats européens adultes ont été présentés pour anorexie, léthargie et vomissements. La biochimie sérique a révélé une hyperbilirubinémie et une augmentation des enzymes hépatiques. L'échographie abdominale a mis en évidence une OBEH nécessitant une intervention chirurgicale. Les cholélithes étaient situés dans la portion proximale et moyenne du canal cholédoque pour le premier cas; dans la portion distale et la papille duodénale majeure dans le second cas; dans la portion moyenne et distale pour le troisième cas. Une cholécystotomie a été suivie d'une rétro-hydropulsion des cholélithes dans la VB, puis une PEC a été placée. Les complications ont été définies comme majeures lorsqu'elles nécessitaient un traitement médical ou chirurgical supplémentaire, ou mineures lorsqu'elles n'en nécessitaient pas.

Trois complications majeures ont été rapportées : chez 2 cas, une anémie sévère a été observée 24 h après l'intervention, nécessitant une transfusion sanguine; chez un cas, une récidive d'obstruction biliaire a eu lieu à 41 jours postopératoire. Tous les patients sont sortis de l'hôpital dans les 4 jours suivant l'opération. Deux cas étaient encore en vie 12 et 14 mois après l'intervention. Pour le dernier cas, la seconde chirurgie a été refusée par les propriétaires et le chat a été euthanasié.

University of Lyon, VetAgro Sup, Veterinary Campus of Lyon, Department of Companion Animals, 1 Av. Bourgelat, 69280 Marcy L'Étoile, France.

Address all correspondence to Dr. Joséphine Roels; email: josephine.roels@vetagro-sup.fr

Message clinique clé :
La cholécystotomie combinée à l'hydropulsion rétrograde des cholélithes a permis le retrait de cholélithes obstructives (dont certaines distales) et la décompression du tractus biliaire chez 3 chats. Les complications majeures incluaient une anémie sévère et une récidive d'obstruction biliaire.

(Traduit par les auteurs)

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Extrahepatic biliary tract obstruction (EHBO) is infrequently encountered in cats and can be caused by inflammatory conditions such as pancreatitis, cholangiohepatitis, cholecystitis, cholelithiasis, or neoplasia (1–5). Cholelithiasis accounts for < 1% of EHBO in cats (1–4). Surgical treatment of EHBO aims to relieve the obstruction and ensure bile duct patency (2,4–12). Cholecystoenterostomy (CCE) is the most frequently described procedure in cats with variable short- and long-term complications (3,5–7,13,14). Mortality rates for biliary tract surgery for non-neoplastic causes in cats range from 40 to 60% (3,5–7,13,14). These reports predominantly involve CCE. Cholecystotomy, cholecystectomy, choledochotomy, and sphincterotomy are infrequently described (4,5,11,12,15–18). Choledochal stenting is a well-established, effective option in cats with EHBO showing few technical complications and potential for prolonged survival (2–5,10).

To our knowledge, there are limited reports of cholecystotomy in cats for the management of EHBO, and retrohydropulsion of choleliths from the common bile duct (CBD) in the gallbladder (GB) is not clearly described (2,5,8,10).

Case descriptions

Case 1

A 15-year-old spayed female domestic shorthair cat was presented to the referring veterinarian with a 2-day history of progressive lethargy, anorexia, and vomiting. Blood biochemistry revealed marked elevations in alanine aminotransferase (ALT: 693 UI/L) and gamma glutamyl transferase (GGT: 10 U/L), severe hyperbilirubinemia (total bilirubin: 50.1 μmol/L), and mild azotemia (urea: 11 mmol/L, creatinine: 156 μmol/L); results from routine hematology testing were unremarkable (Table 1). The cat was referred to our institution for further investigation. On initial examination, the cat was mildly icteric, 5% dehydrated, and had a Grade-II/VI parasternal systolic heart murmur. Blood electrolytes showed moderate hypokalemia, and blood gas and coagulation times were within reference intervals (Table 1). Abdominal ultrasonography showed a distension of the GB (17 × 34 mm, with a wall thickness of 1.1 mm) associated with multiple choleliths in the GB (~3 mm in diameter). Multiple obstructive choleliths (ranging from 4.8 to 9.9 mm in diameter) were visualized in the proximal and middle portions of the CBD, with a distension of the CBD (maximum diameter of 13.7 mm, and a wall thickness of 0.8 mm) anterior to the obstruction site and normalization of its diameter posterior to the obstruction site (2.8 mm). Intrahepatic choleliths were also identified. The pancreas was increased in size, heterogeneous, and hypoechogenic. The duodenal wall was moderately thickened.

Extrahepatic biliary tract obstruction was diagnosed, and the cat was stabilized preoperatively for 24 h with IV fluids (lactated Ringer's solution: AquPharm; Axience, Pantin, France), methadone (0.1 mg/kg, IV, q4h), amoxicillin-clavulanic acid (20 mg/kg, IV, q8h), and pantoprazole (1 mg/kg, IV, q24h).

During the exploratory laparotomy, the CBD and the GB were markedly dilated (Figure 1 A). The GB was packed off with sterile laparotomy sponges. A 2-centimeter incision was made in the apex of the GB wall and the bile was suctioned. Stay sutures were placed across the margins of the incision. The CBD was followed normograde to identify the major duodenal papilla. A 2-centimeter duodenotomy was made to expose and then catheterize the major duodenal papilla with a 4-French urinary catheter (ureteral catheter, cylindrical; Teleflex, Limerick, Ireland) (Figure 1 B). The CBD was flushed retrograde with 0.9% NaCl and multiple choleliths removed *via* the cholecystotomy site (Figure 1 C). The cholecystotomy was then closed in a single-layer continuous pattern, followed by a single-layer inverting Cushing pattern, with 4-0 monofilament absorbable poliglecaprone 25 sutures (Advantime; Peters Surgical, Boulogne-Billancourt, France) (Figure 1 D). A choledochal stent was placed using the terminal portion of a 4-French urinary catheter (ureteral catheter, cylindrical; Teleflex) in the CBD, across the major duodenal papilla, and sutured to the duodenal mucosa with 2 simple interrupted sutures, using a 4-0 monofilament absorbable poliglecaprone 25 suture material (absorbable suture with 50% tensile strength at 1 to 2 wk: Advantime; Peters Surgical) (Figure 1 D). The abdominal wall was closed routinely. Samples were submitted for histopathology of the GB wall and liver and bacterial culture of the bile (Table 1).

Twenty-four hours postoperatively, the cat developed severe anemia [hematocrit: 17% (reference: 24 to 45%)] requiring a blood transfusion (blood type-matched and cross-matched stored whole blood, 10 mL/kg). Serum biochemistry showed persistent severe hyperbilirubinemia (total bilirubin: 79 μmol/L) and elevation of liver enzymes (ALT: 434 UI/L, GGT: 22 U/L). A nasogastric feeding tube was placed due to persistent anorexia, and enteral nutrition was given for 2 d after surgery. Other postoperative treatments included morphine (0.2 mg/kg, IV, q4h), amoxicillin-clavulanic acid (20 mg/kg, IV, q8h), maropitant citrate (1 mg/kg, IV, q24h), pantoprazole (1 mg/kg, IV, q24h), ursodeoxycholic acid (10 mg/kg, PO, q24h), and IV fluids (lactated Ringer's solution). Results from bacteriological assessment were positive *(Staphylococcus simulans)*. The cat continued receiving amoxicillin-clavulanic acid (20 mg/kg, PO, q12h) and ursodeoxycholic acid (PO) for 4 wk.

The cat was discharged 4 d after surgery. Abdominal ultrasound before discharge revealed mild dilation of the

Table 1. Additional biologic findings at presentation and postoperative histopathology results for the 3 cats.

	Case 1	Case 2	Case 3
Additional blood analysis at presentation	Moderate hypokalemia (K = 3.3 mmol/L) Complete blood (cell) count, blood gas, and coagulation time normal	Moderate hypokalemia (K = 3.2 mmol/L) Moderate leukocytosis (WBC = 36.3 × 10^9/L) Acidemia (pH = 7.1) Complete blood (cell) count unremarkable	Marked hypokalemia (K = 2.9 mmol/L) Moderate anemia (Ht = 24%) Coagulation time normal
Histopathology	Moderate diffuse neutrophilic cholangiohepatitis, suppurative and necrotic diffuse acute cholangitis, evidence of diffuse fibrinous and suppurative peritonitis	Portal hepatitis, severe diffuse neutrophilic cholangitis with a severe necrotic and fibrinous inflammation of the gallbladder wall	Severe diffuse neutrophilic cholangiohepatitis, diffuse acute cholangitis

Reference range values: Potassium (K), 3.7 to 5.8 mmol/L; white blood cells (WBC), 5.5 to 19.5 × 10^9/L.

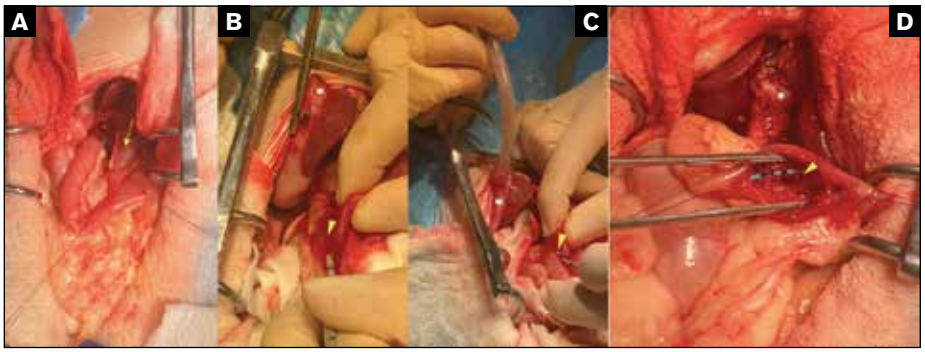

Figure 1. A – The common bile duct (CBD) was markedly dilated (arrow). Traction sutures were placed on the duodenum. B – Duodenotomy allowed CBD catheterization using a urinary catheter (arrowhead). Cholecystotomy at the gallbladder apex (asterisk); flushing of black choleliths. C – Retrograde hydropulsion with sterile saline was performed through the urinary catheter (arrowhead) with concurrent suction, to remove choleliths from the CBD, cystic duct, and gallbladder (asterisk). D – Cholecystotomy was closed with a double-layer continuous suture pattern (asterisk) and a choledochal stent (arrowhead) was placed (terminal portion of 4-French urinary catheter), fixed to the duodenal mucosa with 2 sutures.

CBD (5.7 mm diameter, and a wall thickness of 1.5 mm) without evidence of obstruction. The choledochal stent was still in place. Moderate elevation of liver enzymes (ALT: 417 UI/L) and hyperbilirubinemia (total bilirubin: 87.1 μmol/L) were still present while the cat was eating and recovering.

Short-term follow-up at 11 d after surgery showed normalization of liver enzymes and total bilirubin (ALT: 89 UI/L, total bilirubin: 9.2 μmol/L). Abdominal ultrasound identified no evidence of EHBO, with a GB wall thickness of 1.7 mm and a CBD diameter of 4.3 mm. The choledochal stent remained *in situ*.

The cat was presented 9 mo after surgery with acute onset lethargy, anorexia, and vomiting. A presumptive diagnosis of pyelonephritis was made based on blood tests and abdominal ultrasound. Liver enzymes and total bilirubin remained normal (ALT: 71 UI/L, total bilirubin: 2.5 μmol/L) and there was no evidence of EHBO, with a GB wall thickness of 1.2 mm, a cystic duct diameter of 1.1 mm, and a CBD diameter of 4.5 mm. The cat was medically managed successfully and discharged 3 d later.

Case 2

A 14-year-old neutered male domestic shorthair cat was presented with lethargy, anorexia, polydipsia, polyuria, and vomiting. On initial examination, the cat showed moderate depression, had a severely icteric mucous membrane, and was 8% dehydrated. Blood biochemistry revealed marked elevations in ALT (410 UI/L) and GGT (16 U/L), severe hyperbilirubinemia (total bilirubin: 116.1 μmol/L), and mild azotemia (urea: 14.4 mmol/L, creatinine: 167 μmol/L); routine hematology testing showed moderate leukocytosis (Table 1). Blood electrolytes showed moderate hypokalemia, and blood gas revealed acidemia (Table 1). Abdominal ultrasonography showed distension of the GB (12 × 25 mm, with a wall thickness of 2.8 mm) associated with multiple choleliths in the GB (2.4-millimeter diameter). Multiple obstructive choleliths were localized in the distal portion of the CBD and within the major duodenal papilla (ranging from 2.5 to 4 mm in diameter), with a distension of the CBD (maximum diameter of 10.6 mm, and a wall thickness of 1.8 mm). The pancreas was increased in size, heterogeneous, and hypoechogenic. The duodenal wall was moderately thickened.

Extrahepatic biliary tract obstruction was diagnosed and the cat was stabilized before surgery as described for Case 1. The exploratory laparotomy followed the same procedure described for Case 1.

Twenty-four hours postoperatively, serum biochemistry showed mild hyperbilirubinemia (total bilirubin: 25 μmol/L),

decreased ALT value (ALT: 287 UI/L), and normalization of renal parameters. A nasogastric feeding tube was required for 2 d postoperatively. Other postoperative treatments included morphine (0.2 mg/kg, IV, q4h), amoxicillin-clavulanic acid (20 mg/kg, IV, q8h), maropitant citrate (1 mg/kg, IV, q24h), pantoprazole (1 mg/kg, IV, q24h), ursodeoxycholic acid (10 mg/kg, PO, q24h), and IV fluids (lactated Ringer's solution). Results from bacteriological assessment were positive *(Escherichia coli)*. The cat continued receiving amoxicillin-clavulanic acid (20 mg/kg, PO, q12h) and ursodeoxycholic acid (PO) for 4 wk. The cat was discharged 72 h postoperatively.

Two weeks postoperatively, the cat showed vomiting. Moderate extrahepatic biliary tract dilatation was identified on abdominal ultrasound, with a GB wall thickness of 1.9 mm and a CBD diameter of 4.6 mm. There was no evidence of EHBO or choleliths in the GB or CBD. Serum biochemistry was within normal limits (ALT: 215 UI/L, total bilirubin: 4 μmol/L). Choledochal stenting was not identified on ultrasound.

A telephone interview was conducted 1 y postoperatively with the cat's owners, who reported the cat had a good quality of life with no clinical signs.

Case 3

A 10-year-old neutered male Persian cat was presented with lethargy, anorexia, and vomiting, along with a 1-month history of cholecystitis treated by ursodeoxycholic acid (10 mg/kg, PO, q24h), marbofloxacin (4 mg/kg, PO, q24h), and prednisolone (2 mg/kg, PO, q24h). On initial examination, the cat showed jaundice, was 5% dehydrated, and had a Grade-II/VI parasternal systolic heart murmur and hyperthermia.

Blood biochemistry revealed severe hyperbilirubinemia (total bilirubin: 142 μmol/L), marked elevation in ALT (ALT: 357 UI/L), mild azotemia (urea: 21.7 mmol/L, creatinine: 167 μmol/L), marked hypokalemia, and moderate anemia (Table 1). Coagulation times were within reference intervals (Table 1). Abdominal ultrasonography showed a distension of the GB (13 × 27 mm, with a wall thickness of 1 mm) with a large quantity of biliary sludge within the GB, partially adherent to its wall. Sludge was also visualized in the middle and distal portions of the CBD over 2 cm before the duodenal papilla. Distension of the CBD (with a maximum diameter of 11 mm) was seen before the obstruction site. The pancreas was increased in size, heterogeneous, and hypoechogenic.

Extrahepatic biliary obstruction was diagnosed and the cat was stabilized for 24 h before surgery as described. During the exploratory laparotomy, the CBD and the GB were markedly dilated. The same procedure was performed and numerous millimetric choleliths were removed from the GB.

Twenty-four hours after surgery, the cat developed severe anemia [hematocrit: 10.5% (reference: 24 to 45%)] requiring a blood transfusion (blood type-matched and cross-matched stored whole blood, 10 mL/kg). Serum biochemistry showed mild hyperbilirubinemia (total bilirubin: 66 μmol/L) and decreased ALT value (ALT: 272 UI/L). A nasogastric feeding tube was placed for 2 d after surgery. Other postoperative treatments included morphine (0.2 mg/kg, IV, q4h), marbofloxacin (4 mg/kg, IV, q24h), maropitant citrate (1 mg/kg, IV, q24h), pantoprazole (1 mg/kg, IV, q24h), ursodeoxycholic acid (10 mg/kg, PO, q24h), and IV fluids (lactated Ringer's solution). Results from bacteriological assessment were positive *(Serratia marcescens)*. The cat continued receiving marbofloxacin (4 mg/kg, PO, q24h) for 4 wk, combined with ursodeoxycholic acid and prednisolone (2 mg/kg, PO, q24h). The cat was discharged 3 d after surgery.

Short-term follow-up at 17 d after surgery showed a discrete hyperbilirubinemia (total bilirubin: 7.4 μmol/L) and increased ALT value (ALT: 482 UI/L), with persistent dysorexia but no evidence of EHBO on abdominal ultrasound (maximum GB wall thickness of 1.7 mm, CBD diameter of 5 mm). The choledochal stent was not seen.

The cat was presented at 22 d after surgery with a complication related to misplacement of an esophagostomy tube into the duodenum by the referring veterinarian, resulting in a secondary intussusception. Elevation of total bilirubin (total bilirubin: 14 μmol/L) and ALT (ALT: 692 UI/L) were noted at that time. Exploratory laparotomy was required to remove the feeding tube and treat the intussusception. During the surgery, the CBD appeared mildly dilated and the cholecystotomy site was intact (Figure 2 A, B). Digital pressure was applied over the GB and showed satisfactory patency of the CBD.

The cat was presented at 41 d after surgery for recurrence of clinical signs (persistent anorexia, vomiting). Abdominal ultrasound and clinical pathology findings revealed EHBO recurrence. The cystic duct and the CBD had diameters of 8 mm and 11 mm, respectively; sludge was present in the distal portion of the CBD; and there was hyperbilirubinemia (total bilirubin: 76.8 μmol/L) and markedly increased ALT value (ALT: 1073 UI/L). A CEE was recommended but refused by the owners. The cat was euthanized.

Discussion

This report documents 3 cases of EHBO that were successfully managed by combined cholecystotomy, retro-hydropulsion of choleliths, and choledochal stenting, with 3 major complications. Two major complications occurred in the immediate postoperative period (severe anemia requiring blood transfusion) and 1 was encountered in the short-term follow-up (EHBO recurrence). At the time this report was written, 2 cats were still alive and 1 was euthanized due to EHBO recurrence.

Biliary tract diversion by CCE is the traditional procedure performed in cats to treat EHBO (1,3–6). However, considerable morbidity and mortality are associated with this technique. The short-term mortality rate ranges from 36 to 57% and a 6-month survival rate of 42.9% was reported (1,6,8,13,14). Retrospective studies reported long surgery and anesthesia times, presumably due to the complexity of the technique (1,6,8,13,14). Prolonged anesthesia increases the risk of perioperative complications, including hypotension (1,14,18). As well, the major anatomical modifications required for the CCE predispose patients to complications such as suture site dehiscence, bile leakage, and bile peritonitis, which is a life-threatening condition. In the medium-to-long term, stoma stricture, chronic or recurrent ascending bile duct infection, recurrence of EHBO, pancreatic insufficiency, and chronic vomiting were also reported (1,3,14).

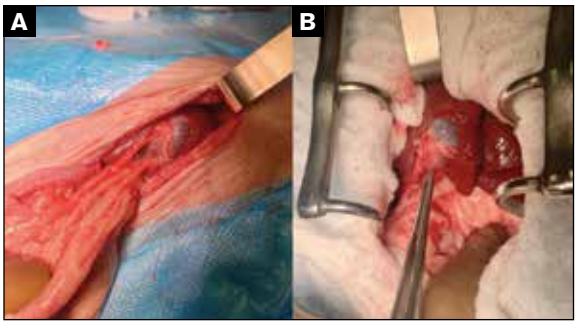

Figure 2. Intraoperative aspect of the cholecystotomy site at 22 d after surgery in Case 3. A – Omental adhesions were visualized on the previous cholecystotomy site. B – The gallbladder wall presented a normal aspect.

We chose to combine cholecystotomy, retro-hydropulsion of choleliths, and choledochal stenting for feline EHBO management. This approach may have lower morbidity and mortality rates than CCE (1,6,8,13,14). This procedure had multiple advantages. First, retrograde hydropulsion of choleliths *via* the cholecystotomy site allowed preservation of the GB and CBD. Preserving the biliary tract leaves room for revision surgery in case of recurrent EHBO. Also, we determined that this procedure required less time than CCE, likely reflecting its lower level of complexity. Thus, it could be performed in an emergency situation, as early surgical decompression of the EHBO improves outcomes and reduces the risk of recurrence (1,2,10). Cholecystotomy and choledochal stenting have already been described for managing EHBO (2,10). The original objective of our report was to demonstrate the association with retrograde hydropulsion to push back obstructive choleliths (from the proximal and middle portions of the CBD in Case 1, from the major duodenal papilla and distal portion of the CBD in Case 2, from the middle and distal portions of the CBD in Case 3) in the GB and remove them through the cholecystotomy site.

Several surgical techniques can be used to treat EHBO depending on the location, size, and number of choleliths in the extrahepatic bile ducts. Due to the lack of studies involving a large number of cases and comparing options, the superiority of one technique over the others cannot be demonstrated (1,4,5,13,14). Choleliths in the GB can be asymptomatic in cats, and therefore do not require surgical treatment (1,3,5). Obstructive choleliths are generally located in the CBD (1,3,5). If a cholelith is located in the proximal or middle portion of the CBD, a choledochotomy may be considered by making a small incision along the CBD over the stone to remove it (5,12). However, due to the small diameter of the CBD, the inflammatory reaction from cholelith passage, and the sutures required, choledochotomy may be associated with a high risk of postoperative stenosis despite choledochal stenting (5,12). If a cholelith is located in the distal portion of the CBD or in the major duodenal papilla, a sphincterotomy has been proposed (16,17). This technique consists of increasing the opening of the major duodenal papilla by making a small, dorsomedial incision over the major duodenal papilla through a duodenotomy and removing choleliths from this larger opening. Choledochal stenting is recommended with this procedure. Sphincterotomy has the advantage of leaving the GB intact, particularly when its wall is severely inflamed. Nevertheless, it is mainly indicated to remove a small number of choleliths (5,16,17).

Choledochotomy and sphincterotomy were not indicated in our 3 cases. Indeed, in Cases 1 and 3, multiple choleliths (Case 1) and a large quantity of biliary sludge (Case 3) were localized along the CBD (in the proximal and middle portions in Case 1; in the middle and distal portions in Case 3). Sphincterotomy or choledochotomy alone would not have allowed total removal of the obstructive material due to the large quantity. In Case 2, choleliths were identified in the major duodenal papilla and in the distal portion of the CBD. Due to their large number and size (up to 9.9 mm), we believe that sphincterotomy would not have been sufficient to remove all the stones. Moreover, numerous choleliths and/or sludge were present within the GB in all cases and could have migrated after the procedure, causing further obstruction. Thus, retro-hydropulsion was an alternative option. We performed choledochal stenting in all cases due to its noninvasive nature, providing reestablishment of choledochal bile flow and showing good efficacy in the treatment of reversible CBD obstructions (2,3,10,11). The placement of the stent is relatively simple and maintains a sufficient opening of the duodenal papilla, subject to pro-inflammatory intraoperative manipulation, to facilitate the elimination of debris possibly present in the bile ducts after surgery (2,3,10,11).

Disadvantages of the procedure used in our 3 cases include failure to remove all choleliths, which may ultimately require biliary tract bypass surgery; and inadvertent retro-hydropulsion of the choleliths into the intrahepatic bile ducts, which may increase risk of re-obstruction in the postoperative period. Furthermore, preserving the GB may permit recurrent cholelithiasis, which is a major risk factor for EHBO (1–5). However, the fundamental principle of the described approach (cholecystotomy and retro-hydropulsion of choleliths) showed less intra- and postoperative risk *versus* an increased risk of postoperative recurrence (4,5,18).

To prevent cholelithiasis recurrence in cats, cholecystectomy has been proposed based on human management of symptomatic cholelithiasis (4,5,11,15). However, in veterinary surgery, many choleliths are identified not only in the GB, but also in the cystic duct, the CBD, and the hepatic bile ducts. Cholecystectomy in the treatment of symptomatic cholelithiasis (stones in the GB) must thus be used with great care and is generally recommended to remove the main source of their production, in the absence of EHBO and in the absence of choledocholithiasis particularly. Cholecystectomy may be technically less demanding than cholecystotomy, but in our opinion, it should be performed sparingly and in a very small number of cases. It has been described in 23 cats with overall satisfactory outcomes, but with a limited number of cases that showed suspected EHBO recurrence in the medium-to-long term (15). One can assume that, even if the GB is the main site of cholelith formation (secondary to biliary stasis), other choleliths might form in the intrahepatic bile ducts and cause EHBO recurrence. The treatment of EHBO in cats that have

already undergone cholecystectomy leaves few possibilities, as bile duct bypass is no longer possible (15).

In our report, severe anemia occurred in the immediate postoperative period in 2 out of the 3 cases and required blood transfusion. This major complication cannot readily be explained, as only 1 cat was anemic before surgery and no significant bleeding occurred during the procedure. The anemia might have been related to EHBO and liver dysfunction secondary to a marked inflammation, as previously reported, and not to the surgical technique, unlike CEE or cholecystectomy, where dissection of the GB from its vesicular fossa may lead to significant bleeding (1,7,8).

Finally, although cholecystotomy is historically associated with a high rate of suture dehiscence, we did not encounter this complication in any of our cases (3,11). As previously recommended, cholecystotomy is contraindicated in the presence of GB wall necrosis (3,11).

Limitations of this case report are related to the small number of cases included. In addition, we did not reevaluate for persistent choleliths in the immediate postoperative period. Perioperative abdominal imaging could be obtained to ensure removal of all choleliths.

In conclusion, cholecystotomy with retrograde hydropulsion of choleliths and choledochal stenting to remove stones from the major duodenal papilla, the CBD, and the cystic duct back in the GB has not been clearly reported in small animals for EHBO. Here, we describe that this technique was feasible in 3 cases and permitted successful decompression of the biliary tract. Major complications were reported, with severe anemia in 2 cases and EHBO recurrence 41 d after surgery in 1 case. There were no complications related to the cholecystotomy site among these 3 cases. Larger studies are warranted to compare with existing protocols, which are associated with considerable complications and high mortality rates. CVJ

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Case Report Rapport de cas

Recognizing uterine torsion as a differential diagnosis in pregnant cats with severe anemia to provide appropriate and timely care in the absence of a definitive presurgical diagnosis

Shogo Kimura, WeiChun Huang, Elroy V. Williams, Kevin C. Cosford

Abstract — A pregnant female domestic longhair cat ~8 mo of age was referred to the Western College of Veterinary Medicine (Saskatoon, Saskatchewan) for a diagnostic evaluation of severe anemia (PCV: 10.8%) after a 2-day period of lethargy. A CBC, serum biochemistry profile, FeLV/FIV testing, and abdominal radiographs were completed and did not determine a cause for the anemia. Abdominal ultrasonography identified 1 viable and 6 nonviable and fetuses, anechoic fluid in the uterus, and a mild volume of peritoneal effusion. A whole-blood transfusion and C-section with ovariohysterectomy were performed even though a definitive presurgical diagnosis for the anemia had not yet been established. Exploratory surgery revealed a left uterine horn torsion with a necrotic base, severe congestion, and 7 nonviable fetuses. Following surgery, the queen made a full clinical recovery.

Key clinical message:
Uterine torsion can be easily overlooked as a cause of severe anemia due to the relative infrequency of this condition in cats and the low sensitivity of ultrasonography to provide a definitive presurgical diagnosis. Client communication must emphasize the need for a prompt surgical intervention to establish the diagnosis and to save the cat, despite poor rates of neonatal survival. Once the animal is stabilized after surgery, further diagnostic tests and procedures are indicated if the cause of anemia has not yet been identified.

Résumé — Reconnaître la torsion utérine comme un diagnostic différentiel chez les chattes gestantes souffrant d'anémie sévère afin de fournir des soins appropriés et opportuns en l'absence d'un diagnostic pré-chirurgical définitif. Une chatte domestique à poils longs, âgée d'environ 8 mois, a été référée au *Western College of Veterinary Medicine* (Saskatoon, Saskatchewan) pour une évaluation diagnostique d'anémie sévère (hématocrite : 10,8 %) après une période de léthargie de 2 jours. Une formule sanguine complète, un profil biochimique sérique, des tests FeLV/FIV et des radiographies abdominales ont été réalisés et n'ont pas permis de déterminer la cause de l'anémie. L'échographie abdominale a identifié 1 fœtus viable et 6 non viables, du liquide anéchoïque dans l'utérus et un léger volume d'épanchement péritonéal. Une transfusion de sang total et une césarienne avec ovariohystérectomie ont été réalisées même si le diagnostic pré-chirurgical définitif de l'anémie n'avait pas encore été établi. La chirurgie exploratoire a révélé une torsion de la corne utérine gauche avec une base nécrotique, une congestion sévère et 7 fœtus non viables. Après l'opération, la chatte s'est complètement rétablie cliniquement.

Message clinique clé :
La torsion utérine peut facilement être négligée comme cause d'anémie sévère en raison de la rareté relative de cette affection chez le chat et de la faible sensibilité de l'échographie pour fournir un diagnostic pré-chirurgical définitif. La communication avec le client doit souligner la nécessité d'une intervention chirurgicale rapide pour établir le diagnostic et sauver le chat, malgré de faibles taux de survie néonatale. Une fois l'animal stabilisé après la chirurgie, d'autres tests et procédures de diagnostic sont indiqués si la cause de l'anémie n'a pas encore été identifiée.

(Traduit par Dr Serge Messier)

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Department of Small Animal Clinical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan S7N 5B2.

Address all correspondence to Dr. S. Kimura; email: kshogo@upei.ca

Uterine torsion is considered a rare complication of mid- to late-term pregnancy in the cat (1–3). The available literature consists mainly of isolated case reports, making it an under-recognized condition. It is characterized as a twisting of the uterine body or horn perpendicular to its long axis; the etiology remains unclear (4–6). Most accessible reports document feline uterine torsion in mid- to late-gestation (1–3), but a few cases in non-gravid cats with or without pyometra have also been reported (6,7). Although physical activity, fetal movement, uterine contraction, lack of uterine tone, and rough handling have all been proposed to contribute to the condition of uterine torsion, the etiology is not known (4,7). In the case reports of non-gravid cats, it was proposed that an enlarged and flaccid uterus with stretched broad ligaments and ovarian pedicles was predisposed to torsion (6,7).

Previously reported clinical presentations of uterine torsion included acute onset of vulvar discharge, weakness, depression, anorexia, pallor, and hypothermia (1,8,9). Hematological findings may reveal severe anemia (8,9), as well as neutrophilia consistent with an inflammatory response (8). Disseminated intravascular coagulation can be seen, due to vascular stasis and tissue hypoxia or damage (8). Some reported metabolic abnormalities include marked elevation in ALT, hyperkalemia, and hyponatremia. The authors of those case reports considered the hyperkalemia a result of significant tissue death from ischemia and reduced renal excretion, and the hyponatremia a consequence of fluid pooling within the uterus (8). Abdominal ultrasonography did not diagnose the uterine torsion but identified mainly nonviable fetuses and minimal or small-volume abdominal fluid (8,9).

This report describes the case of a cat with severe anemia of unknown origin at an unknown stage of gestation that was ultimately diagnosed with uterine torsion. Presurgical imaging was nondiagnostic and the decision to perform a laparotomy was both diagnostic and therapeutic. The presence of marked anemia may suggest intra-abdominal hemorrhage due to uterine torsion in pregnant cats, despite a constellation of inconclusive clinical and diagnostic information. A traditional abdominal laparotomy will serve not only to facilitate an emergency C-section and ovariohysterectomy, but also to explore the abdomen, which will establish the diagnosis and thereby identify the source of intra-abdominal hemorrhage. If the cause of anemia is neither identified nor resolved after surgery, then further diagnostic testing and treatment protocols can be initiated once the queen is stabilized. It should be noted that, depending on the stage of pregnancy when uterine torsion occurs, many of the fetuses may be too premature to survive. The primary goal is ultimately to save the adult cat. Improving neonatal survival requires early intervention but is only possible if the fetuses are developed enough to potentially survive.

Case description

A pregnant female domestic longhair cat 8 mo of age was presented to a local emergency hospital after a 1-day period of lethargy and anorexia. One episode of vomiting had also been noted the previous day without any history of toxin or foreign material ingestion. The cat was found as a stray and adopted

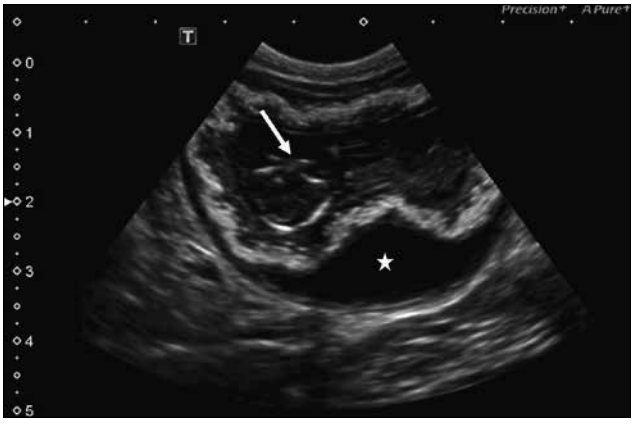

Figure 1. Ultrasonographic image of a nonviable fetus (arrow) within the markedly distended feline uterus containing anechoic uterine fluid (star).

3 wk before presentation, at which time routine preventative care was provided by the primary veterinarian. It was also strongly suspected that the cat was pregnant. Five days before referral, radiographs were taken by the primary veterinarian to determine the pregnancy status. The uterus was enlarged and distended but there were no mineralized fetal skeletons detected. Feline fetal skeletons are detectable radiographically within 25 to 29 d of parturition (10). Given that the mean length of gestation in *Felis catus* is 65 d with a range of 57 to 72 d, the cat in this case was estimated to be early- to mid-gestation (11,12). Note that the onset of fetal skeletal radio-opacity may not be sufficient to accurately stage a pregnancy, so the exact term cannot be provided in this case.

On presentation at a local emergency hospital, 1 d before referral to the Western College of Veterinary Medicine (WCVM), the cat was determined to be hypothermic (35.5°C) with a prolonged capillary refill time (2 to 4 s), which became normal after receiving the following treatment overnight: ampicillin (22 mg/kg, IV, q8h), maropitant (1 mg/kg, IV, q24h), pantoprazole (1 mg/kg, IV, q12h), and IV fluid therapy (lactated Ringer's solution) at maintenance rate. A CBC revealed a moderately regenerative severe anemia characterized by a hematocrit of 10.8% [reference range (RR): 30.3 to 52.3%] with 2.7% reticulocytes and an absolute reticulocyte count of 86.1 K/μL (RR: 3 to 50 K/μL). Leucocyte parameters showed neutrophilia (20.90×10^9/L; RR: 2.30 to 10.29×10^9/L), consistent with an inflammatory response. A serum biochemistry profile documented hyponatremia (140 mmol/L; RR: 150 to 165 mmol/L). Infectious disease screening was negative for FeLV/FIV and panleukopenia with commercially available ELISA kits (SNAP FIV/FeLV Combo Test and SNAP Parvo Test; IDEXX Laboratories, Westbrook, Maine, USA). Hemostasis testing consisted of a platelet count (200 K/μL; RR: 151 to 600 K/μL), prothrombin time (22.0 s; RR: 15.0 to 22.0 s), and activated partial thromboplastin time (93.0 s; RR: 65.0 to 119.0 s). On the following day, the client accepted a referral with the expectation of attaining a definitive diagnosis for the anemia to facilitate the decision-making process before any intervention.

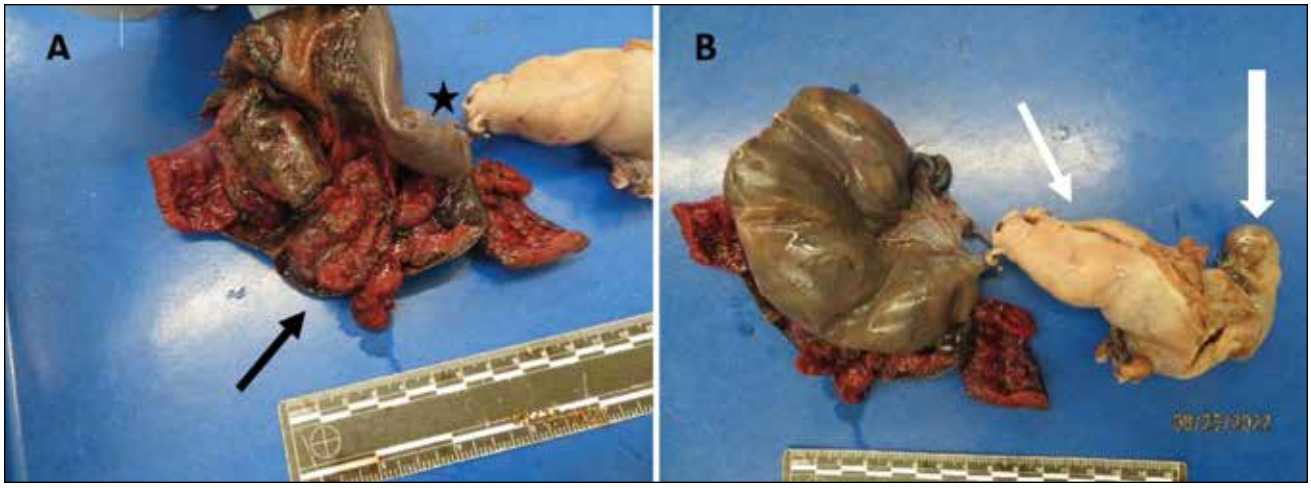

Figure 2. Gross necropsy of the formalin-fixed uterus of a cat with a fetus. A – The left uterine horn (arrow) was moderately dilated with a dark red, hemorrhagic, and severely congested uterine wall. The left uterine horn rotated 360° around its base in the clockwise direction (star). B – The pale white right uterine horn (thin arrow) was not involved in the torsion. A nonviable fetus is also visible (thick arrow).

On arrival at the WCVM, the cat was lethargic but alert and responsive. Vital parameters were as follows: temperature: 38.4°C, HR: 180 bpm, respiratory rate: 24 breaths per minute, mucous membrane colour: pale, capillary refill time: < 2 s. Physical examination revealed a Grade-II/VI systolic heart murmur and multiple abdominal masses. Point-of-care blood parameters were obtained: PCV: 10%, glucose: 9.5 mmol/L, urea: 5 to 15 mmol/L, total solids/protein: 6.5 g/dL.

After the blood type (A) was determined, a 60-milliliter whole-blood transfusion was given over 4 h, which increased the PCV from 10 to 16%. A second, 60-milliliter whole-blood transfusion was started as diagnostic tests were undertaken and surgical intervention was initiated. The PCV increased further, from 16 to 25%. During the blood transfusion, the cat spontaneously aborted a dead fetus. The cat was also treated with maropitant (1.0 mg/kg, IV, q24h) and lactated Ringer's solution at a 1.5× maintenance rate. During stabilization, a clinical pathologist reviewed a blood smear, which showed polychromatophils indicative of a regenerative anemia. There was no evidence of agglutination, spherocytosis, Heinz bodies, or *Mycoplasma* organisms. The PCR panels for *Mycoplasma haemofelis* and *M. haemominutum* were also submitted at this time.

An emergency abdominal ultrasound examination was also done and identified 1 viable and multiple nonviable fetuses, diffuse peritonitis, and a mild volume of anechoic peritoneal effusion. The uterus was distended and contained anechoic fluid (Figure 1). A nonviable fetus was within the pelvic canal. A uterine torsion was not evident on abdominal ultrasound. An abdominocentesis was not attempted given that the small volume of peritoneal effusion present might have required multiple attempts to adequately sample or have been nonproductive. It might also have further delayed surgery and would not have influenced the ultimate recommendation to perform an exploratory laparotomy.

Even though a definitive explanation for this cat's anemia could not be provided at that point and there was potential for multiple disease processes, the option to proceed with exploratory laparotomy and C-section was offered to the client. Multiple nonviable fetuses had been identified and the remaining, viable fetus was likely too premature for survival even if still alive at the time of surgery. Thus, to facilitate understanding and confidence in moving forward with emergency surgical intervention to save the adult cat, 2 scenarios were discussed with the client. The first scenario was that an exploratory abdominal surgery might identify the source of the anemia and treat it as part of the C-section with ovariohysterectomy. The second scenario was that the source of anemia may not be identified or treated in surgery, suggesting a yet-unidentified source of anemia. After surgery, the cat's anemia would be monitored and further diagnostic tests would be employed if it did not resolve. Regardless of the source of anemia, the cat needed an emergency exploratory laparotomy, C-section, and ovariohysterectomy to save its life. The client elected to move forward with surgical intervention and monitoring.

General anesthesia involved fentanyl (5 μg/kg, IV) and dexmedetomidine (5 μg/kg, IV) as premedicants, alfaxalone (2.5 mg/kg, IV) and midazolam (0.2 mg/kg, IV) for induction, and isoflurane for maintenance. Throughout the procedure, IV fluid therapy was maintained with an isotonic crystalloid (Normosol-R; ICU Medical, Lake Forest, Illinois, USA) at 9 mL/h and a fentanyl continuous rate infusion (10 μg/kg per hour). A standard ventral midline approach was made to the abdomen. A moderate amount of serosanguinous fluid was observed upon entry into the peritoneal cavity. Once suctioned, an active source of hemorrhage was not appreciated. The uterus was exteriorized and isolated with laparotomy sponges. A uterine torsion was identified at the pedicle of the left horn, which was distended and severely congested with a necrotic base (Figure 2 A, B). Seven nonviable fetuses were identified. Ovariohysterectomy was followed by a full abdominal exploration, with no bleeding observed at any other site, including upon reassessment of the ovarian and uterine stumps. Other

perioperative medications included cephazolin (22 mg/kg, IV), ephedrine (0.05 mg/kg, IV) for hypotension, and an incisional bupivacaine splash block.

The cat made an uneventful recovery from surgery with a return to normal activity levels with normal appetite and energy. Packed cell volumes of 28% at 1 d post-surgery and 18% at 2 d post-surgery were recorded. The reason for the drop in PCV at 2 d post-surgery remained unclear. Some possibilities can be offered, including a technical error in measurement with a microhematocrit tube, fluid dilution, or possible hemolysis, even though no overt transfusion reactions were observed. The cat was discharged 2 d post-surgery due to good clinical improvement; instructions were to monitor her clinically and reassess her red blood cell mass.

Histopathology revealed marked hemorrhage, congestion, and necrosis in the uterus without significant suppurative inflammation. The placenta and fetus at the site of torsion showed coagulative necrosis. Results of aerobic and anaerobic bacterial culture testing of the amniotic fluid were negative, and no organisms were visible histologically. The *M. haemofelis* and *M. haemominutum* PCR panels submitted previously were negative.

During telephone conversations 1 mo and 10 mo post-surgery, the cat's owner reported that the cat was acting completely normally in terms of appetite, activity levels, and behavior, without any clinical signs indicating relapse of anemia. The client indicated that she was satisfied with her cat's clinical status as evidence that the anemia was resolved and she did not wish to pursue reevaluation of a hematology profile.

Discussion

This case emphasized clinical decision-making and client communication when navigating an undetermined cause of severe anemia during mid-gestation in a cat. It is important to consider uterine torsion during pregnancy as a cause of intrauterine or abdominal hemorrhage. It may be more challenging for veterinarians to make the connection between anemia and an obstetrical emergency for cats in early stages of pregnancy without mineralized fetuses. In such cases, the clinician is faced with a severe anemia that is typically regenerative. At the same time, the clinician is also faced with the dilemma of how the pregnancy and its potential complications might be contributing to the anemia *versus* a separate infectious, toxic, immune, or neoplastic etiology. Although normocytic, normochromic anemia with reticulocytosis is known to occur during late gestation in normal cats, this anemia is not expected to be as severe as that occurring in cats with uterine torsion (1).

Ultimately, exploratory abdominal surgery diagnosed a uterine torsion and a serosanguinous effusion. Histopathology of the uterus revealed congested blood vessels and hemorrhage in the uterine wall. Significant neutrophilic inflammation or organisms were neither seen nor cultured to support a septic component. This case is consistent with previously published reports in which anemia caused by uterine torsion ranged from 9 to 22% (5,6,8,9,13,14), indicating a significant loss of blood in the uterus and vascular stasis in its wall. If these animals survive after transfusion and ovariohysterectomy, there is typically a short clinical recovery time and resolution of the anemia (6,8,9,13,14).

The volume of actual abdominal fluid may be less than anticipated to explain the severity of the anemia, as a ruptured blood vessel(s) might not exist. Instead, there may be a transudation or weeping of fluids due to increased hydrostatic pressure or permeability of the affected uterine vessels because of the torsion itself or secondary inflammation. The authors speculate that the absence of a large-volume hemoperitoneum may further lead clinicians away from the potential of intra-abdominal hemorrhage, as the blood is within the uterus itself and the fluid lost into the abdomen may be lost at a rate slow enough that reabsorption mechanisms are not overwhelmed.

Another key message is that uterine torsion itself is not typically diagnosed with ultrasonography, but rather during exploratory laparotomy. In published case studies, uterine torsion was not determined *via* either abdominal radiography or ultrasonography; only exploratory celiotomy definitively diagnosed the torsion (2,5–9,13,14). In human medicine, the usefulness of a CT scan in diagnosing uterine torsion was discussed due to the inadequate sensitivity of ultrasonography (15,16). This diagnostic modality should theoretically have the same potential in veterinary medicine. The possible benefits of conducting a CT scan to attempt to establish a presurgical diagnosis and the radiation exposure risk to neonates remain to be determined in cats.

In conclusion, uterine torsion should be strongly considered as a differential diagnosis in a severely anemic cat during the mid to late stage of pregnancy if routine blood work, FeLV testing, and abdominal imaging do not provide an alternative explanation. Practitioners may be initially hesitant to proceed with surgery given the fact that clients and veterinarians may have expectations for a definitive presurgical diagnosis to explain the anemia, as it is possible another etiology may exist for the severe anemia. Despite inconclusive preoperative diagnostic investigations, practitioners often already consider a C-section with ovariohysterectomy once evidence of fetal distress, spontaneous abortion, or dystocia arises. Saving the cat will require stabilization, including possible blood transfusion and an exploratory laparotomy with emergency C-section and ovariohysterectomy, followed by monitoring, with further investigation into the anemia should it not resolve postoperatively (2,5–9,13,14,17).

Acknowledgments

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Article

Preliminary evaluation of an indwelling epidural catheter for repeat methylprednisolone administration in canine lumbosacral stenosis

Marie-Philippe Bussières, Stefania Grasso, Philip Jull

Abstract

Objective

To determine the complications, outcomes, and patency of a permanent epidural catheter and subcutaneous access port system (ECAPS) as part of conservative management of degenerative lumbosacral stenosis in dogs.

Animals and procedure

Medical records of 11 client-owned dogs that underwent an ECAPS insertion were evaluated retrospectively. Clinical signs, complications related to the procedure, and system patency are reported.

Results

All dogs had lumbosacral pain at their initial neurological assessment, with comfort levels adequately controlled following epidural infiltrations. None suffered from complications related to the ECAPS procedure. In 10 dogs, there were no malfunctions for the duration of the study. However, in 1 dog, there was a suspected leak at Day 814. The longest duration of patency reported in this study was 870 d (at the time of writing).

Conclusion

Placement of an ECAPS is a feasible technique and a viable option to permit repeated epidural injections of steroids in dogs with degenerative lumbosacral stenosis that is managed conservatively. Further studies are required to evaluate complication rates.

Résumé

Évaluation préliminaire d'un cathéter épidural permanent (à demeure) pour l'administration répétée de méthylprednisolone lors de sténose lombosacrée dégénérative chez le chien

Objectif

Décrire la technique, les complications, les résultats et la perméabilité d'un système composé d'un cathéter épidural et d'un port d'injection sous-cutanée (ECAPS) pour le traitement médical de la sténose lombosacrée dégénérative chez le chien.

Animaux et protocole

Les dossiers médicaux de 11 chiens appartenant à des clients ayant subi l'implantation d'un ECAPS ont été évalués de façon rétrospective. Cette étude décrit les signes cliniques, les complications reliées à la procédure et la perméabilité du système.

Résultats

Tous les patients inclus présentaient de la douleur lombosacrée à l'examen initial. Le niveau de confort de tous les patients suite aux injections épidurales fut maitrisé de façon adéquate. Aucun des patients n'a subi de complications reliées à l'implantation du système. Le système n'a pas démontré de dysfonctionnement dans le cas de dix patients. Chez un des patients, une fuite fut suspectée au jour 814. La durée maximale de perméabilité enregistrée dans cette étude est de 870 jours (au moment de la rédaction).

Conclusion

L'implantation d'un système ECAPS représente une option faisable et viable pour l'administration additionnelle de stéroïdes pour une gestion conservatrice de sténose lombosacrée dégénérative chez les chiens atteints. Des recherches supplémentaires sont requises pour l'évaluation des taux de complications.

(Traduit par les auteurs)

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The Veterinary Specialty Clinic for Anesthesia and Neurology (VSCAN), 2300 Carling Avenue, Ottawa, Ontario K2B 7G1.

Address all correspondence to Marie-Philippe Bussières; email: mariephilippe.bussieres90@gmail.com

Introduction

Canine degenerative lumbosacral stenosis syndrome (DLSS) is a multifactorial neurologic condition characterized by compression and inflammation of the *cauda equina* and resulting in lumbar pain with or without neurological dysfunction (1). Middle-aged and medium-to-large dogs are predisposed (2); however, small dogs and cats can also be affected (3). The most common clinical signs are difficulty rising, jumping, or climbing stairs; low tail carriage; and urinary or fecal incontinence. Neurologic deficits include withdrawal reflex deficits and pain on various tests (low lumbar dorsal deep palpation, hip extension-induced lordosis, rectal dorsal palpation, and the tail-up stretch test). In more severe cases, proprioceptive deficits can also be observed (1,2,4–6). Degenerative lumbosacral stenosis syndrome includes multiple degenerative changes such as lumbosacral spondylosis, lumbosacral stenosis, end plate sclerosis, disc protrusion or extrusion, hypertrophy of the interarcuate ligament or facet joint, disc space collapse, and varying degrees of vertebral misalignment or retrolisthesis of the sacrum (1,2). Various imaging modalities are used, including radiographs, computed tomography (CT), and magnetic resonance imaging (MRI), with severity of imaging findings often not matching clinical signs (1). The final diagnosis is based on history, clinical presentation, medical imaging findings, and occasionally, electrodiagnostics (1,2).

Management of DLSS traditionally involves either conservative (medical) management or surgical intervention. The former includes oral analgesics, specific neuro-analgesics (gabapentin) and anti-inflammatory medications (NSAIDs or corticosteroids) combined with restricted exercise. In 1 report, ~1/2 of dogs treated conservatively had good outcomes (6). If oral medications are not effective, epidural infiltrations with methylprednisolone acetate can be given (2). The majority (79%) of dogs had improvement and 53% had complete resolution of clinical signs in 1 study evaluating repeat epidural steroid administration (2). Physical rehabilitation, acupuncture, intradiscal injection of mesenchymal stem cells or celecoxib, and epidural injections of platelet-rich plasma have also been reported; however, further studies are required to establish their efficacy (1,7–9).

Surgical management of DLSS includes lumbosacral (L7-S1) dorsal laminectomy, foraminotomy, and distraction/stabilization (1,4), with reported success rates of 67 to 97% (5,10–12). The decision between conservative and surgical treatment is usually made based on multiple factors, including the dog's age, severity of clinical signs, level of activity (*e.g.,* whether the animal is a working dog), initial responses to conservative management, owner's commitment, and concomitant diseases. Surgical management requires anesthesia, may result in surgical complications, and requires prolonged recovery, all of which are undesirable in geriatric animals with comorbidities. Repeated L7-S1 epidural infiltrations are an efficient way to provide pain relief and control clinical signs over time with a success rate comparable to the surgical approach (2). Unfortunately, this strategy requires the dog to undergo repeated hospitalization, sedation, and general anesthesia. Since DLSS is more prevalent in geriatric dogs, multiple anesthetic events is an additional downside, especially for those presenting with concomitant diseases.

The purpose of this retrospective case series study was to describe a novel technique combining an indwelling epidural catheter and subcutaneous access port system (ECAPS) to deliver corticosteroids epidurally, on an as-needed basis, without repeated sedation or general anesthesia, and to determine the complications, outcomes, and patency of the system. This is a preliminary report of the clinical use of this technique, duration of ECAPS patency, and potential short-term complications. We hypothesized that placement of an ECAPS is a feasible and successful method to permit repeat epidural corticosteroid administration in dogs with DLSS. To the best of the authors' knowledge, this technique has not yet been reported as part of conservative management of DLSS in veterinary medicine.

Materials and methods

Inclusion criteria and data collection

We reviewed the medical records of dogs treated at a private referral neurology practice in Canada, The Veterinary Specialty Clinic for Anesthesia and Neurology (VSCAN; Ottawa), that underwent ECAPS placement between June 2018 and January 2022. Dogs were included if they met the following criteria: clinical signs compatible with L4-S3 spinal cord segment localization, diagnosis of DLSS based on MRI, owner's choice of conservative management, good clinical response to the first L7-S1 epidural infiltration with steroid performed immediately after MRI while the animal was under anesthesia, and successful placement and use of the ECAPS for at least 1 infiltration. Dogs were excluded if medical records were incomplete (*e.g.,* missing follow-up assessments). Dogs with concomitant orthopedic, neurological, cardiovascular, or metabolic conditions were not excluded, considering the small sample size and the DLSS prevalence in older dogs, which often have other diseases. Eleven records that fulfilled the inclusion/exclusion criteria were identified and the following data extracted: signalment, clinical signs at presentation, findings of initial neurological examination, MRI diagnosis, date of ECAPS placement, results of follow-up neurological examinations, epidural infiltration intervals, duration of ECAPS patency, and date of last communication with owner.

Magnetic resonance imaging protocol

A Board-certified neurologist and neurology resident evaluated all dogs. After the neurological assessment, a presumptive diagnosis of DLSS was made. Clinical signs and examination findings are summarized in Table 1. Under general anesthesia, dogs underwent a thoracolumbar MRI obtained with a 1.5 Tesla High-Field MRI unit (Philips Medical Systems, Eindhoven, Netherlands). We obtained T2-weighted images (T2WI) and T1-weighted images (T1WI) pre- and post-IV administration of gadolinium (ProHance; Bracco Imaging Canada, Montreal, Quebec), 60 mg/kg, in sagittal, axial, and coronal planes. A HASTE sequence and a proton density image were also obtained in a sagittal plane, and short tau inversion recovery (STIR) along with principle of selective excitation technique (PROSET) images were obtained in the coronal plane.

Materials

The ECAPS had 2 components: an epidural catheter (Epidural Pain Management Kit; MILA International, Florence, Kentucky,

Table 1. Descriptions of dogs that received an indwelling epidural catheter for repeat methylprednisolone administration.

Dog	Breed	Age at port placement (y)	Sex	Difficulty rising/ paraparesis/ circumduction (11/11)	Difficulty jumping (9/11)	Ataxia (2/11)	Proprioception deficits (6/11)	Withdrawal deficits in pelvic limbs (9/11)	Pain upon palpation of lumbosacral area or hip extension or rectal exam (10/11)	Concomitant orthopedic conditions
1	Labrador retriever	6	MN	+	+	−	−	+	+	−
2	German shepherd	9	ME	+	−	+	+	+	−	−
3	Pug	13	MN	+	−	−	−	−	+	Right shoulder and elbow OA[a]
4	Mixed-breed	12	MN	+	+	−	+	−	+	Stifle pain Bilateral medial patellar luxation Right shoulder and elbow pain
5	Australian shepherd	7	MN	+	+	−	+	+	+	−
6	Great Dane	8	MN	+	+	−	−	+	+	−
7	Australian shepherd	14	MN	+	+	−	+	+	+	Hip and shoulder pain
8	Mixed-breed	8	MN	+	+	−	−	+	+	−
9	Labrador retriever	12	MN	+	+	−	+	+	+	Stifle remodelling — suspected OA
10	Labrador retriever	10	FS	+	+	−	−	+	+	Elbow, shoulder, and hip pain — OA
11	English bulldog	7	FS	+	+	+	+	+	+	−

FS — Female spayed; ME — Male entire; MN — Male neutered; OA — Osteoarthritis.
"+" symbol — Present; "−" symbol — Absent.
[a] This condition developed at the last follow-up (870 d).

USA) (Figure 1 A) and a subcutaneous vascular access port (Le Petite CompanionPort; Norfolk Vet Products, Skokie, Illinois, USA) (Figure 1 B). Epidural kit components included: 18 G × 3.5 in (1.2 mm × 8.9 cm) or 20 G × 1.75 in (0.9 mm × 4.5 cm) Tuohy needle, 24 G × 10 in (0.51 mm × 25.4 cm) or 20 G × 36 in (0.9 mm × 91.4 cm) epidural catheter, and a loss-of-resistance syringe (Figure 1 A). The Tuohy needle and epidural catheter sizes were chosen based on dog size. The access port package included a 4 Fr (1.3 mm) rounded-tip silicone catheter and a 22 G (0.7 mm) Huber needle (Figure 1 B, C).

Epidural catheter and access port system (ECAPS) placement procedure

The anesthetic protocol was at the anesthesiologist's discretion. Premedication included either butorphanol alone or in combination with dexmedetomidine or acepromazine. Anesthetic induction and maintenance were achieved with propofol alone or in combination with ketamine or midazolam and isoflurane, respectively. All dogs received IV fluids during the anesthesia. Each dog was placed in sternal recumbency with pelvic limbs extended cranially. The L6-S1 area was clipped and surgically prepped. A sterile transparent adhesive drape was placed on the prepped area and a 1-millimeter skin incision was made with a #11 surgical blade, to facilitate insertion of the Tuohy needle. At the level of L7-S1, the needle was introduced perpendicular to the skin and advanced into the epidural space. Adequate needle placement was guided by the "pop" sensation felt during puncture of the ligamentum flavum and the hanging-drop technique (13,14). If the hanging drop was negative, then lack of resistance to injection using a syringe was used as verification. Final needle placement was confirmed on sagittal, axial, and coronal reconstruction of a multi-slice (8 slices) CT scan (Toshiba Aquilion; Toshiba Medical Systems, Otawara, Tochigi, Japan) of the lumbar spine.

Once the desired Tuohy needle position was achieved (Figure 2 A), the stylet was removed, and the epidural catheter placed through the Tuohy needle and advanced cranially in the epidural space until the catheter tip reached vertebrae L5 or L6 (Figure 2 B). The CT imaging was repeated throughout the procedure to visualize the catheter course and the final position was confirmed by injecting 0.5 to 1 mL of contrast (iohexol: Omnipaque; GE Healthcare, Mississauga, Ontario), 240 mg/mL. The Tuohy needle and the catheter guide wire were removed, leaving only the epidural catheter *in situ*.

A second incision (~3 cm) was made in the skin and subcutaneous tissues of the L6-S1 area on the midline, caudal to the

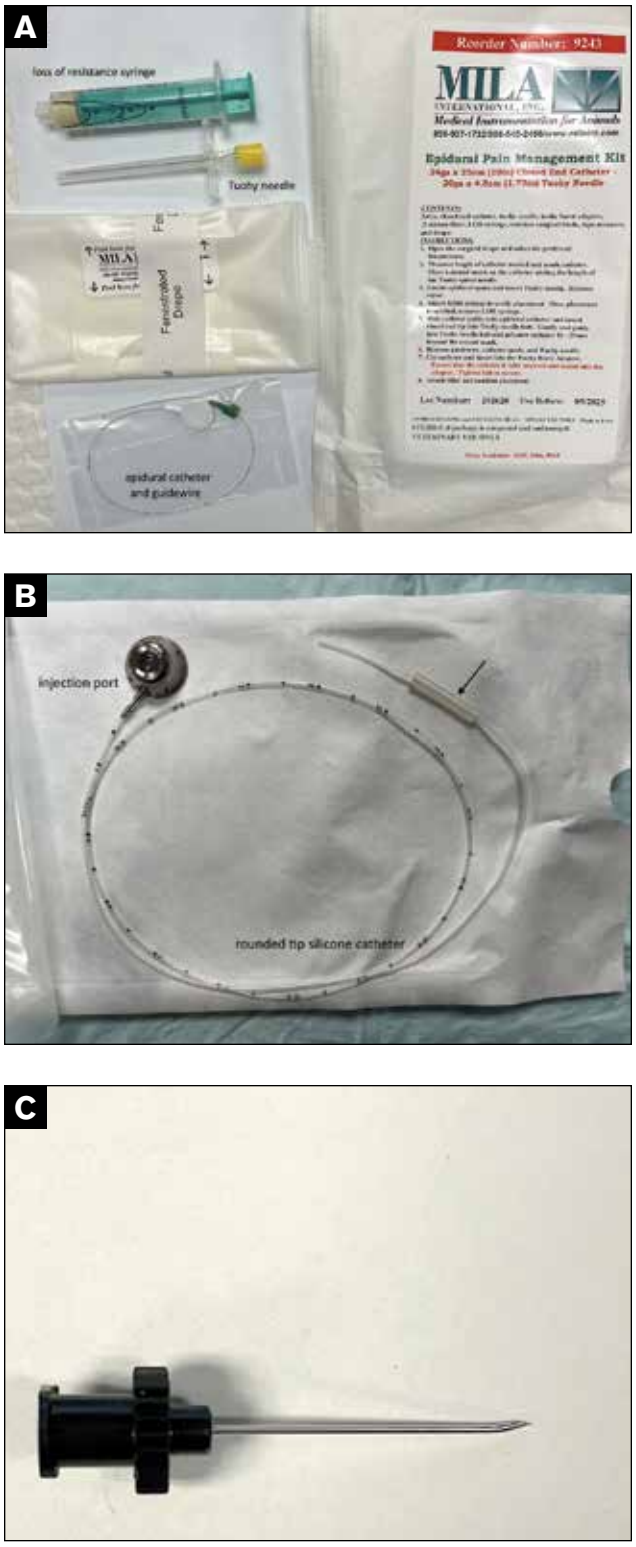

Figure 1. A – At left, the epidural catheter kit components: loss-of-resistance syringe, Tuohy needle, fenestrated drape, and epidural catheter with guidewire. At right, the MILA Epidural Pain Management Kit package (MILA International, Florence, Kentucky, USA). B – Le Petite CompanionPort subcutaneous vascular access port and rounded-tip silicone catheter (Norfolk Vet Products, Skokie, Illinois, USA). The cylindrical silicone piece (arrow) was used for connecting (see Figure 3 B). C – The Huber needle included in Le Petite CompanionPort kit. This is a specially designed, non-coring needle for penetration of the port septum.

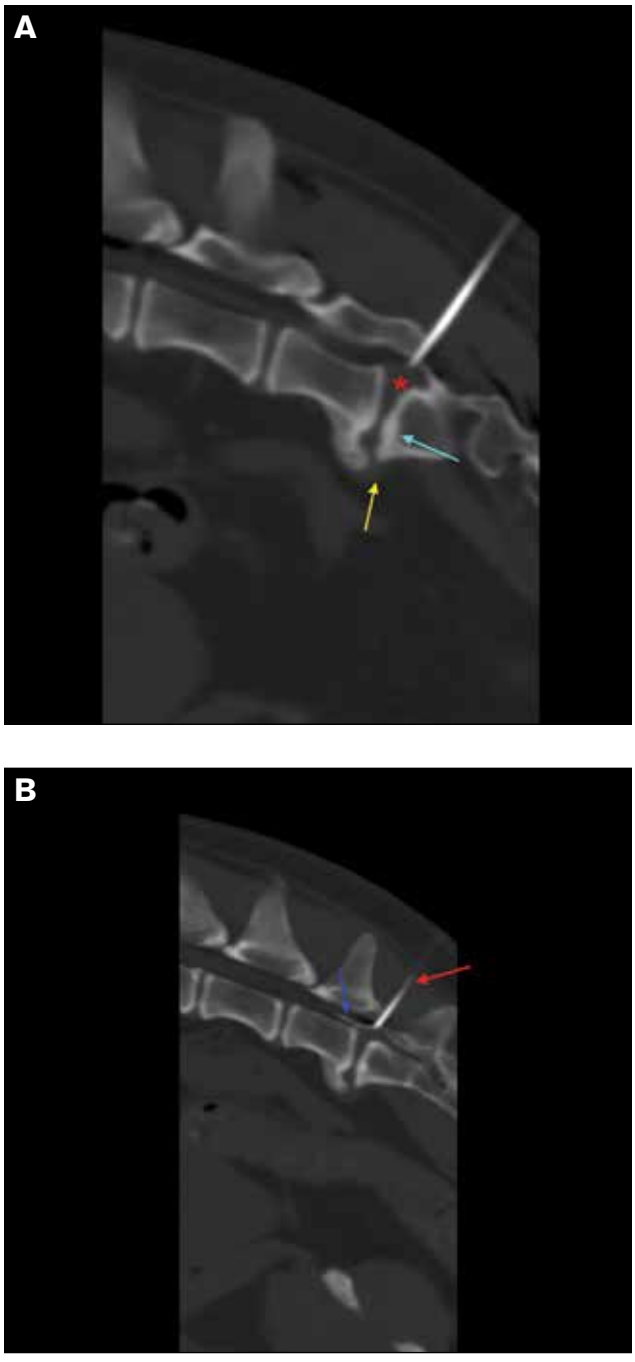

Figure 2. A – Sagittal computed tomographic (CT) image of the lumbosacral area during epidural catheter placement in a dog. Note the correct position of the Tuohy needle at the level of L7-S1. The red star represents the L7-S1 protruded disc. Sclerotic endplates (blue arrow) and spondylosis (yellow arrow) are present. B – Sagittal CT image of the lumbosacral area, showing the epidural catheter (blue arrow) exiting from the tip of the needle (red arrow) being advanced cranially in the epidural space.

incision used to place the epidural catheter. The incision was extended cranially to the previous incision. Subcutaneous tissues were bluntly dissected lateral to the midline to create a pocket where the access port was secured to subcutaneous tissues and muscular fascial layer with 4-0 polypropylene (Prolene; Ethicon, Raritan, New Jersey, USA) The 4 Fr (1.3 mm) rounded-tip silicone catheter was used to connect the access port to the epidural

catheter. A minimum of 2 loops of the silicone catheter were created (~2 cm long) to enable local movement. The silicone and epidural catheters were inserted into each other with a 2-centimeter overlap (Figure 3 A, B, C). Insertion was facilitated by cutting ends obliquely and moistening with saline. For added protection, an additional cylindrical silicone piece (Figure 3 B, blue arrow) was positioned to cover the overlap. A Huber needle (22 G) was used to flush the port and the catheter with 0.5 mL sterile saline, to visualize leaks at the connection sites. A volume exceeding that needed for priming the system (volume of the port and of the catheter, 0.1 mL each) was used to ensure that leaks were visualized. Patency was assessed by confirming minimal resistance while performing the injection (Figure 4 A, B, C). Methylprednisolone acetate (Depo-Medrol 40 mg/mL; Pfizer Canada, Kirkland, Quebec), 1 mg/kg, was injected *via* the epidural port. The epidural port was then flushed with 0.3 to 0.5 mL of sterile saline. Subcutaneous tissue and skin were closed routinely with PDS and Monocryl. Following skin closure, it was verified that the access port could be easily palpated under the dog's skin (Figure 5 A, B, C).

Postoperative period

All dogs were discharged on the day of the procedure. For the first 3 d, oral medications included gabapentin (10 to 15 mg/kg q8h), codeine (0.5 to 0.7 mg/kg q8h), and diazepam (0.2 mg/kg q8h), in various combinations, as needed. Long term, dogs were given gabapentin, methocarbamol (Robaxin; Pfizer, Mississauga, Ontario), 22 to 37 mg/kg q12h or q8h, and NSAIDs drug or agent, including robenacoxib (Onsior; Elanco, Mississauga, Ontario), 1 mg/kg q24h, or meloxicam (0.1 mg/kg q24h), in various combinations, depending on the dog's comfort level. Two weeks of strict rest was advised following the procedure. The surgical incision was assessed 2 wk postoperatively, either using pictures sent by the owner or at the first follow-up examination.

Long-term epidural infiltration protocol

The interval of the first 3 epidural infiltrations was based on the protocol described by Janssens *et al* (2). The first infiltration of methylprednisolone acetate (1 mg/kg) was administered immediately after the MRI. The second infiltration was administered 2 wk thereafter, either through the port at the time of the ECAPS placement or as a single infiltration for patients without ECAPS. The third infiltration was administered through the ECAPS 4 wk after the second treatment. Subsequent infiltrations were administered through the ECAPS based on patient comfort and neurological assessment. Each time, the patency of the ECAPS was tested by flushing 1 mL of sterile saline while assessing for high or low resistance during injection. Testing for resistance during steroid infiltrations was done in the same fashion. Before injecting into the ECAPS, the fur over the access port was parted and the skin aseptically prepared using 70% alcohol and 0.5% chlorhexidine. A Huber needle was inserted perpendicularly into the port and, after checking for patency, the calculated volume of methylprednisolone acetate was injected, followed by a final saline flush (0.3 to 0.5 mL) to deposit the medication epidurally.

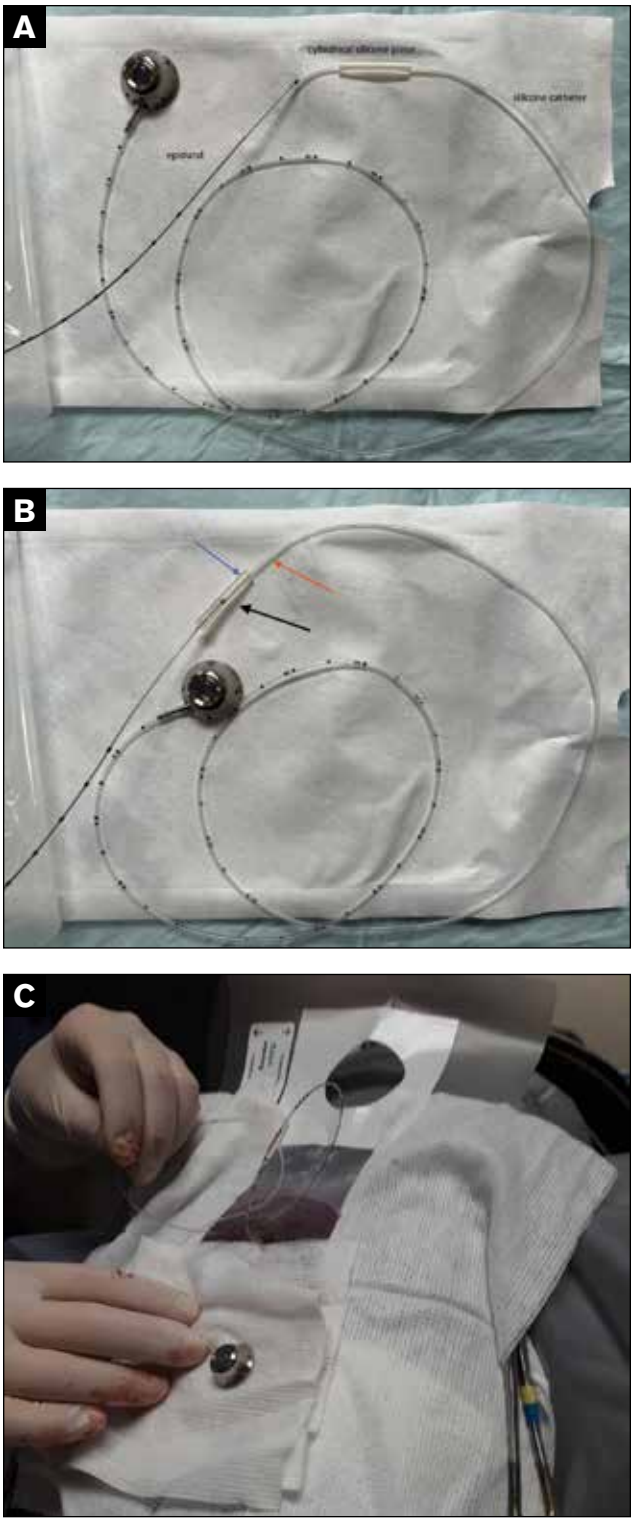

Figure 3. A – Connection of the 2 components of the system (epidural and silicone catheters). The setup in this figure is used as an example and was not used during the procedure. B – Connection of 2 catheters, showing their overlap (in this picture, the overlap is only halfway). The black arrow indicates the tip of the epidural catheter, and the red arrow indicates where advancement of the epidural catheter should finish, resulting in ~2 cm of overlap in order to avoid disconnection. An additional, cylindrical silicone piece (blue arrow) is positioned to cover the overlap and enhance protection. C – The epidural catheter is shown in place with the connection to the silicone catheter and port.

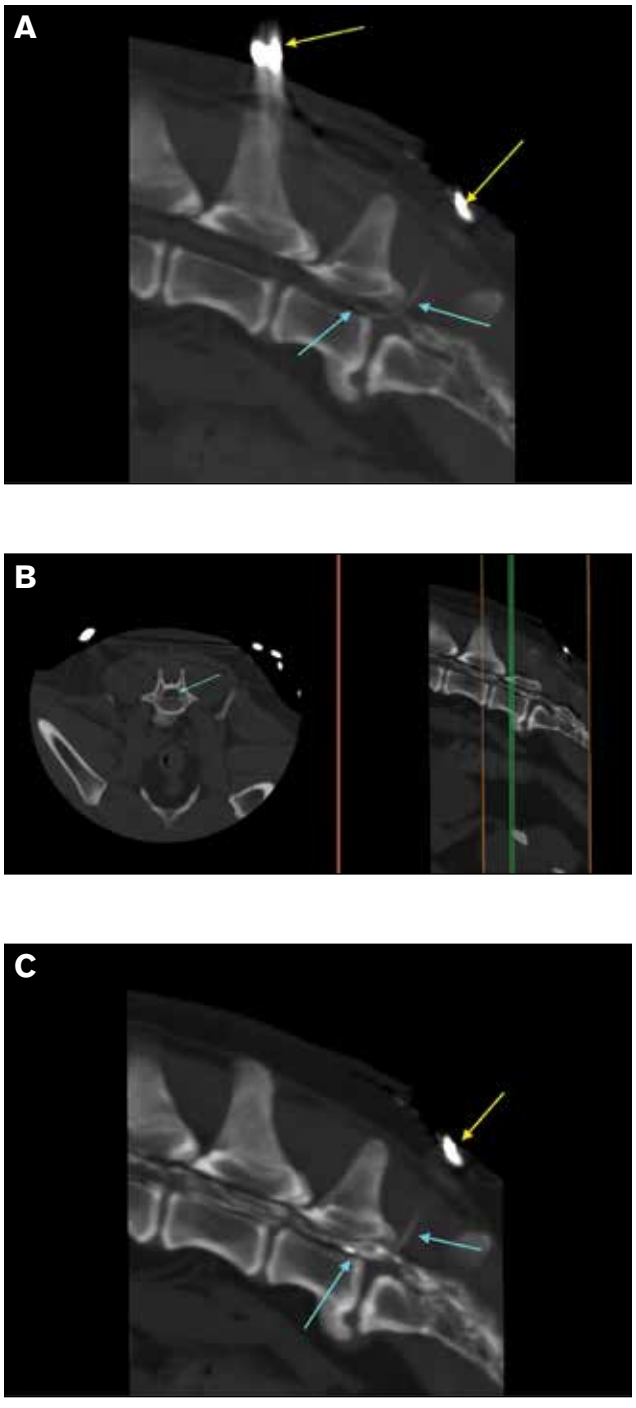

Figure 4. Computed tomographic (CT) images acquired during the placement of an indwelling epidural catheter in a dog. A – Sagittal CT image of the lumbosacral area before contrast administration. Note the hyperattenuating material (blue arrows) in the epidural space, representing the epidural catheter. The dorsal hyperattenuating material (yellow arrows) is Backhaus forceps used to hold the epidural catheter. B – Left: Transverse CT image of the lumbosacral area, showing hyperattenuating material (white dot identified by the blue arrow) in the epidural space. This represents the epidural catheter following contrast injection and illustrates the relatively small size of the catheter. Right: Sagittal section showing the level at which the transverse section was taken (L7 vertebral body, green vertical line). C – Sagittal CT image of the lumbosacral area following contrast administration, showing hyperattenuating material in the epidural space representing the epidural catheter (blue arrows) and the port (yellow arrow) in the subcutaneous space.

Outcome evaluation

Complete anamnesis and neurological examination were used at follow-up examinations. Dogs were examined for lumbosacral pain using 4 tests: low lumbar dorsal deep palpation, hip extension-induced lordosis, rectal dorsal palpation, and the tail-up stretch test. Comfort levels were evaluated based on the reactions. All clients were asked whether dogs had difficulty rising or jumping; or any changes in tail carriage, gait, energy, appetite, urination, or drinking. Each dog's ability to perform usual activities at home without signs of discomfort was also discussed.

Results

Clinical outcomes

Medical records of 11 dogs that fit inclusion criteria, including a positive first response to an epidural infiltration, were reviewed; these included 3 Labrador retrievers, 2 Australian shepherds, and 1 each of German shepherd, Great Dane, Newfoundland cross, English bulldog, pug, and mixed-breed. There were 9 male and 2 female dogs. The age range was 6 to 14 y (median: 9 y). Clinical signs were paraparesis and reluctance to rise (11/11), pain upon palpation of the L7-S1 area or hip extension (10/11), difficulty jumping (9/11), withdrawal deficits (incomplete tarsus flexion) (9/11), mild proprioceptive deficits in pelvic limbs (6/11), proprioceptive ataxia in the pelvic limbs (2/11), and fecal and urinary incontinence (1/11) (Table 1). The MRI findings included varying degrees of disc protrusion and foraminal stenosis at L7-S1 (11/11), spondylolisthesis at L7-S1 (5/11), varying degrees of multiple thoracolumbar disc protrusions cranial to L7-S1 (4/11), thoracic spine vertebral canal narrowing in the thoracic spine and dorsal compartment remodelling (2/11), dorsolateral arachnoid diverticulum (1/11), gliosis (1/11), irregular vertebral endplates with faint contrast enhancement (1/11), and contrast enhancement of the L4 spinal nerve (1/11) (Table 2).

At the first follow-up, all 11 dogs had an absence of pain using the 4 tests listed (Table 3). Improved strength and tail carriage were reported by the owners and all dogs had improvement in the degree of paraparesis. The pelvic limb ataxia that was initially present in 2 dogs (Dogs 2 and 11) remained unchanged at the first and second follow-ups; these dogs had additional, concomitant neurological diseases affecting the T3-L3 segment (Tables 1 and 2) that explained this clinical finding. Dog 2 had a presumptive diagnosis of discospondylitis based on mildly irregular endplates and faint contrast enhancement noted at the L7-S1 disc on MRI. This dog was administered 6 wk of cephalexin and azithromycin that was discontinued without deterioration. Difficulty jumping was still present in 2 dogs, proprioception deficits in 4 dogs, and withdrawal deficits in 5 dogs (Table 3). Additional oral pain-relief medications were used for 8 dogs after the first follow-up. Dog 11 was diagnosed with T8-T9 subarachnoid diverticula causing urinary and fecal incontinence that remained unchanged after ECAPS placement.

At the second follow-up, mild paraparesis was observed in 3 dogs (Table 4), but owners consistently reported less difficulty to rise. Ten dogs continued to have adequate comfort levels afterwards with repeat epidural infiltrations. In 1 dog (Dog 4),

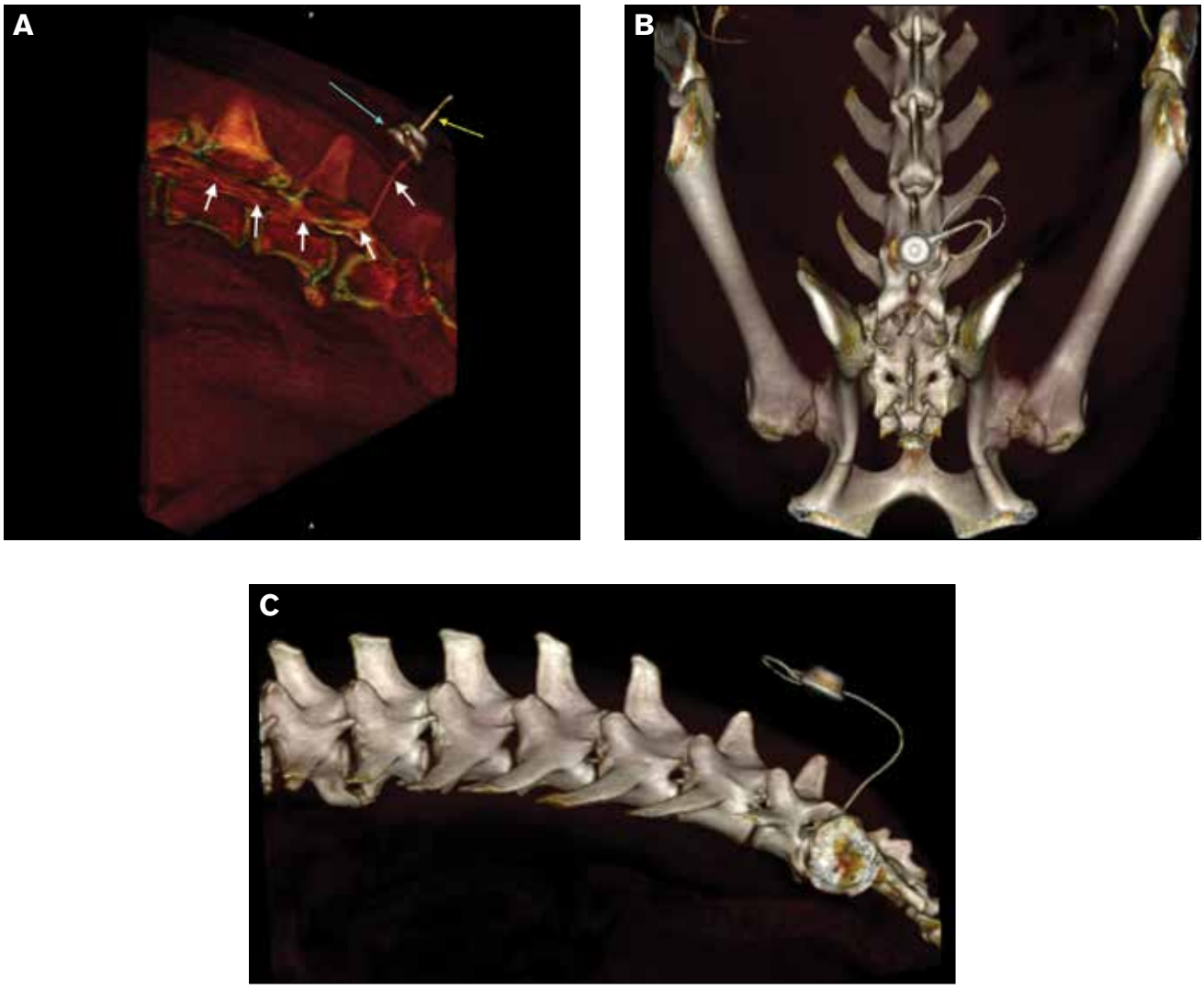

Figure 5. A – Sagittal computed tomographic image of the lumbosacral area in a dog, with soft tissue reconstruction representing the subcutaneous injection port connected to the epidural catheter (white arrows) inserted at L7-S1. This image illustrates that the subcutaneous port (blue arrow) is quite easy to feel/identify under the skin when inserting the Huber needle (yellow arrow). B – Computed tomographic imaging with 3-dimensional reconstruction, showing the lumbosacral spine after the epidural catheter and port system implantation, in a dorsal view. C – Computed tomographic imaging with 3-dimensional reconstruction, showing the lumbosacral spine after the epidural catheter and port system implantation, in a sagittal view.

occasional discomfort at home and upon unilateral hip extension was noted at 3-week intervals, and the methylprednisolone dose was increased to 2 mg/kg after the second follow-up. At the second follow-up, difficulty jumping was still present in 5 dogs, proprioception deficits in 5 dogs, and withdrawal deficits in 6 dogs (Table 4). Additional oral pain-relief medications were used for 6 dogs (Table 4). Dog 3 developed fecal incontinence throughout the follow-up period (665 d after implantation) but otherwise had an overall stable neurological status throughout subsequent follow-ups.

Five dogs underwent the ECAPS placement 2 wk following initial epidural infiltration and the remaining 6 later (mean: 12.5 wk, median: 5 wk) following the first epidural infiltration (Table 5). After the third epidural infiltration, the shortest interval between subsequent infiltrations was 4 wk (Dog 4) and the longest was 16 wk (Dog 5; Table 5). Dog 1 had an excellent response to the epidural protocol. This dog was evaluated by the same neurologist at 60 d and 120 d following the last epidural infiltration through the ECAPS. Based on the absence of discomfort and neurological deficits, the methylprednisolone infiltrations were discontinued. The total follow-up interval was 4 to 28 mo (mean: 15, median: 14). At the time of writing, 5 dogs had been euthanized (for reasons unrelated to the DLSS), thereby shortening the follow-up period.

Patency of the epidural catheter and access port system (ECAPS)

The duration of ECAPS presumed patency was 74 to 870 d (mean: 466 d, median: 448 d; Table 5). All ECAPS systems were presumed patent for the full follow-up period in each dog, apart from in 1 case (Dog 4). Short-term complications related to the procedure and the presence of the ECAPS were not observed. In Dog 4, at Day 814, there was high resistance to injection and visible local subcutaneous fluid leakage, implying malfunction,

Table 2. Magnetic resonance imaging (MRI) findings at the time of diagnosis of degenerative lumbosacral stenosis for dogs subsequently given an indwelling epidural catheter for repeat methylprednisolone administration.

Dog	MRI findings
1	Foraminal stenosis and spondylolisthesis L7-S1
2	Mildly irregular endplates and faint contrast enhancement L7-S1 Multiple disc protrusions[a] Severe foraminal stenosis, disc protrusion, and spondylolisthesis L7-S1
3	Foraminal stenosis and spondylolisthesis L7-S1 Multiple disc protrusions[b]
4	Foraminal stenosis and mild disc protrusion L7-S1
5	Foraminal stenosis and moderate disc protrusion L7-S1 Gliosis T11-T12[c]
6	Foraminal stenosis and moderate disc protrusion L7-S1
7	Moderate foraminal stenosis and moderate disc protrusion L7-S1 Multiple disc protrusions[d]
8	Foraminal stenosis, moderate disc protrusion, and spondylolisthesis L7-S1 Mild cranial thoracic vertebral canal narrowing associated with mild dorsal compartment proliferation
9	Foraminal stenosis, moderate disc protrusion, and spondylolisthesis L7-S1 Multiple disc protrusions and L4-L5 lesion[e]
10	Foraminal stenosis and moderate disc protrusion L7-S1
11	Foraminal stenosis L7-S1 Chronic disc disease throughout thoracolumbar spine Dorsolateral arachnoid diverticulum T8-T9 with moderate spinal cord compression

[a] T13-L1, L1-L2, L2-L3, L3-L4.
[b] Mild at T10-T11, L2-L3, L5-L6, L7-S1; and moderate at T12-T13 and T13-L1.
[c] Moderate vertebral canal narrowing causing secondary spinal cord compression.
[d] Multifocal, moderately compressive disc protrusions at T13-L1, L1-L2, and L2-L3.
[e] Diffuse, chronic, mild-to-moderate disc protrusions from T13 to L3-L4. Focal area of avid contrast enhancement along the nerve root of L4-L5, suspicious for nerve sheath tumor (no biopsy available).

displacement, or disconnection of the ECAPS. Repeat imaging with contrast medium was considered but was not done as the dog was euthanized soon thereafter for a concomitant cardiac condition.

Discussion

Degenerative lumbosacral stenosis syndrome is a common condition in dogs. In general, it is recommended that dogs with DLSS associated with severe deficits or with inadequate response to conservative management undergo surgical intervention. However, management with oral analgesics, exercise restriction, and corticosteroids administered epidurally improved clinical signs in 79% of cases, a similar outcome to that reported for surgical procedures (1,4–6). The main concern with long-term epidural infiltrations is the need for repeated sedation or general anesthesia (2). In the current retrospective study, insertion of an epidural catheter connected to an access port was a feasible technique for long-term delivery of steroids with the main advantage of avoiding repeated anesthesia.

Epidural steroid infiltrations are frequently used in human medicine to alleviate lumbosacral radicular pain (15,16). Complications related to epidural steroid infiltrations are reported in human and veterinary medicine, but their occurrence remains low as long as prevention guidelines are followed (15,16). Possible complications include technical failure, infection, hematoma, urinary retention, aggravation of neurological status, accidental intrathecal injection, local pruritus and, in dogs, local delayed hair growth (17,18). In a study by Janssens and colleagues, the only veterinary study evaluating serial steroid epidurals, polyuria and polydipsia were reported in 8% of cases (2). In the current study, polydipsia and/or polyuria were not reported in the medical records.

In humans, repetitive epidural infiltration of opioids, local anesthetics, or steroids has been used for pain associated with cancer or neuropathy (19–23), and an indwelling epidural catheter with or without a subcutaneous access port has been described (21–24). The main reported technical problems associated with indwelling catheters in humans include dislodgment, infection, pain on injection, leakage, and occlusion. When a subcutaneous access port was used in conjunction with the epidural catheter, the overall complication rate decreased (24). Early side effects described with implantation of a subcutaneous port in humans included local hematoma and edema, whereas long-term issues were catheter dislodgment, occlusion, leakage, infection, and epidural fibrosis (22). One case report described an additional, rare complication associated with the epidural catheter and subcutaneous port, consisting of an epiduro-subcutaneous connection (direct connection between the epidural space and subcutaneous tissues) (22).

In small animals, epidural catheters have had the following complications: misplacement of the catheter (intrathecal or paraspinal), pain upon local pressure of the lumbar area, coiling of the catheter, and 1 case of focal aseptic necrosis of a dorsal spinous process at necropsy (25,26). In 1 study, 21% of dogs developed complications when the catheter was left *in situ* for 1 to 7 d postoperatively (27). Apart from catheter dislodgment, some dogs experienced infection from fecal contamination at the catheter site, and inflammation such as local dermatitis (27). In large animals, reported complications were similar and included catheter dislodgment, obstruction, knotting and looping of the catheter, leakage, infection, and inflammation (26).

In veterinary medicine, this is the first report documenting use of an indwelling epidural catheter connected to a subcutaneous access port. However, implanted port systems are used for other purposes in veterinary specialty settings (28–30), including repeated blood sampling, chemotherapy, pyothorax management, subcutaneous ureteral bypass (SUB), and management of pleural effusion (28–32). The SUB devices use a system similar to that used in our study, involving an indwelling catheter and a subcutaneous access port, with complications including obstruction due to mineralisation, kinking, or blood clot formation (31,33).

As noted by others, maintaining patency of these devices is a main concern. In our study, patency was assessed by flushing the ECAPS and gauging resistance to the injection, as recommended by the subcutaneous access port manufacturer. This is also the first method used to assess patency of other systems, such as IV catheters and epidural catheters in humans (34–37).

Table 3. Clinical outcomes at first follow-up for dogs given an indwelling epidural catheter for repeat methylprednisolone administration.

Dog	First follow-up[a]	Difficulty rising/ paraparesis/ circumduction (0/11)	Difficulty jumping (2/11)	Ataxia (2/11)	Proprioception deficits (4/11)	Withdrawal deficits in pelvic limbs (5/11)	Pain upon palpation of lumbosacral area or hip extension or rectal exam (0/11)	Additional oral pain-relief medications (8/11)
1	2	−	−	−	−	−	−	Gabapentin
2	4	−	−	+	+	+	−	Gabapentin
3	2	−	−	−	−	−	−	Gabapentin Methocarbamol
4	2	−	+	−	−	−	−	Gabapentin Methocarbamol
5	6	−	−	−	−	−	−	−
6	6	−	−	−	−	+	−	−
7	6	−	−	−	+	+	−	−
8	6	−	−	−	−	−	−	Gabapentin
9	4	−	+	−	+	+	−	Gabapentin
10	5	−	−	−	−	−	−	Gabapentin
11	4	−	−	+	+	+	−	Gabapentin

"+" symbol — Present; "−" symbol — Absent.
[a] Interval (wk) from catheter placement to first follow-up.

Table 4. Clinical outcomes at second follow-up for dogs given an indwelling epidural catheter for repeat methylprednisolone administration.

Dog	Second follow-up[a]	Difficulty rising/ paraparesis/ circumduction (3/11)	Difficulty jumping (5/11)	Ataxia (2/11)	Proprioception deficits (5/11)	Withdrawal deficits in pelvic limbs (6/11)	Pain upon palpation of lumbosacral area or hip extension or rectal exam (1/11)	Additional oral pain-relief medications (6/11)
1	10	−	−	−	−	−	−	Gabapentin
2	14	−	−	+	+	+	−	−
3	8	−	−	−	−	−	−	Gabapentin Methocarbamol
4	6	−	+	−	−	−	+[b]	Gabapentin Methocarbamol
5	14	−	+	−	+	+	−	−
6	12	+	+	−	−	+	−	−
7	13	−	+	−	+	+	−	−
8	8	−	−	−	−	−	−	Gabapentin
9	10	+	+	−	+	+	−	Gabapentin
10	12	−	−	−	−	−	−	Gabapentin
11	8	+	−	+	+	+	−	−

"+" symbol — Present; "−" symbol — Absent.
[a] Interval (wk) from catheter placement to second follow-up.
[b] Reaction on hip extension only.

When loss of patency is suspected, radiographs or CT imaging with contrast could be obtained to confirm obstruction or leakage (35–37).

A retrospective study assessing outcomes following SUB placement reported a median duration of patency of 204 d, with a potential of 1678 d (31). In humans, 2 case reports described ECAPS patency of 2 mo and 31 mo, respectively (21,23). In our study, the longest duration of presumed patency achieved was 870 d, limited by the time of writing. Overall, the current study indicated an excellent duration of presumed patency attributed related to multiple factors. First, the ECAPS was flushed with saline at each appointment. Second, the short length of the

Table 5. System patency and follow-up observations for dogs given an indwelling epidural catheter for repeat methylprednisolone administration.

Dog	Interval[a]	Complications	Mean infiltration interval (wk)	Clinical response	Catheter patency[b]
1	20	None	8	Excellent	136
2	6	None	8	Good	700
3	5	None	5 to 6	Good	870
4	2	Leakage at 814 d	4	Good	814
5	2	None	12 to 16	Good	600
6	8	None	5 to 6	Good	448
7	2	None	10 to 12	Good	570
8	2	None	4 to 5	Good	365
9	20	None	5	Good	74
10	2	None	6 to 8	Good	395
11	16	None	6 to 8	Good	157

[a] Interval (wk) between first epidural and port placement.
[b] Minimum duration (d) of catheter patency.

ECAPS may also explain why the duration of patency was longer than the median duration described with SUB devices. Finally, the epidural environment is less conductive to obstruction compared to the location of an SUB device, where obstruction with urinary sediment is common. This may explain why even the dog with the longest interval between infiltrations (16 wk) did not experience obstruction.

In our study, leakage was presumed in 1 dog at Day 814, perhaps due to device migration, a disconnection between the epidural catheter and port, or catheter malfunction (*e.g.,* permeability), although the exact cause was unfortunately not confirmed. In the authors' experience, forming the connection (Figure 3 B) between the epidural catheter and the rounded-tip silicone catheter of the subcutaneous access port is the most challenging and time-consuming step, especially for small-diameter catheters. The authors tried multiple methods to make the connection in an efficient manner. Lubricating sterile jelly was avoided as it could leave residue in the catheter and increase the likelihood of occlusion. An attempt was made to use the guidewire provided in the epidural catheter kit as a support for gliding of the 2 catheters. However, the diameters of both catheters were too small and there was plication when the guidewire was removed. The latter method could risk weakening or damaging catheters and predisposing to leakage. We concluded that cutting ends obliquely and moistening with saline while performing gentle rotations was the most efficient way to ensure sufficient overlap.

Epidural infiltrations through the ECAPS were administered without clipping fur. It was not considered essential to clip fur, as some dogs had very short hair and, in most, hair at the injection site did not regrow as fast or as long. Epidural infiltrations were performed after 2 abundant 70% alcohol soaks separated by 2-minite waits. None of the dogs in the study had clinical signs of infection. The decision to not re-clip fur was supported by limited fur present at the L7-S1 area. There are multiple reports of human neurosurgeries (38) in hairy areas for which there were no increases in infection rates without presurgery shaving. However, if fur is thick or difficult to part and cannot be readily cleaned, we recommend clipping dogs to avoid risks of introducing bacteria in the ECAPS.

A definitive diagnosis of discospondylitis in Dog 2 was not obtained in the absence of additional testing (*e.g.,* CSF analysis, CSF culture, urine, blood or intervertebral disc culture), due to the owner's constraints. Infiltrations were continued during and after a 6-week antibiotic course. No problems were encountered, attributed to the corticosteroids being delivered locally, minimizing systemic side effects. Dog 3 developed fecal incontinence during the course of the epidural infiltrations. We suspect this was related to progression of concomitant conditions in the spine rather than the presence of an epidural catheter. However, this cannot be confirmed due to a lack of repeat MRI.

Ten dogs responded well to the dose of methylprednisolone used in this study, which was selected based on the findings of Janssens *et al* (2). One dog (Dog 4) had residual discomfort at palpation, and a short interval between reoccurrence of clinical signs was noted. Therefore, the methylprednisolone dose was increased to 2 mg/kg and continued long-term, with a satisfactory clinical response. This increased dose was based on perineural injections for cervical disc disease in dogs (39).

The authors recognize multiple limitations of the current study, including its retrospective nature, small sample size, variable follow-up intervals, and lack of repeated imaging. The small sample size may have underestimated complications related to ECAPS implantation. Further, many dogs had multiple, concomitant conditions that could have affected pain evaluation. Seven dogs had additional neurological conditions in the thoracolumbar spine (*e.g.,* possible discospondylitis, IVDD, diverticula, and suspected neoplasia) and were therefore receiving oral analgesics before epidural infiltrations. In these cases, epidural methylprednisolone was considered as additional pain relief, and discontinuation of oral medications was not necessarily expected. Repeated imaging was not considered due to financial implications and a desire to avoid repeated general anesthesia. Our aim was not to compare surgical *versus* medical management of DLSS, but rather to present an innovative technique to deliver repeated epidural infiltrations without repeated sedation or anesthesia. Due to the retrospective nature of the study, there were no control group, standardized pain-scoring system, or validated client questionnaires.

We concluded that implantation of an ECAPS for repetitive epidural steroid infiltrations is a feasible, minimally invasive option for dogs with DLSS. The procedure did not cause short-term complications. This system allows for easy adjustment of injection frequency for each animal since it does not require

repeated general anesthesia, avoiding long-term costs and risks. Regular flushing of the ECAPS may decrease the likelihood of obstruction. Further studies, including randomized controlled trials with larger sample sizes and objective pain scoring, are required to better assess efficacy and safety of the ECAPS. CVJ

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Article

Perioperative analgesic effects of the erector spinae plane block with bupivacaine or bupivacaine-dexmedetomidine in dogs undergoing hemilaminectomy: A randomized controlled trial

Bartolome Rico Pérez, Cristina Parra Martínez, Francesco Santoro, Manuel E. Herrera-Linares, Carolina Palacios Jiménez

Abstract

Objective

To compare the perioperative opioid requirements among dogs receiving an erector spinae plane (ESP) block with bupivacaine, with or without dexmedetomidine, and a control group.

Animals and procedure

Thirty client-owned, healthy adult dogs undergoing hemilaminectomy were included in this randomized, prospective, blinded clinical study. Dogs were randomly assigned to 1 of 3 treatment groups: Group B, ESP block with bupivacaine; Group BD, ESP block with bupivacaine and dexmedetomidine; and Group C, control. Rescue intra- and postoperative analgesia consisted of fentanyl and methadone, respectively. Postoperative pain was evaluated using the short form of the Glasgow Composite Measure Pain Scale (CMPS-SF).

Results

In Group BD, 0/10 dogs required intraoperative fentanyl, compared to 9/10 in Group C ($P < 0.001$), whereas 1/10 required postoperative methadone, compared to 9/10 in Group B ($P = 0.003$) and 10/10 in Group C ($P < 0.001$). The total amount of intraoperative fentanyl (μg/kg) was 0 (0 to 4) in Group B and 0 (0 to 0) in BD, compared to 6 (0 to 8) in C ($P = 0.004$ and $P < 0.001$, respectively). Postoperative methadone (mg/kg) required during the first 12 h was 0.5 (0 to 1.4) in Group B ($P = 0.003$) and 0 (0 to 0) in BD ($P < 0.001$), compared to C ($P = 0.003$ and $P < 0.001$, respectively).

Conclusion

An ESP block with bupivacaine, with or without dexmedetomidine, was associated with a reduction in perioperative opioid consumption and provided effective acute pain control.

Résumé

Effets analgésiques périopératoires du bloc des érecteurs du rachis avec de la bupivacaïne ou de la bupivacaïne-dexmédétomidine chez les chiens subissant une hémilaminectomie : un essai contrôlé randomisé

Objectif

Comparer les besoins périopératoires en opioïdes chez les chiens recevant un bloc des érecteurs de la colonne vertébrale (ESP) avec de la bupivacaïne, avec ou sans dexmédétomidine, et un groupe témoin.

Animaux et procédure

Trente chiens adultes en bonne santé appartenant à des clients subissant une hémilaminectomie ont été inclus dans cette étude clinique randomisée, prospective et en aveugle. Les chiens ont été répartis au hasard dans 1 des 3 groupes de traitement : groupe B, bloc ESP avec bupivacaïne; groupe BD, bloc ESP avec bupivacaïne et dexmédétomidine; et groupe C, témoin. L'analgésie de secours peropératoire et postopératoire consistait respectivement en fentanyl et en méthadone. La douleur postopératoire a été évaluée à l'aide du formulaire abrégé de l'échelle de mesure de la douleur de Glasgow (CMPS-SF).

Clinical Science and Services, The Royal Veterinary College, Hawkshead Lane, Hatfield AL7 9TA, UK.

Address all correspondence to Bartolome Rico Pérez; email: bricoperez20@rvc.ac.uk

Résultats

Dans le groupe BD, 0/10 chiens ont eu besoin de fentanyl peropératoire, contre 9/10 dans le groupe C ($P < 0,001$), tandis que 1/10 ont eu besoin de méthadone postopératoire, contre 9/10 dans le groupe B ($P = 0,003$) et 10/10 dans le groupe C ($P < 0,001$). La quantité totale de fentanyl peropératoire (μg/kg) était de 0 (0 à 4) dans le groupe B et de 0 (0 à 0) dans le groupe BD, contre 6 (0 à 8) dans le groupe C ($P = 0,004$ et $P < 0,001$, respectivement). La méthadone postopératoire (mg/kg) nécessaire au cours des 12 premières heures était de 0,5 (0 à 1,4) dans le groupe B ($P = 0,003$) et de 0 (0 à 0) dans le groupe BD ($P < 0,001$), par rapport au groupe C ($P = 0,003$). et $P < 0,001$, respectivement).

Conclusion

Un bloc ESP avec de la bupivacaïne, avec ou sans dexmédétomidine, a été associé à une réduction de la consommation peropératoire d'opioïdes et a permis un contrôle efficace de la douleur aiguë.

(Traduit par Dr Serge Messier)

Can Vet J 2024;65:473–480

Introduction

The erector spinae plane (ESP) block is an injection of local anesthetic (LA) between the erector spinae muscle group and the transverse processes of the thoracic vertebrae (1). In veterinary medicine, the ESP block was recently described in 4 cadaveric studies in dogs, at the thoracic and lumbar levels, using various approaches (parasagittal and transverse) (2–5). Although controversies regarding the mechanism of action have been discussed, these studies reported consistent staining of the dorsal branches of the spinal nerves (DBSN). In particular, medial and lateral DBSN innervate the epaxial muscles, the vertebral laminae, the facet joints, and the skin of the dorsolateral aspect of the trunk (6). Therefore, an ultrasound-guided ESP block has been used to provide analgesia in dogs undergoing hemilaminectomy (7–9). A preoperative ESP block in dogs undergoing hemilaminectomy was associated with a reduced incidence of intraoperative bradycardia and hypotension, a lower perioperative opioid requirement, and a shorter interval to voluntary food intake (7–10). However, these findings should be interpreted with caution due to their retrospective nature.

The combination of LA with α2-adrenoceptor agonists, such as dexmedetomidine, has recently gained attention as an approach to prolong the sensory nerve blockade. In particular, perineural injections of dexmedetomidine (0.5 or 1 μg/kg) combined with 0.5% ropivacaine or 0.5% bupivacaine, respectively, improved the sensory duration of femoral and sciatic nerve blocks in dogs, with no reported adverse effects (11,12).

After an online literature search using PubMed (http://www.pubmed.gov/), ScienceDirect (https://www.sciencedirect.com), Wiley Interscience (https://onlinelibrary.wiley.com), and Google Scholar (https://scholar.google.com) databases, the authors did not identify any prospective study evaluating the analgesic efficacy of the ESP block in dogs undergoing hemilaminectomy. The primary aim of this study was to evaluate the postoperative methadone requirements in dogs receiving an ESP block *versus* those not receiving any block, and the secondary aim was to investigate the effects of combining dexmedetomidine with bupivacaine for the ESP block. We hypothesised lower postoperative methadone requirements in dogs receiving the block, and a longer analgesic effect when dexmedetomidine was added to the LA.

Materials and methods

Animals

The study was approved by the Institutional Animal Care and the Clinical Research and Ethical Review Board at The Royal Veterinary College; and signed, written, informed consent was obtained from all owners before the dogs entered the study. This study is reported following the CONSORT guidelines (13) and a flow diagram is provided (Figure 1).

The study included 30 dogs with acute progressive painful/non-painful T3-L3 myelopathy. Physical examination, venous blood gas, creatinine, PCV, and total solids were used to assess the health statuses of the dogs. Inclusion criteria were client-owned dogs > 1 y of age; American Society of Anesthesiologists physical status classification II to III; and intervertebral disc extrusion Hansen Type I or II undergoing thoracic, lumbar, or thoracolumbar hemilaminectomy. Dogs were excluded if they were receiving any behavior-modifying drugs, had the presence of dermatological lesions in the region of the dorsum, required intramuscular premedication, were deep pain negative, or required hemilaminectomy on > 2 consecutive spaces.

Anesthesia

All dogs included in the study underwent magnetic resonance imaging (MRI) for diagnosis and neurolocalization of the intervertebral disc extrusion, followed by surgery under the same general anesthetic.

After aseptic preparation, a catheter was placed in the cephalic vein. The dog was then premedicated intravenously (IV) with 0.2 mg/kg methadone and 1 μg/kg dexmedetomidine. After 10 min, preoxygenation was performed using a flow-by technique and administering oxygen at 6 L/min for 3 min. Anesthesia was induced with propofol titrated to effect, and then dogs were intubated and general anesthesia was maintained with 2.5% sevoflurane in 100% oxygen (initial flow rate of 4 L/min). Volume-control ventilation was set at a tidal volume of 10 mL/kg I:E (inspiratory:expiratory) 1:2, and the respiratory rate was adjusted to maintain normocapnia ($P_E'CO_2$ 35 to 45 mmHg).

Electrocardiography, heart rate (HR), noninvasive blood pressure, esophageal temperature (T), respiratory rate, capnography, $P_E'CO_2$, pulse oximetry, and end tidal sevoflurane

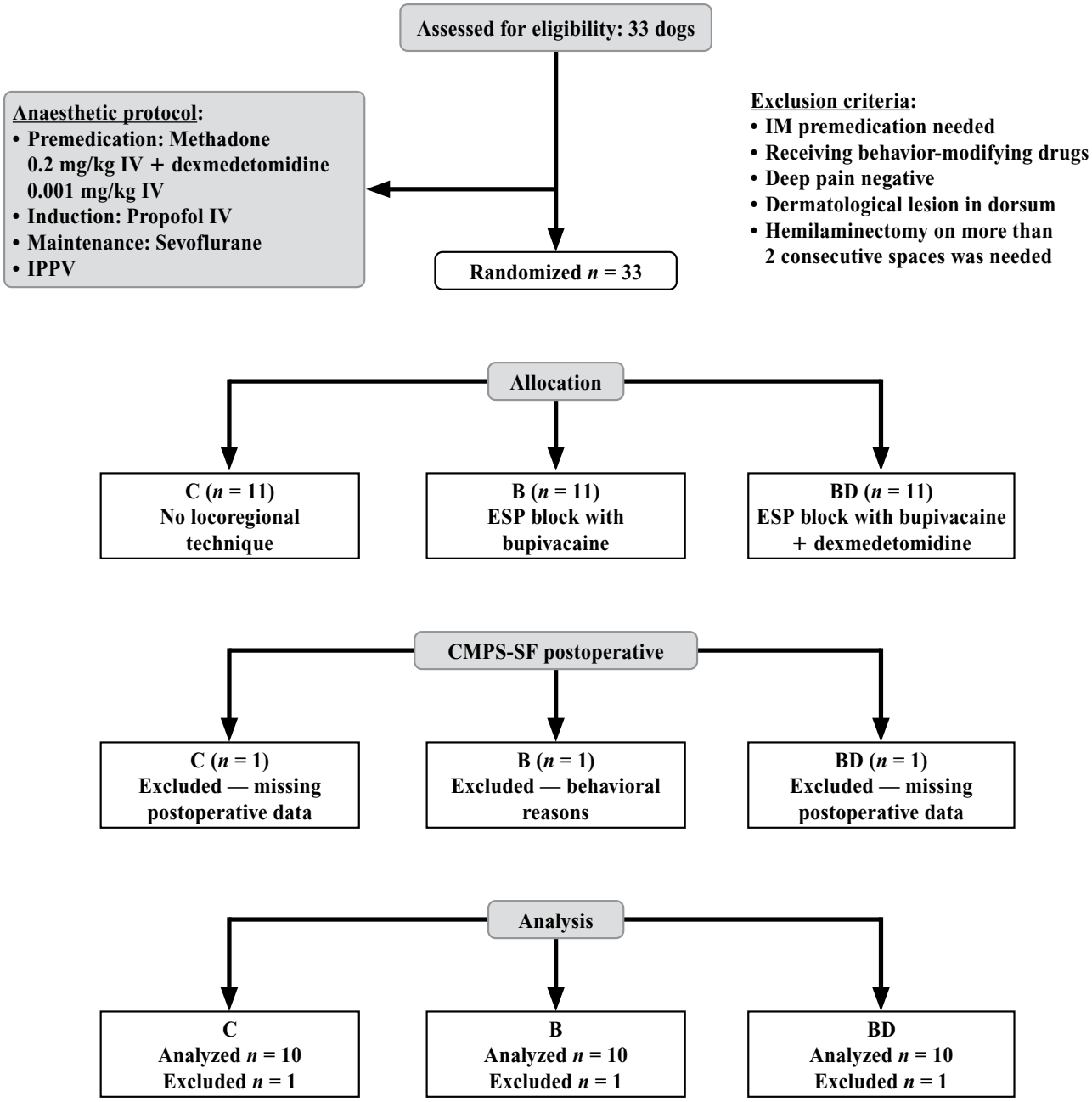

Figure 1. Consolidated Standards of Reporting Trials (CONSORT) flow diagram of the progress through the phases of a parallel randomized trial of 3 groups.

B – Bupivacaine; BD – Bupivacaine + dexmedetomidine; C – Control; CMPS-SF – Glasgow Composite Measure Pain Scale; ESP – Erector spinae plane; IM – Intramuscular; IPPV – Intermittent positive-pressure ventilation; IV – Intravenous.

(Fe′Sevo) were measured and recorded every 5 min from induction to the end of the procedure. Two multiparametric monitors (Expression IP5 multiparametric monitor; MRI-Devices, Philips, Farnborough, UK and Datex-Ohmeda S/5; Datex-Ohmeda, Buckinghamshire, UK) were used during MRI and surgery, respectively. The only vital parameter not monitored during the MRI scan was the ECG. The size of the blood pressure cuff was based on published guidelines and placed in the antebrachium area (14). Hartmann's solution was administered at 5 mL/kg per hour. After MRI, the dog was transferred to an anesthesia preparation room where the hair was clipped and the skin aseptically prepared. At this stage, the anesthetist (anesthesia nurse or veterinarian) in charge of the case was asked to leave the room to remain blinded with regards to the experimental intervention. The dog was then randomly allocated to a treatment group and the treatment completed. Thereafter, the dog was transferred to the surgical theatre and connected to a rebreathing system and ventilator. Active warming was provided during surgery in case of hypothermia ($T < 37°C$).

During general anesthesia, hypotension was defined as mean arterial blood pressure (MAP) < 60 mmHg (15) and treated

after 2 consecutive readings. If bradycardia (HR < 60 bpm) was present at the time of hypotension, glycopyrrolate was administered IV at 10 μg/kg.

Experimental protocol

Prior to hemilaminectomy, an online tool (www.randomizer.org) was used to randomly assign dogs to 1 of the 3 study groups: bupivacaine (B), bupivacaine + dexmedetomidine (BD), and control (C). In Group B, an ultrasound-guided ESP injection with bupivacaine (Marcain Polyamp Steripack 0.5%; Pfizer), 0.4 mL/kg, was administered; in Group BD, the same block was administered using dexmedetomidine, 1 μg/kg, diluted in bupivacaine 0.5%, with a total volume of 0.4 mL/kg. No locoregional technique was used in dogs in Group C. All blocks were performed unilaterally by 3 anesthesia residents trained for this technique under the direct supervision of a European College of Veterinary Anaesthesia and Analgesia diplomate, using parasagittal approaches described by Portela *et al* (2020) (3) for the thoracic area, and by Medina-Serra *et al* (2021) (4) for the lumbar region.

Ultrasound-guided blocks were completed using a 15-6 MHz linear transducer (SonoSite, Bothell, Washington, USA) and the last rib as the reference anatomical landmark to identify the correct space. The target vertebral chosen was the cranial in the surgical site (*i.e.,* T13 in case of T13-L1 hemilaminectomy; L1 in case of L1-L3 hemilaminectomy). The transducer was positioned longitudinal to the spine on the site to be operated and was moved medially until the transverse process was visualised. Using an in-plane technique, an 8-centimeter echogenic insulated needle (22-gauge, 80-millimeter Ultraplex 360 needle; B. Braun Medical, Chapeltown, UK) was advanced through the erector spinae musculature in a craniodorsal-to-caudoventral direction until the tip was visualized in contact with the dorsal aspect of the transverse process. Hydrodissection of the thoracolumbar fascia after a test volume injection of 0.5 mL of LA subtracted from the total volume was used to confirm correct positioning of the needle. Only 1 test injection was done in each case.

Once in the operating theatre, for all dogs, Fe′Sevo was initially targeted at a value of 2.3% before skin incision, to achieve an adequate plane of anesthesia, and then maintained during surgical procedure based of clinical endpoints (blood pressure, palpebral reflex, jaw tone). Five minutes before skin incision, baseline values of HR and MAP were recorded. Rescue analgesia was fentanyl, 2 μg/kg, IV, when a 20% increase above baseline was observed in HR and/or MAP (16). If 3 fentanyl doses were required within 20 min, a constant rate infusion (CRI) of fentanyl, 12 μg/kg per hour, was started. At tracheal extubation, a recovery score was assigned by the main anesthetist, using a published scale (17): 1 — calm, 2 — transient whining or limb movement, 3 — uncoordinated behavior, 4 — agitation. Dogs with a score > 2 received dexmedetomidine, 1 μg/kg, IV.

Postoperative pain assessments were completed by 4 qualified veterinary nurses unaware of the study group allocation, using the short form of the Glasgow Composite Measure Pain Scale (18). Pain scores were assessed immediately after extubation, every hour during the first 4 h post-extubation, and every 4 h during the following 20 h. In case of a score above 5/20, methadone, 0.2 mg/kg, IV, was administered as rescue analgesia. In case of a pain score remaining above the threshold value 20 min after methadone administration, a fentanyl CRI at 3 to 6 μg/kg per hour was started after a loading dose of 2 μg/kg fentanyl, IV. The total amount of opioids required during the first 24 h post-hemilaminectomy was recorded. Non-opioid analgesic drugs such as gabapentin, paracetamol, and NSAIDs, were included in the postoperative analgesic protocol, based on preference of the neurologist, who was blinded to group allocation.

Statistical analyses

Sample size calculation using *a priori* analysis based on data retrospectively collected from the same institution, assuming probability (power) of 0.8 and α of 0.05, concluded that 10 dogs per group were necessary. The total dose of postoperative methadone was chosen as the outcome of interest for power calculation, assuming a standard deviation of 0.6, based on a retrospective analysis comparing dogs receiving systemic analgesia or an ESP block with bupivacaine for hemilaminectomy during the first 12 h postoperatively. A *post-hoc* analysis was done to confirm the adequacy of the sample size. Dedicated statistical software was used for sample size calculation and *post-hoc* power analysis (G*Power version 3.1.9.6; http://www.gpower.hhu.de/en.html).

Data distribution was assessed with Shapiro-Wilk test, with data presented as median (range). Chi-squared and Fisher exact tests for pairwise comparisons, with Bonferroni correction, were used to compare breed distribution; target vertebra for ultrasound-guided needle placement (thoracic *versus* lumbar); number of intervertebral discs affected (1 *versus* 2); incidence of hypotension and hypothermia; number of dogs requiring fentanyl, methadone, any rescue opioid; and postoperative non-opioid analgesic drug distribution among groups. Differences in age, weight, duration of general anesthesia, surgical time, baseline HR and MAP, recovery score, total intraoperative dose of fentanyl (excluding CRIs), and postoperative total dose of methadone (total and within 12 h after extubation) were assessed using Kruskal-Wallis and Dunn tests, with Bonferroni correction for pairwise comparisons.

Kaplan-Meier curves were used to assess differences in time to rescue analgesia, both in the intra- and postoperative periods. Log-rank test and Bonferroni correction were used for pairwise comparison between curves. Statistical significance was considered with $P < 0.05$.

Data analyses were conducted using SPSS software (Version 28; IBM, Armonk, New York, USA). Graphs were generated with Prism software (Version 9.4.1; GraphPad, San Diego, California, USA).

Results

A total of 33 dogs were recruited for this study and allocated to groups with a ratio of 1:1:1. A total of 3 dogs were excluded from the study: 1 due to behavioral reasons that did not allow a reliable postoperative pain assessment, and 2 due to missing postoperative data. Descriptive statistics are in Tables 1 and 2. There were no significant differences among groups for age, weight,

Table 1. Descriptive statistics of the studied population of dogs. Results expressed as median (range).

Variable	Group C (n = 10)	Group B (n = 10)	Group BD (n = 10)	P-value
Age (y)	5.5 (4 to 8)	5 (3 to 7)	4.5 (3 to 12)	0.449
Weight (kg)	6.45 (3.5 to 11.3)	9.65 (4.88 to 40)	10.2 (4.8 to 16.2)	0.071
GA duration (min)	215 (180 to 240)	195 (100 to 270)	180 (135 to 320)	0.365
Surgical time (min)	165 (120 to 210)	150 (60 to 240)	120 (70 to 300)	0.412
Baseline HR (bpm)	55 (35 to 90)	53 (40 to 83)	58 (35 to 80)	0.810
Baseline MAP (mmHg)	75 (60 to 90)	75 (60 to 85)	80 (65 to 90)	0.592
Median FE′Sevo (%)	2.3 (2.2 to 2.4)	2.3 (2.2 to 2.3)	2.3 (2.2 to 2.3)	0.909
Recovery score	1 (1 to 2)	1 (1 to 1)	1 (1 to 2)	0.328

Group C — Control; Group B — Erector spinae plane block with bupivacaine; Group BD — Erector spinae plane block with bupivacaine and dexmedetomidine.
FE′Sevo — End tidal sevoflurane; GA — General anesthesia; HR — Heart rate; MAP — Mean arterial blood pressure.

Table 2. Descriptive statistics of the studied population of dogs. Results expressed as number of dogs.

Variable	Group C (n = 10)	Group B (n = 10)	Group BD (n = 10)	P-value
Dachshunds *(n)*	8	4	5	0.259
Hemilaminectomy 2 spaces *(n)*	3	3	3	1.000
Target vertebra for the block (nT; nL)	5T; 5L	5T; 5L	7T; 3L	0.723
Hypothermia *(n)*	7	6	4	0.562
Hypotension *(n)*	3	5	2	0.498
Postoperative gabapentin *(n)*	4	3	4	1.000
Postoperative paracetamol *(n)*	7	6	7	1.000
Postoperative meloxicam *(n)*	7	8	7	1.000

Group C — Control; Group B — Erector spinae plane block with bupivacaine; Group BD — Erector spinae plane block with bupivacaine and dexmedetomidine.
L — Lumbar. T — Thoracic.

Table 3. Dogs requiring perioperative rescue opioids, intraoperative fentanyl, and postoperative methadone boluses administered as rescue analgesia. Results expressed as *n* or median (range).

Variable	Group C (n = 10)	Group B (n = 10)	Group BD (n = 10)
Dogs requiring intraoperative fentanyl *(n)*	9	3	0[a]
Dogs requiring postoperative methadone *(n)*	10	9	1[a,b]
Dogs requiring perioperative opioids *(n)*	10	9	1[a,b]
Intraoperative fentanyl (μg/kg)	6 (0 to 8)	0 (0 to 4)[a]	0 (0 to 0)[a]
First 12 h postoperative methadone (mg/kg)	0.6 (0.2 to 0.8)	0.5 (0 to 1.4)[a]	0 (0 to 1.2)[a]
12 to 24 h postoperative methadone (mg/kg)	0.7 (0 to 0.08)	0.4 (0 to 0.8)	0 (0 to 0.8)[a]
Total 24 h postoperative methadone (mg/kg)	1.3 (0.2 to 1.4)	0.5 (0 to 1.4)	0 (0 to 1.2)[a]

Group C — Control; Group B — Erector spinae plane block with bupivacaine; Group BD — Erector spinae plane block with bupivacaine and dexmedetomidine.
[a] Different from Group C ($P < 0.05$).
[b] Different from Group B ($P < 0.05$).

duration of general anesthesia, surgical time, pre-incisional HR and MAP, median FE′Sevo, recovery score, breed, number of intervertebral discs affected, target vertebra for ultrasound-guided needle placement, incidence of hypotension and hypothermia, and postoperative non-opioid analgesic drugs. All hypotensive dogs were also bradycardic and received glycopyrrolate shortly after the beginning of the MRI scan. None of the dogs included in the study required additional dexmedetomidine administered during the recovery period. The number of non-opioid analgesic drugs (single treatment, 2 or 3 drugs concurrently) did not differ among groups (P = 0.775).

The number of dogs requiring rescue opioids (intraoperatively, postoperatively and overall), and the amounts of intraoperative fentanyl and postoperative methadone administered as rescue analgesia are in Table 3. The number of dogs requiring intraoperative fentanyl differed for Groups BD and C ($P < 0.001$). The number of dogs requiring methadone postoperatively differed for Groups B and BD (P = 0.003) and Groups BD and C ($P < 0.001$). When the overall perioperative opioid requirement was considered, the number of dogs receiving a rescue dose of either fentanyl or methadone differed between Groups B and BD (P = 0.003) and Groups BD and C ($P < 0.001$).

Dogs in Groups B and BD received a lower dose of fentanyl (μg/kg) than dogs in Group C (P = 0.004 for Group B *versus* C, and $P < 0.001$ for Group BD *versus* C). A fentanyl CRI was required in 3/30 (3.3%) dogs, all allocated to Group C. When the dose of postoperative methadone required during the first 12 h was compared among groups, dogs in Groups BD and B received a lower dose compared to those in Group C (P = 0.004 for Group B *versus* C, and $P < 0.001$ for Group BD *versus* C). Regarding the dose of methadone received between 12 and 24 h

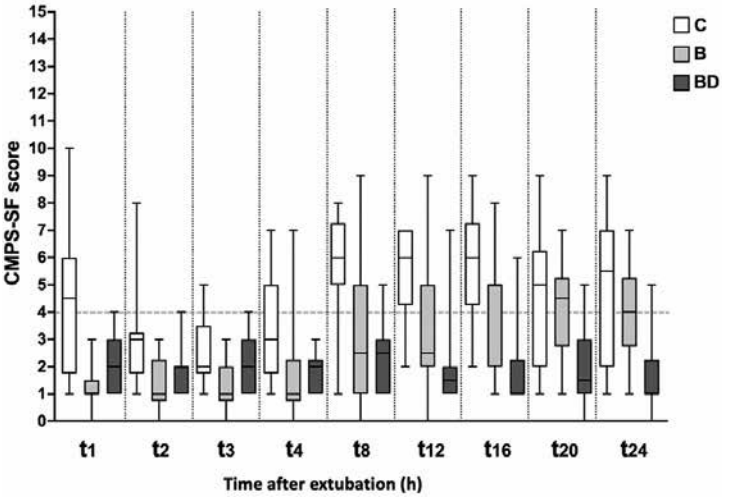

Figure 2. Box-and-whisker plot of Glasgow Composite Measure Pain Scale (CMPS-SF) scores in the 3 groups at the first 4 h post-extubation (t1, t2, t3, t4) and every 4 h (t8, t12, t16, t20, t24) during the first 24 h postoperatively.

C – Control; B – Bupivacaine; BD – Bupivacaine + dexmedetomidine.

The CMPS-SF reported also included dogs that received opioid rescue analgesia as well as other postoperative analgesics.

and the total dose of postoperative methadone, the only significant difference was between Groups BD and C ($P = 0.009$).

Postoperative Glasgow Composite Measure Pain Scale scores are in Figure 2, including data for dogs receiving postoperative rescue analgesia. Differences in pre- and postoperative time to first rescue analgesic treatment in the 3 groups are in Figures 3 and 4. Based on anesthetists' notes, 8/10 dogs in Group C received the first rescue fentanyl bolus at the time of skin incision and 1/10 received it during muscle incision. In Group B, 3/10 dogs received fentanyl during removal of disc material from the spinal canal. No dog reacted intraoperatively in Group BD.

For time-to-event analysis, there were differences among groups in the time of first fentanyl ($P < 0.001$) and methadone ($P < 0.001$) doses. There were differences between Groups B and C ($P = 0.001$) and Groups BD and C ($P < 0.001$) for fentanyl, and between Groups B and C ($P < 0.001$), BD and C ($P < 0.001$), and B and BD ($P = 0.001$) for methadone.

No complications other than those commonly related to general anesthesia were recorded for any of the dogs at any time.

Discussion

The findings of our study agreed with those of previous studies (8,9) in which an ESP block with bupivacaine reduced the intraoperative rescue fentanyl administration and methadone requirements during the first postoperative 12 h in dogs. Intraoperatively, none of the dogs in Groups B or BD received fentanyl during skin incision and muscle dissection, consistent with cadaveric studies describing staining of the DBSN (2–4).

Methadone use from the 12th to the 24th postoperative hour was significantly lower in dogs from Group BD compared to Group C, but was not different when comparing Groups BD *versus* B, or Groups B *versus* C. Furthermore, the time to first rescue analgesia postoperatively was significantly longer in Group BD compared to Group B and the control group. Although dexmedetomidine could have contributed to prolonging the sensory blockade in Group BD, the limited number of dogs included in the study could have masked differences between groups.

Notwithstanding the paramount role of full μ-agonist opioids in the management of postoperative pain (19), their use can result in several adverse effects. In fact, the use of these drugs in dogs has been associated with higher incidence of bradycardia, hypoventilation, ileus, postoperative nausea and vomiting, dysphoria, prolonged hospitalization, and hyperalgesia (8,20). Although clinical effects of some of these side effects in dogs are still to be clarified, a reduction of the total dose of opioids administered perioperatively could be an important factor in the overall well-being of hospitalized dogs.

Based on our findings, we inferred that the ESP block has an important role in a multimodal analgesic protocol for dogs undergoing hemilaminectomy, and that the adjunction of dexmedetomidine at 1 μg/kg could prolong postoperative analgesia, in agreement with studies in humans (21,22).

There are various theories to explain the analgesic effect of dexmedetomidine, though the actual mechanism remains unclear. First, dexmedetomidine can cause local vasoconstriction, thus delaying absorption and prolonging the effect of the co-administered LA (23). The second theory regards the direct effects of dexmedetomidine on peripheral nerve activity, such as blockade of the hyperpolarization-activated cation current and attenuation of the acute LA-induced perineural inflammation (24). A third hypothesised mechanism is inhibition of nociception by reducing release of neurotransmitters such as substance P and glutamate (25).

Paravertebral and epidural spread of the injectate have been described in humans after ESP block (26). Although

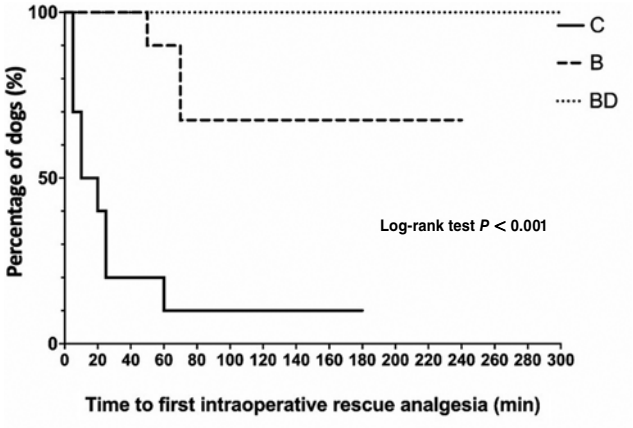

Figure 3. Kaplan-Meier curve of the time to first intraoperative fentanyl rescue bolus in the 3 groups over time, defined as minutes intraoperatively.

C – Control; B – Bupivacaine; BD – Bupivacaine + dexmedetomidine.

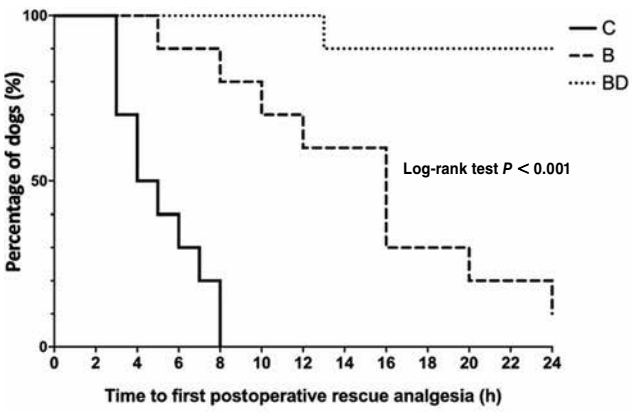

Figure 4. Kaplan-Meier curve of the time to first postoperative methadone rescue bolus in the 3 groups over time, defined as hours postoperatively.

C – Control; B – Bupivacaine; BD – Bupivacaine + dexmedetomidine.

never reported in canine cadaveric studies using a parasagittal approach, this spread could be a mechanism of action of this block in our population.

The dose of dexmedetomidine chosen for this study was based on a study by Marolf *et al* (2021) (11), in which the duration of sensory blockade was prolonged when this drug was co-administered perineurally at this dose, without any reported adverse effects. Further studies testing various doses of dexmedetomidine as an adjunctive drug for this block are needed to investigate any dose-effect relationship in dogs.

The present study had several limitations. The sample size was adequate for assessing the number of methadone doses needed postoperatively, but statistical power calculation could be lower for other comparisons. The main limitation of our study was the inclusion of dogs undergoing hemilaminectomy in various thoracic and lumbar intervertebral spaces, representing a lack of standardization of the surgical site. Anatomical differences among regions may have an important role in the spread of LA in the fascial plane. Portela *et al* (2020) (3) reported staining of 4 (2–7) medial branches and 4 (2–5) lateral branches after injection of 0.3 mL/kg at the level of the ninth thoracic vertebra, whereas Medina-Serra *et al* (2021) (4) reported staining of 2 (2–3) DBSN using the transversal approach and 0 (0–3) DBSN using the parasagittal approach with the injection of 0.4 mL/kg at the level of the 4th lumbar vertebra. In our study, there were no significant differences among groups in the distribution of thoracic *versus* lumbar surgery. However, as this was not considered the main outcome of the study, the sample size may not have been adequately powered for these data.

Although the distribution of breeds was not different among groups from a statistical perspective, most dogs in Group C were dachshunds. One limitation of the study is a possible Type II error when assessing differences in the number of dogs of this breed between groups, since the study was not powered for this outcome. Dachshunds have previously been associated with lower HR under anesthesia (27), which may have affected the intraoperative rescue analgesia threshold.

Another limitation was the use of dexmedetomidine as premedication agent in all dogs, which could have affected the duration of nerve blockade in both Groups B and BD. There are some reports of a prolonged sensory and motor blockade when dexmedetomidine was administered IV in dogs (28). However, the dose used in the present study was lower than that in a previous paper (11) that did not report any effect of systemic dexmedetomidine administered IV at least 1 h before a peripheral block in dogs.

Concurrent administration of other analgesic drugs, such as gabapentin, meloxicam, and paracetamol, is considered another limitation of our study since these have disparate analgesic and sedative properties that could have influenced the study results. Thus, the postoperative pain scores should be interpreted with caution. However, there were no significant differences among groups in concurrent drug administration.

A previous retrospective study in dogs investigating the effects of ESP block reported postoperative benefits up to 48 h after surgery (8). In light of this finding, limiting the postoperative period of this study to the first 24 h post-extubation could be considered another limitation. A longer study interval may have yielded more data about the long-term effects of the dexmedetomidine co-administration. A final limitation was the heterogeneity of neurosurgeons and anesthetists involved with the cases. Even though all used the same surgical technique, differences in level of experience and tissue handling may have affected the extent of tissue trauma, nociception, and pain among dogs. Three anesthesia residents performed the ESP blocks, and this variability could have influenced the effectiveness of the technique. To minimize this potential source of bias, all injections were completed under the supervision of the same European College of Veterinary Anaesthesia and Analgesia diplomate.

Notwithstanding the limitations discussed, the authors believe that the overall potential for bias can be considered inherent to the nature of the study, as the design reflected clinical practice in the referral hospital where the study was done. Further prospective studies are needed to evaluate the efficacy of ESP block

with bupivacaine and dexmedetomidine in dogs undergoing hemilaminectomy.

In conclusion, the ESP block with bupivacaine, with or without dexmedetomidine, was associated with a reduction in the perioperative opioid consumption in dogs undergoing hemilaminectomy, and provided effective acute pain control. CVJ

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Article

Serum concentrations of selenium, copper, and zinc in neonatal foals: Influence of failure of passive transfer and age-related changes

Lydia T. Stahl, Anja Müller, Judith Krohn, Kathrin Büttner, Axel Wehrend

Abstract

Background

An adequate supply of trace elements is very important for equine neonates, as deficiencies can lead to health problems and even death.

Objective

This study investigated serum concentrations of selenium (Se), copper (Cu), and zinc (Zn) in neonatal foals up to the 8th day of life. The influences of disease, age, and failure of passive transfer (FPT) on these concentrations were analyzed.

Animals and procedure

Serum concentrations of Se, Cu, and Zn were determined from blood samples of 93 foals by means of inductively coupled plasma mass spectrometry. The foals were divided into 2 groups based on health status: clinically sick (n = 51) and clinically healthy (n = 42). The latter group was further divided into foals with FPT (n = 20) and those without (n = 22).

Results

Mean serum concentrations for Se, Cu, and Zn were 60 ± 40 μg/L, 0.25 ± 0.22 mg/L, and 605 ± 285 μg/L, respectively. A significant influence of age on serum Cu concentration was observed ($P < 0.0001$). No differences were observed between any of the serum concentrations in clinically sick and clinically healthy foals on the 1st day of life. The FPT status was not associated with reduced serum concentrations of Se, Cu, or Zn.

Conclusion and clinical relevance

It is not necessary to supplement trace elements in all foals with FPT.

Résumé

Concentrations sériques de sélénium, de cuivre et de zinc chez les poulains nouveau-nés : influence de l'échec du transfert passif et des changements liés à l'âge

Contexte

Un apport suffisant en oligo-éléments est très important pour les nouveau-nés équins, car des carences peuvent entraîner des problèmes de santé, voire la mort.

Objectif

Cette étude a examiné les concentrations sériques de sélénium (Se), de cuivre (Cu) et de zinc (Zn) chez les poulains nouveau-nés jusqu'au 8ème jour de vie. Les influences de maladies, de l'âge et de l'échec du transfert passif (FPT) sur ces concentrations ont été analysées.

Animaux et procédure

Les concentrations sériques de Se, Cu et Zn ont été déterminées à partir d'échantillons de sang de 93 poulains au moyen d'une spectrométrie de masse à plasma à couplage inductif. Les poulains ont été divisés en 2 groupes en fonction de leur état de santé : cliniquement malades (n = 51) et cliniquement sains (n = 42). Ce dernier groupe a été divisé en poulains avec FPT (n = 20) et ceux sans (n = 22).

Veterinary Clinic for Reproductive Medicine and Neonatology, Justus-Liebig-University Giessen, Frankfurter Str. 106, 35392 Gießen, Germany (Stahl, Krohn, Wehrend); Vet Med Labor GmbH IDEXX Laboratories, Humboldtstrasse 2, 70806 Kornwestheim, Germany (Müller); Unit for Biomathematics, Justus-Liebig-University Giessen, Frankfurter Str. 95, 35392 Gießen, Germany (Büttner).

Address all correspondence to Lydia T. Stahl; email: Lydia.T.Stahl@vetmed.uni-giessen.de

Résultats

Les concentrations sériques moyennes de Se, Cu et Zn étaient respectivement de 60 ± 40 µg/L, 0,25 ± 0,22 mg/L et 605 ± 285 µg/L. Une influence significative de l'âge sur la concentration sérique de Cu a été observée ($P < 0{,}0001$). Aucune différence n'a été observée entre les concentrations sériques chez les poulains cliniquement malades et cliniquement sains au premier jour de leur vie. Le statut FPT n'était pas associé à une réduction des concentrations sériques de Se, Cu ou Zn.

Conclusion et pertinence clinique

Il n'est pas nécessaire de supplémenter tous les poulains en oligo-éléments avec FPT.

(Traduit par Dr Serge Messier)

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Introduction

Foals are susceptible to various diseases very early in life, which are often accompanied by nonspecific signs such as poor feed intake, exhaustion, or problems in rising (1,2). Due to the epitheliochorial structure of the equine placenta, intrauterine transfer is restricted. Although nutrients and trace elements are partially transferred *in utero,* there is (almost) no intrauterine transfer of immunoglobulins. Foals are born almost agammaglobulinemic (3–5). The causes of neonatal diseases can be manifold, but besides an inadequate supply of immunoglobulins, an inadequate supply of trace elements can promote diseases and lead to severe consequences (1,2,6). At present, there are limited data investigating serum concentrations [especially of copper (Cu) and zinc (Zn)] in equine neonates during the first 8 d of life (7,8).

Selenium (Se) is an antioxidative essential trace element that protects the body from oxidative stress. It is present in > 30 selenoproteins, some of which have an enzymatic effect (5). Compared to other tissues, muscle cells are supplied with fewer antioxidants as they have mainly Se-dependent glutathione peroxidase and only a small number of vitamin E-mediated defence mechanisms. This renders the musculature a vulnerable organ for oxidative stress-induced damage (9). Selenium also plays an important role in thyroid metabolism and body growth by activating thyroxine to the active form triiodothyronine (3). Clinical manifestations of Se deficiency are primarily observed in neonates. The typical clinical condition is referred to as rhabdomyolysis or, more specifically, nutritive myodegeneration (1). Rhabdomyolysis in foals is often caused by an intrauterine Se deficiency as a result of an inadequate supply of Se in the diet of the dam during pregnancy (1). Selenium also plays an important role in the function of the immune system. Work by Montgomery *et al* (10) focused on the relationship between Se supplementation and foal immunity. The authors showed an effect of Se supplementation of the mare depending on Se source (organic or inorganic) and gene expression of certain lymphocyte cytokines in the foal (10). This highlighted the importance of adequate nutrition of the mare for preventing various diseases due to different mechanisms of action of Se.

Copper is an essential trace element involved in many metabolic processes. It is a component of many important enzymes and plays a role in haemoglobin synthesis, the immune system, bone metabolism, growth, the nervous system, and hair pigmentation (2). Copper deficiency is associated with poor immune system function, vascular degeneration due to lack of cross-linking of elastin and collagen fibres, and depigmentation of skin and hair. Other signs include microcytic hypochromic anaemia, skeletal damage, hypercholesterolaemia, neutropenia, diarrhea due to atrophy of the villi, infertility due to impaired steroid synthesis, hyperextension of limbs, and demyelination in the central nervous system with immobilisation, blindness, and death (enzootic ataxia) (2,9). Increased skeletal damage in the form of *osteochondrosis dissecans* has been observed in foals experiencing Cu deficiency (6,11), though a more recent study could not find a clear relationship between the 2 factors (12).

Zinc is one of the most abundant essential trace elements in the body, second only to iron (13). It plays a key role in the physiological function of the immune system and has antioxidant effects in the metabolization of fats, carbohydrates, neurotransmitters; collagen synthesis and degradation; and testicular development, as it converts testosterone to the active metabolite dihydrotestosterone (2,3). Consequences of a Zn deficiency include growth disorders in young animals, a disturbance of sexual development in male animals, problems with skin and hair coat in the form of skin lesions, parakeratosis due to disturbance of keratinisation, delayed wound healing, and alopecia (2,9).

The concentration of trace elements in colostrum is highest and decreases measurably within the first few days of lactation (3,8,14,15). Breedveld *et al* (15) investigated the concentrations of Se, Cu, and Zn in mares' milk at the time of birth and in the weeks after birth, and compared them with the serum concentrations in the foals obtained at the same times. There was a weak correlation between mares' milk and foals' serum concentration for Se and Zn and a strong negative correlation for Cu (15). At present, research investigating the concentration of trace elements in foals with failure of passive transfer (FPT) is lacking. Failure of passive transfer occurs as a consequence of an insufficient intake of maternal colostrum ingredients. This may be due to poor colostrum quality, late colostrum intake, or diseases of the foal, and it is measured by the amount of IgG absorption (7,16).

This study aimed to report serum concentrations of Se, Cu, and Zn in neonatal foals up to 8 d of life, and to investigate differences in concentrations between sick and healthy neonatal foals. Furthermore, we wanted to clarify whether it is necessary to supplement all foals with FPT with trace elements.

Materials and methods

Serum samples from 93 foals born between April 16, 2019 and July 3, 2020 were examined. The group consisted of warmbloods (73.1%), small horses and ponies (18.3%), thoroughbreds (5.4%), and draft horses (3.2%). These equine neonates

Table 1. Trace element concentrations in serum of neonatal foals presented to or born at the Veterinary Clinic for Reproductive Medicine and Neonatology, Justus-Liebig-University Giessen, Germany, between April 2019 and July 2020.

Trace element	Group	*n*	Mean	SD	Median	Range
Selenium (μg/L)	All foals	93	60	40	52	9 to 261
	Healthy foals on the 1st day of life	31	67	33	64	15 to 177
	Sick foals on the 1st day of life	28	52	46	44	9 to 261
	Clinically healthy foals without FPT	22	72	33	66	36 to 177
	Clinically healthy foals with FPT	20	66	32	66	15 to 128
Copper (mg/L)	All foals	93	0.25	0.22	0.17	0.08 to 1.59
	Healthy foals on the 1st day of life	31	0.16	0.05	0.14	0.09 to 0.28
	Sick foals on the 1st day of life	28	0.18	0.08	0.16	0.08 to 0.51
	Clinically healthy foals without FPT	22	0.19	0.10	0.14	0.09 to 0.50
	Clinically healthy foals with FPT	20	0.18	0.09	0.15	0.10 to 0.49
Zinc (μg/L)	All foals	93	605	285	564	167 to 1960
	Healthy foals on the 1st day of life	31	641	228	637	279 to 1310
	Sick foals on the 1st day of life	28	572	304	532	167 to 1720
	Clinically healthy foals without FPT	22	653	240	665	301 to 1310
	Clinically healthy foals with FPT	20	651	240	610	279 to 1380

FPT — Failure of passive transfer.

(up to 8 d of age) were either born in the Veterinary Clinic for Reproductive Medicine and Neonatology, Justus-Liebig-University Giessen, Germany, or admitted because of disease. Foals that had already been treated by plasma transfusion or application of trace elements before presentation were excluded from the study.

The foals were divided into 2 groups: clinically sick (n = 51) and clinically healthy (n = 42). The group of clinically healthy foals was further divided into foals with (n = 20) and without (n = 22) FPT. Sick foals were not subdivided, to avoid the confounding effect of disease. Clinically healthy foals consisted of 31 foals seen on the 1st day of life, 6 foals on the 2nd day of life, 2 foals on the 3rd day of life, 1 foal on the 4th day of life, and 2 foals seen on the 5th day of life. Clinically sick foals consisted of 28 foals seen on the 1st day of life, 12 foals on the 2nd day of life, 5 foals on the 3rd day of life, 2 foals on the 4th day of life, and 2 foals each seen on the 7th or 8th days of life.

Foals were classified as "clinically sick" or "clinically healthy" based on clinical examination and laboratory diagnostic tests. Furthermore, the "Giessener Vorsorgeschema I, II" was used to determine whether a foal developed normally or was clinically sick (17). Recording even subtle abnormalities, the "Giessener Vorsorgeschema" is a scheme for early detection of endangered or highly endangered foals.

Sampling and sample processing

A blood sample from each foal was taken upon arrival at the clinic, as part of the initial examination; or at 18 h after the first colostrum intake, if the foal arrived < 18 h after birth or was born at the clinic. Blood samples were obtained for diagnostic tests or for monitoring response to therapy. Blood remaining from each sample was used for assessment of parameters defined for this investigation. Approval for the study was granted by the supervisory authority (file number: kTV8-2017). Each sample was taken from the jugular vein after disinfection of the collection site using a sterile disposable cannula (Sterican, 18 G × 1.5", Ø 1.2 × 40 mm; B. Braun, Melsungen, Germany) and a 4-milliliter serum tube (Vet Med Labor IDEXX, Ludwigsburg, Germany). Within 1 h following blood collection, samples were centrifuged at 1500 × g for 10 min (centrifuge model 1710, Rotina 35 R; Hettich, Tuttlingen, Germany) and serum was transferred to a 3.5-milliliter polypropylene tube (Sarstedt, Nümbrecht, Germany) and frozen at −18°C.

At IDEXX Laboratories (Kornwestheim, Germany), the concentrations of Se, Cu, and Zn were measured using inductively coupled plasma mass spectrometry (ICP-MS, Varian MS 820; Analytik Jena, Jena, Germany). The intra- and interassay coefficients of variation for Se, Cu, and Zn were 3.8% and 5.3%, 1.6% and 4.2%, and 2.3% and 4.7%, respectively.

To assign the foals to the groups with and without FPT, blood was checked for occurrence of FPT. Failure of passive transfer was defined according to Sievert *et al* (16), who investigated 3 different methods for measuring IgG concentration, using a quantitative ELISA as a reference method. Based on the results for each foal, FPT was diagnosed by an IgG concentration < 400 mg/dL as determined by the SNAP Foal IgG Test (IDEXX Laboratories), a total protein < 51 g/L (measured by hand refractometer HRM18-T; Krüss, Hamburg, Germany), or a globulin concentration (measured indirectly as total protein minus albumin, photometrically by the bromocresol green method) < 24 g/L (16).

Statistical evaluation

Statistical analyses were conducted using the statistical program SAS (18). Descriptive statistics summarizing serum concentrations of the groups and tabulation of means, standard deviations, median values, minima, maxima, and sample sizes were calculated. In the text, trace element concentrations are reported using the median with range and/or mean with standard deviation for ease of comparison with relevant studies. The relationship between age and serum concentration was tested using Spearman's rank correlation. Comparisons using independent samples t-tests were made between serum concentrations of healthy and sick foals on the 1st day of life. In the event of equal variances, a pooled samples t-test was employed; Welch's t-test

was used in the case of unequal variances. In the group of clinically healthy foals, the serum concentrations of foals with and without FPT were compared using a Wilcoxon test.

In each box-and-whisker plot, the central box represents the values from lower to upper quartiles (25th to 75th percentile), the middle line represents the median, and the "×" inside the box represents the mean. The vertical line (*i.e.,* the whisker) extends from the minimum to the maximum value, excluding outliers, defined as values that are smaller than the lower quartile minus 1.5× the interquartile range, or larger than the upper quartile plus 1.5× the interquartile range. Outliers are represented by dots.

Statistical significance was assessed based on $\alpha = 0.05$. Accordingly, results with $P \leq 0.05$ were considered statistically significant.

Results

Trace element concentrations

In foals, the mean Se serum concentration was 60 ± 40 µg/L, mean Cu serum concentration was 0.25 ± 0.22 mg/L, and mean Zn serum concentration was 605 ± 285 µg/L (Table 1).

Influence of age

No statistically significant correlations were identified between age and serum concentrations of Se ($P = 0.84$) or Zn ($P = 0.49$). Copper concentration and age demonstrated a highly significant positive relationship of moderate strength ($r = 0.64$, $P < 0.0001$). The concentrations are shown in Figure 1 and in Table 1.

Influence of disease

To reduce the confounding effect of age, only foals on the 1st day of life were compared in the analysis of influence of disease. There were 31 healthy foals and 28 sick foals in the respective groups. No statistically significant differences were identified between the groups for Se ($P = 0.17$), Cu ($P = 0.19$), or Zn ($P = 0.09$) concentrations. All results are shown in Table 1. Figure 2 shows Se, Cu, and Zn concentrations in healthy and sick foals.

Influence of failure of passive transfer

To avoid confounding effects of disease, only the clinically healthy foals ($n = 44$) were compared in the analysis of influence of FPT. Clinically healthy foals with FPT ($n = 20$) and clinically healthy foals without FPT ($n = 22$) showed no statistically significant differences in serum concentration of Se ($P = 0.49$), Cu ($P = 0.60$), or Zn ($P = 0.77$). Table 1 lists the concentrations and Figure 3 illustrates the serum concentrations of foals without and with FPT.

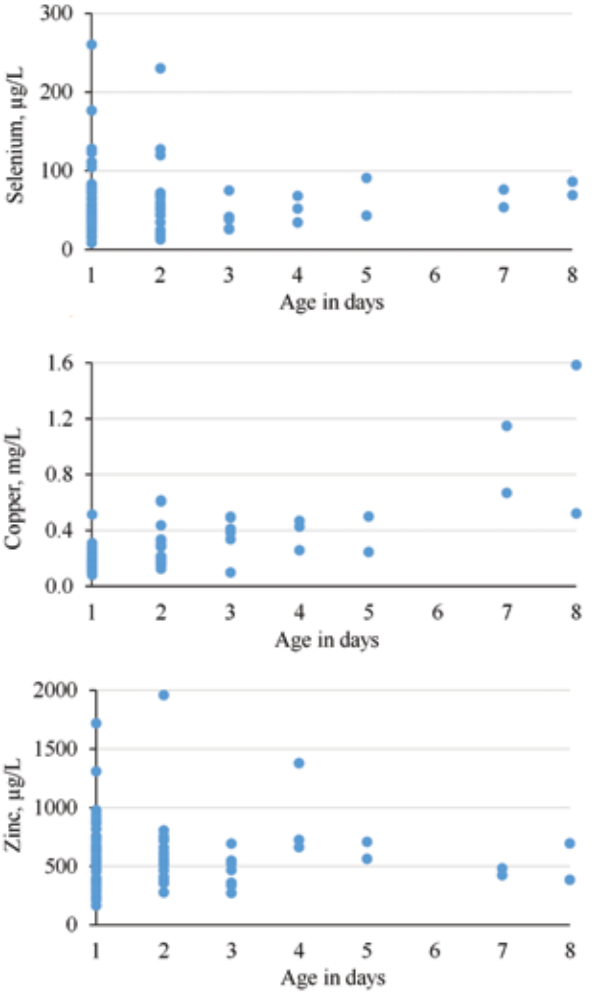

Figure 1. Relationships between age in days and serum concentrations of selenium, copper, and zinc, without consideration of health status, in foals up to 8 d of life (N = 93). Selenium and zinc concentrations demonstrated no statistically significant relationships with age. Copper concentrations increased in the first days of life ($r = 0.64$, $P < 0.0001$). The individual dots represent individual animals.

Discussion

Selenium

Selenium content in the whole body of an animal can be reliably determined using the serum concentration (19). The laboratory-specific reference values for Se from IDEXX Laboratories are 70 to 170 µg/L for adult horses and 50 to 90 µg/L for "foals;" however, the age of "foals" is not specified. The concentrations determined in this study for foals (60 ± 40 µg/L) are similar to those observed by Rauchhaupt (7) (62.7 ± 27.8 µg/L), which were recorded in the serum of 46 foals aged 1 to 3 d using hydride technique atomic absorption spectrometry. Wolff *et al* (20) only examined Se concentrations for foals up to 1 y of age and cited other sources that do not provide detailed age information. Muirhead *et al* (21) determined mean Se concentrations of 0.059 ± 0.053 ppm (equivalent to 59 ± 53 µg/L) in the serum of 50 foals aged 1 to 3 d (measured with atomic absorption spectrometry in Canada), whereby a very high standard deviation was particularly noticeable. Notably, higher Se concentrations are described more frequently in recent studies than in older studies, which possibly reflects the fact that increasing importance has been attributed to an adequate Se supply for pregnant mares over the years (7,8,15,21).

Limited studies investigated the relationship between age and Se concentration. Lee *et al* (14) observed a relatively constant Se concentration within the first 28 d of life. Zentek

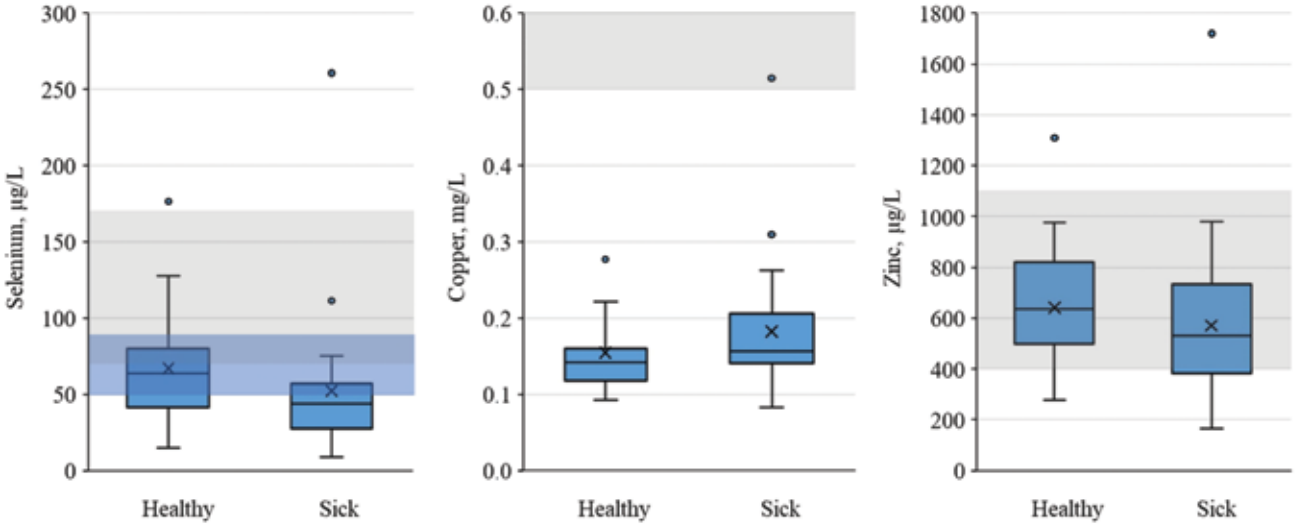

Figure 2. Serum selenium (Se), copper (Cu), and zinc (Zn) concentrations compared between clinically Healthy ($n = 31$) and clinically Sick ($n = 28$) neonatal foals on the 1st day of life. No statistically significant differences were identified between the groups. For each box-and-whisker plot: central box represents 25th to 75th percentile; middle line represents median; "×" inside box represents mean; vertical line represents minimum value to maximum value, excluding outliers, which are represented by dots. Blue area indicates the laboratory-specific reference ranges from the company IDEXX Laboratories for foals (unspecified age): Se: 50 to 90 µg/L, Cu and Zn: no reference ranges given for foals. Gray areas indicate the laboratory-specific reference ranges from the company IDEXX Laboratories for adult horses: Se: 70 to 170 µg/L, Cu: 0.5 to 1.5 mg/L, Zn: 400 to 1100 µg/L.

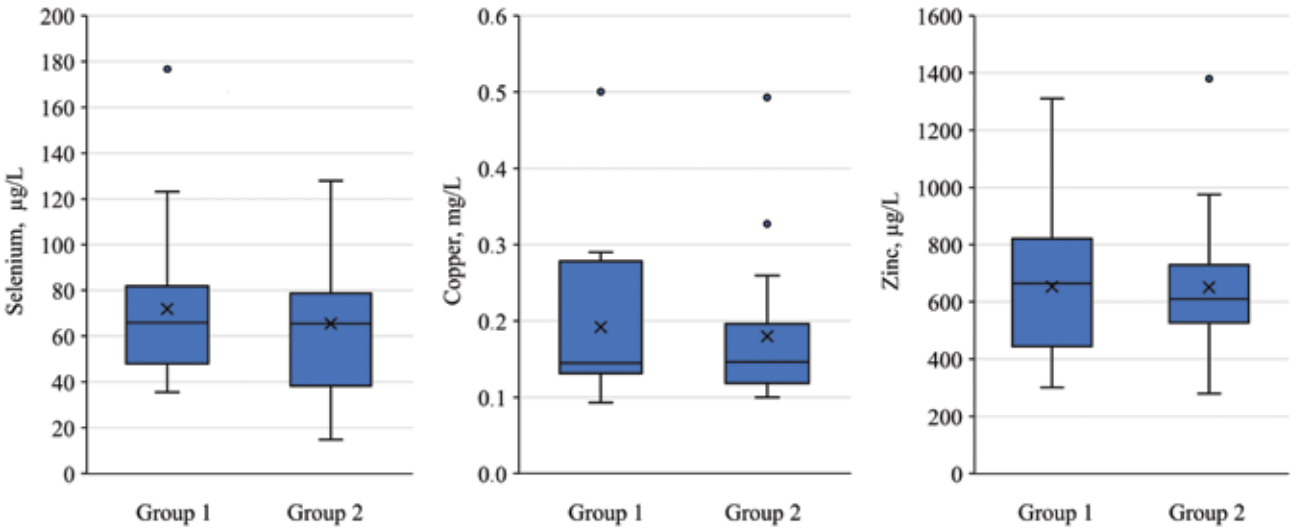

Figure 3. Box plot diagram of selenium, copper, and zinc concentrations in clinically healthy neonatal foals up to 5 d of life. Comparisons are between Group 1 (without failure of passive transfer; $n = 22$) and Group 2 (failure of passive transfer; $n = 20$). No statistically significant differences between the groups were identified. For each box-and-whisker plot: central box represents 25th to 75th percentile; middle line represents median; "×" inside box represents mean; vertical line represents minimum value to maximum value, excluding outliers, which are represented by dots.

et al (4) observed different Se concentrations in a single sampling but concluded that variance in Se concentrations cannot be attributed to the influence of age, instead attributing it to the mare's Se status. The results of our study suggest there were no age-associated differences in Se concentration within the study period.

To analyze the influence of disease in this study, only foals on the 1st day of life were compared, with 31 foals in the healthy group and 28 foals in the sick group. When comparing Se concentrations of healthy and sick foals, no difference was observed ($P = 0.17$). This lack of difference between healthy and sick foals was probably due to the definition of "clinically sick" foals including all animals in need of intensive care. A difference might be observed when restricting the definition to septic foals, as Renko *et al* (22) showed a significant decrease in the concentration of Se and the transport protein selenoprotein P in mice as part of an acute-phase reaction. Nichol *et al* (23) determined that plasma Se concentrations decreased in the context of an acute phase reaction after minor elective surgical procedures in humans. At present, comparable studies in neonatal foals

observing the changes in Se concentrations after an (induced) inflammation process are not available. Montgomery *et al* (10) stated that the source of Se (organic or inorganic) provided to the dam influences gene expression of certain lymphocyte cytokines (interferon gamma, interleukin-2, tumor necrosis factor alpha). In foals, decreased Se concentrations are probably mainly due to a poor supply from the dam. Further studies should investigate the relationship between inflammation and blood Se concentrations in neonatal foals in more detail.

Copper

Evaluation of Cu concentrations revealed very low mean serum concentrations for foals (0.25 ± 0.22 mg/L) compared to adult horses (IDEXX Laboratories reference values: 0.5 to 1.5 mg/L). Rauchhaupt (7) also reported a mean serum concentration of 0.25 ± 0.11 mg/L. In the literature, mean Cu concentrations in the 1st week of life have been reported to be between 0.13 and 0.73 mg/L (15,24,25). Accordingly, age-specific reference values should be established for Cu. However, it should be noted that Cu values should be reported in narrow age ranges due to the sharp increase in concentrations depending on age.

Neonates store Cu in high concentrations in the liver, and Cu serum concentrations appear low in different species and change markedly depending on age (7,15,24–29). In general, serum Cu concentration does not adequately reflect the actual Cu supply (30). In the neonatal phase, there are some differences from the Cu metabolism of adult animals. Embryonic-type Cu metabolism (ETCM) changes to adult-type Cu metabolism (ATCM) in the first weeks of life (26). In the ETCM phase, there is no excretion *via* bile (the main excretory pathway in adult animals) and the gene expression of ceruloplasmin (a transport protein that binds the majority of the Cu in the plasma) is suppressed (26). The mRNA expression of Cu-regulating proteins such as ceruloplasmin, the Wilson protein Atb7b, the antioxidant protein 1, and the Cu transporter Ctr1 increases with age. In rats, there is a sharp drop in liver Cu concentration and an increase in serum Cu concentration after 2 wk (26,27). In this study, a highly significant influence ($P < 0.0001$) of age on Cu concentration was shown in the first 8 d of life, with a correlation coefficient of 0.64, indicating a moderate correlation between age and serum Cu concentration. The change from ETCM to ATCM in foals may be faster or begin earlier than in other species. Further investigation on this specific topic should be done.

No difference in serum Cu concentration was observed between healthy and sick foals ($P = 0.19$). Again, this lack of difference between healthy and sick foals is probably due to the definition of "clinically sick" foals, which was not limited to septic foals. In the case of inflammation, serum Cu concentration is often elevated due to increased ceruloplasmin synthesis in the liver (2,31). Humann-Ziehank *et al* (31) also reported for piglets an increase in serum Cu concentration of about 18% within 4 d of infection. However, the serum Cu concentration in neonates is not suitable as a potential marker for inflammation because the concentration rises sharply in the first days of life. Thus, daily individual reference values would have to be established.

Zinc

Rauchhaupt (7) reported a mean Zn concentration of 611 ± 210 μg/L, which is very similar to the concentrations determined in this study (605 ± 285 μg/L). In the literature, mean Zn concentrations between 732 and 1190 μg/L in the 1st week of life have been described (15,24,25). The significance of Zn concentration in serum for determining Zn content of the whole body has not yet been conclusively clarified, especially in the horse. A reliable biomarker to determine the actual Zn status is still lacking, according to a review by Gammoh and Rink (13).

The laboratory-specific reference values (IDEXX Laboratories) for the Zn concentration of adult horses are 400 to 1100 μg/L. Because the average Zn concentration in the blood of neonatal foals is in this range, no specific reference range for Zn in neonatal foals seems necessary. Zinc is physiologically accumulated in the liver during the fetal period and released postnatally. Within the first 2 wk, Zn concentration in the liver of the rat is halved, which relates to an increase in mRNA expression of a Zn transporter (Znrt1) that is responsible for the export of Zn from the liver. However, the serum Zn concentration does not change during growth (27).

In addition, Breedveld *et al* (15) and Bell *et al* (24) did not find any significant differences in Zn concentrations in foals. This is consistent with the results of our study, which also identified no statistically significant influence of age on Zn concentrations.

The median Zn concentration of sick foals was numerically lower than that of healthy foals (532 *versus* 637 μg/L, $P = 0.09$; Table 1). Studies in adult horses demonstrated decreased Zn concentrations in the context of inflammation, and Murase *et al* (32) recommended serum Zn concentration as a marker of inflammation in thoroughbred horses (32,33). No comparable studies are available for neonatal foals.

Influence of colostrum

The results for clinically healthy foals with and without FPT demonstrated that trace element concentrations in the blood were not associated with a deficient colostrum supply. Breedveld *et al* (15) suggested that concentrations in mares' milk do not provide a good indication of concentrations in foal blood obtained at the same time. Several studies investigated how the nutrition of the dam effects the neonate. Supplementation of Se increases the Se concentration in milk, and therefore increases the Se uptake of the foal (9). Abdelrahmen and Kincaid (34) reported that Se supplementation of the dam significantly increased Se concentration in the dam's blood and in colostrum. Also, the neonates of supplemented dams showed higher Se concentrations in blood and higher Se reserves in the liver (34). An increased supplementation of Cu or Zn did not affect serum Cu or Zn concentrations of dams and foals (15). To determine the Cu status, liver biopsies are more informative, but are not often done due to the invasiveness of the method. Supplementation of Cu in the dam led to increased Cu concentrations in the liver of both dam and foal (28). Marques *et al* (35) reported increased Cu and Zn concentrations in the livers of calves from dams supplemented with organic Cu and Zn in comparison to those with no supplementation.

The results of our study support the assumption that the supply of trace elements to the neonatal foal primarily occurs antepartum, through transplacental means. Supply *via* the colostrum plays a minor role. Consequently, no supplementation of trace elements is necessary if FPT is observed.

In conclusion, the results of this study clearly highlight the need to establish age-specific reference values for foals, especially for serum Se and Cu concentrations. Lower concentrations of Se, Cu, or Zn in the serum were not observed in foals with FPT. Thus, the decision regarding the necessity of a supplementation, especially of Se, should not be solely dependent on the presence of FPT.

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Article

Temporal patterns of bovine leukemia virus infection in dairy herds in Atlantic Canada

Emily E. John, Marguerite Cameron, Henrik Stryhn, Greg Keefe, J Trenton McClure

Abstract

Objective

The primary objective was to determine the youngest age group where bovine leukemia virus (BLV)-infected dairy animals were identified. The secondary objective was to investigate associations between age-specific management practices and BLV infection status of different age groups of dairy calves and heifers.

Procedure

For enrolled herds, BLV status was determined using blood samples from pre-weaned calves, weaned calves, and breeding-age heifers; and bulk tank milk from the adult herd. A questionnaire investigating age-specific management factors was administered for each herd. Ordinal logistic regression was performed to identify management factors associated with the youngest age range in which BLV was identified.

Results

Fifty-three dairy herds from the 4 provinces in Atlantic Canada were enrolled. Bovine leukemia virus was most commonly earliest identified in pre-weaned heifers (18 herds, 32.1%) and the adult herd (18 herds, 32.1%). Ordinal logistic regression revealed that BLV was first identified in older age groups more often than in younger age groups when herds regrouped weaned heifers at least once, when fly control was used for breeding-age heifers, when herds practiced foot trimming on breeding-age heifers, and when bred heifers were brought in.

Conclusion

Producers can use results to identify the youngest age group(s) in which BLV is identified and to tailor management strategies to prevent new infections.

Résumé

Tendances temporelles de l'infection par le virus de la leucémie bovine dans les troupeaux laitiers des provinces atlantiques canadiennes

Objectif

L'objectif principal était de déterminer le groupe d'âge le plus jeune dans lequel les animaux laitiers infectés par le virus de la leucémie bovine (BLV) ont été identifiés. L'objectif secondaire était d'étudier les associations entre les pratiques de gestion spécifiques à l'âge et le statut d'infection par le BLV de différents groupes d'âge de veaux et de génisses laitiers.

Department of Health Management, Atlantic Veterinary College, University of Prince Edward Island, 550 University Avenue, Charlottetown, Prince Edward Island C1A 4P3.

Address all correspondence to Dr. Emily E. John; email: ejohn@upei.ca

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Procédure

Pour les troupeaux inscrits, le statut BLV a été déterminé à l'aide d'échantillons de sang provenant de veaux pré-sevrés, de veaux sevrés et de génisses en âge de se reproduire; et de lait de réservoir en vrac du troupeau adulte. Un questionnaire portant sur les facteurs de gestion spécifiques à l'âge a été administré pour chaque troupeau. Une régression logistique ordinale a été réalisée pour identifier les facteurs de gestion associés à la tranche d'âge la plus jeune dans laquelle le BLV a été identifié.

Résultats

Cinquante-trois troupeaux laitiers des quatre provinces atlantiques canadiennes ont été inscrits. Le virus de la leucémie bovine a été le plus souvent identifié le plus tôt chez les génisses pré-sevrées (18 troupeaux, 32,1 %) et dans le troupeau adulte (18 troupeaux, 32,1 %). La régression logistique ordinale a révélé que le BLV a été identifié pour la première fois plus souvent dans les groupes d'âge plus âgés que dans les groupes d'âge plus jeunes lorsque les troupeaux regroupaient au moins une fois les génisses sevrées, lorsque le contrôle des mouches était utilisé pour les génisses en âge de se reproduire, lorsque les troupeaux pratiquaient le parage des pattes des génisses en âge de se reproduire., et quand les taures saillies étaient intégrées au troupeau.

Conclusion

Les producteurs peuvent utiliser les résultats pour identifier le(s) groupe(s) d'âge le plus jeune dans lequel le BLV est identifié et pour adapter les stratégies de gestion afin de prévenir de nouvelles infections.

(Traduit par Dr Serge Messier)

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Introduction

Enzootic bovine leukosis is a disease of cattle caused by persistent infection with bovine leukemia virus (BLV), a delta-retrovirus (1). Although all cattle can be infected, it is primarily of concern for dairy herds in Canada, compared to beef herds. The virus does not cause overt clinical signs in most BLV-infected cattle, but ~30% of infected cows will develop persistent lymphocytosis, and up to 5% of infected cows will develop lymphoid tumors in a number of organ systems (2). There has historically been relatively little emphasis placed on control of enzootic bovine leukosis in North America due to its limited clinical expression and presumed minimal economic effect. However, recent investigations of the impact of BLV have identified a negative effect on cow health and susceptibility to disease, as well as overall farm economic impact, resulting in increased interest among dairy producers to control BLV on their farms (3–7).

Bovine leukemia virus is mainly transmitted to naïve cows *via* blood transfer from infected cows (1,8). For adult cattle, this has resulted in control measures focused on minimizing blood transfer between cows; *e.g.,* single use of hypodermic needles and rectal sleeves, cleaning of communal equipment such as hoof trimming and dehorning implements, and segregation of BLV-infected and -negative cows in particularly committed herds (1). However, there is also evidence that cows can become infected with BLV as calves or young heifers, either through blood contamination or possibly through ingestion of colostrum or milk from BLV-infected cows (9–11). Less commonly, BLV can be transmitted *in utero* or at parturition (10–12).

Despite increased awareness of BLV infection and its adverse effects, herd-level prevalence of BLV infection is high in Atlantic Canada, with ~90% of dairy herds having at least one BLV-seropositive cow (13). Communication with local animal owners has suggested that disease-control measures for older heifers and adult cattle have been implemented, but specific practices for calves and young heifers have not been prioritized. The primary objective of this study was to determine the youngest age group in which BLV infection was identified in dairy herds in Atlantic Canada. The secondary objective was to investigate associations between age-specific management practices and BLV infection status of different age groups on dairy farms in Atlantic Canada.

Materials and methods

Sample collection

Inclusion criteria for herd recruitment included participation in the concurrent regional BLV surveillance program and completion of a BLV-specific risk assessment and management program workbook with each herd's regular veterinarian (14). The goal was to recruit 60 herds in total across all 4 Atlantic Canada provinces (New Brunswick, NB; Newfoundland and Labrador, NL; Nova Scotia, NS; Prince Edward Island, PE), in proportions approximating the total number of dairy herds present in each province. This would result in recruiting 20 herds in NB, 2 herds in NL, 21 herds in NS, and 17 herds in PE. Information regarding each herd's bulk tank status and estimated within-herd prevalence for the adult milking herd was obtained from data collected for the ongoing regional BLV surveillance program, for the year in which the samples were collected.

For each herd, 6 blood samples were collected from individual animals in each of 3 age groups: pre-weaned heifer calves (generally younger than 2 mo of age), weaned heifers that were not old enough for breeding (generally between 2 and 14 mo of age), and breeding-age heifers (generally older than 14 mo of age, until calving). Animals were selected randomly from each age group. For smaller farms where the number of animals in a certain group was less than 6, all animals in that age group were sampled. Blood was collected by the primary author from either the jugular vein or coccygeal vein/artery into plain, red-top vacutainer tubes, depending on calf or heifer size. Some herds were sampled by the regular veterinarian, who then shipped the blood samples to the Maritime Quality Milk laboratory at the Atlantic Veterinary College, University of Prince Edward Island;

samples were shipped chilled on ice within 48 h of collection. All blood samples for a single farm were collected on the same day, and all samples were allowed to clot at room temperature before either being shipped chilled on ice and/or refrigerated at 4°C until sample processing. Samples were processed on a weekly basis during the sample collection phase.

Sample processing

Blood samples were brought to room temperature and then centrifuged at 2500 × *g* for 15 min. Serum was collected from each sample and used for further analyses. For pre-weaned calves, samples were tested for the presence of BLV genetic material using qPCR for the BLV *pol* gene (Bovine leukemia virus pol gene qPCR, PCRmax; Stone, Staffordshire, United Kingdom), following the manufacturer's instructions, after RNA extraction (QIAmp Viral RNA Mini Kit; Qiagen Canada, Montreal, Quebec). The test results were reported as PCR cycle threshold values (Ct), where a Ct of $>$ 38 indicated a negative result, a Ct of $>$ 0 and $\leq$ 38 indicated a positive result, and a Ct of $\leq$ 0 indicated a failed reaction or lack of DNA template. Weaned heifer and breeding-age heifer samples were tested for the presence of anti-BLV antibodies using a commercial indirect ELISA kit (SVANOVIR BLV gp51-Ab; Svanova, Uppsala, Sweden), following the manufacturer's instructions. The test results were reported as percent positivity (PP) values, PP = ($OD_{corrected}$ sample/$OD_{corrected}$ positive control) × 100, where OD is optical density. A PP of $\geq$ 15 indicated a positive result and a PP of $<$ 15 indicated a negative result. However, after using this kit on the first set of weaned heifer samples and obtaining a higher proportion of BLV-seropositive results compared to the pre-weaned and breeding-age heifers, we tested weaned heifers instead using the qPCR assay used for the pre-weaned calves. This was due to likely persistence of BLV from maternal antibodies in weaned heifers younger than 6 mo that were included in this age group. The same commercial ELISA kit was used on bulk tank milk samples to determine if the adult milking herd was BLV-infected. The PP cutoff for bulk tank milk was $<$ 5 to classify a herd as BLV-negative and $\geq$ 5 to classify a herd as BLV-positive. Pre-weaned calves and weaned heifers were considered BLV-positive if they had a Ct value of $>$ 0 and $\leq$ 38 on qPCR from a serum sample, breeding-age heifers were considered BLV-positive if they had a PP of $\geq$ 15 on ELISA from a serum sample, and the milking herd was considered BLV-positive if it had a PP of $\geq$ 5 on ELISA from a bulk tank milk sample.

Questionnaire administration

A questionnaire with questions about age-specific management practices was administered either at the time of sample collection or *via* telephone after sample collection, depending on producer availability. The questionnaire is included in Appendix 1 (available online from: www.canadianveterinarians.net). Data were entered into EpiInfo v7.2.2.6 software (Centers for Disease Control and Prevention, Atlanta, Georgia, USA) and then exported to either Excel 2013 (Microsoft Corporation, Redmond, Washington, USA) or Stata 16.1 (Statacorp, College Station, Texas, USA) software for analysis.

Statistical analysis

All analysis was performed using Stata 16.1 software. Each age group on a farm was considered BLV-positive if at least 1 calf or heifer in the age group was PCR-positive or ELISA-positive, depending on the test used for each age group. The adult herd was considered BLV-positive if at least 1 bulk tank milk sample was BLV-positive on ELISA testing. The number of calves or heifers per age group testing positive was divided by the total number of calves or heifers sampled for each age group, to determine the BLV prevalence of the animals sampled in each age group for each farm.

The outcome variable used for statistical analysis was the youngest age group in which BLV infection was identified: pre-weaned calves, weaned heifers, breeding-age heifers, or adult cows. The 4 values that the outcome variable could take were 1 = BLV infection identified in pre-weaned calves, 2 = BLV identified in weaned heifers, 3 = BLV identified in breeding-age heifers, and 4 = BLV identified in adult cows as the youngest age group. For outcome variable 1, herds that had BLV-positive pre-weaned calves did not necessarily have BLV infection identified in the weaned heifers or breeding-age heifers; similarly, for outcome variable 2, herds that had BLV-positive weaned heifers did not necessarily have BLV infection identified in the breeding-age heifers. However, all the pre-weaned calves in these herds were BLV-negative. Because the outcome variable was ordinal categorical and had 4 distinct values (corresponding with the 4 age groups stated above), ordinal logistic regression was used, rather than standard logistic regression (15). With the 4 age group categories, ordinal logistic regression uses 3 thresholds that correspond to dichotomizing the ordinal outcome at different groupings of the age group categories. These thresholds in this model correspond to the odds of BLV first being identified in the 3 older age groups (weaned heifers, breeding-age heifers, and adult cows) *versus* in pre-weaned calves, BLV first being identified in breeding-age heifers or adult cows *versus* in pre-weaned calves or weaned heifers, and BLV first being identified in adult cows *versus* in all calf and heifer age groups. The probabilities and odds generated from the model are for the outcome (youngest age group in which BLV infection was identified) being in a higher age group category *versus* being in a lower age group category. Both the "ologit" and the "gologit2" commands were used in Stata to explore whether the predictors met the proportional odds assumption for ordinal logistic regression. Predictors that meet the proportional odds assumption have the same OR for all investigated thresholds, corresponding to the predictor having the same effect on the model across all the investigated thresholds. Predictors that do not meet the proportional odds assumption have different ORs at different thresholds.

The questionnaire contained a total of 75 variables relating to management factors in the 4 different age groups. Univariable analyses were performed for all independent variables, and those with a *P*-value of $<$ 0.2 were retained for further analysis (15). The Wald test was used to assess overall *P*-values for variables with more than 2 categories and to assess whether a variable met the proportional odds assumption. Table S1 (available online from: www.canadianveterinarians.net) contains results of all the univariable analyses; 16 variables were excluded from analysis

Table 1. Summary of herds enrolled in each province compared to the total number of herds active in the Atlantic Canada region in 2016 to 2017.

Province[a]	Herds enrolled	Total herds	% of total enrolled	Goal for her enrollment	% of goal
NB	19[b]	195	9.7	20	95
NL	2	27	7.4	2	100
NS	25[c]	217	11.5	21	119
PE	10	166	6.0	17	59
Total	56	605	9.3	60	93

[a] NB — New Brunswick; NL — Newfoundland and Labrador; NS — Nova Scotia; PE — Prince Edward Island.
[b] Two herds were removed from data analysis: 1 herd had no bovine leukemia virus (BLV)-positive animals identified and 1 herd was removed due to questionnaire nonresponse.
[c] One herd was removed from data analysis due to questionnaire nonresponse.

due to > 90% of herds having the same option selected from the list of possible answers. Due to the large number of potential predictors and the small size of the dataset, forward selection was used to build the final multivariable ordinal logistic regression model. For predictors that did not meet the proportional odds assumption, model-building used the "gologit2" software package to allow for nonproportional odds for that predictor(s) while maintaining proportional odds assumptions for the other predictors. A *P*-value of < 0.05 was considered significant for variable inclusion in the final model.

Results

A total of 56 herds were recruited across all 4 Atlantic Canadian provinces; detailed information is included in Table 1. The anticipated numbers of herds were recruited from NL and NS, but fewer herds than anticipated were recruited from NB and PE. Additional herds from NS were interested in participating, so those herds were included to increase the total number of participating herds. All but 1 herd were BLV-infected based on bulk tank milk samples. The single BLV-negative herd was excluded from data analysis (all 4 age groups were BLV-negative). Two additional herds were excluded from statistical data analysis due to nonresponse when contacted to complete the questionnaire, leaving a total of 53 herds included in the data analysis. The number of calves and heifers sampled in each age group per farm ranged from 2 to 7; when < 6 calves or heifers were sampled per age group on a farm, the number of sampled animals comprised the entire age group present at the time of sampling.

Table 2 shows the youngest age group where BLV-positive animals were identified. Approximately 1/3 of herds had pre-weaned calves as the youngest BLV-infected age group, 11% of herds had weaned heifers as the youngest BLV-infected age group, 23% of herds had breeding-age heifers as the youngest BLV-infected age group, and 32% of herds had no BLV-positive calves or heifers and but had BLV-positive adults. Not all herds where BLV-positive pre-weaned heifers were identified also had BLV-positive weaned heifers or breeding-age heifers identified; similarly, not all herds with BLV-positive weaned heifers also had BLV-positive breeding-age heifers identified. All 53 herds contained BLV-positive adult cows based on bulk tank milk ELISA results.

For the 18 herds where pre-weaned heifers were the youngest identified BLV-positive age group, the median prevalence within the sampled calves was 20.0% (range: 14.3 to 83.3%). Thirteen herds had 1 BLV-positive pre-weaned calf, 3 herds had 2 BLV-positive pre-weaned calves, and 1 herd each had 3 and 5 BLV-positive pre-weaned calves. For the 13 herds where weaned heifers were the youngest identified BLV-positive age group, the median prevalence within the sampled heifers was 16.7% (range: 14.3 to 50.0%). Ten herds had 1 BLV-positive weaned heifer, 2 herds had 2 BLV-positive weaned heifers, and 1 herd had 3 BLV-positive weaned heifers. For the 29 herds where breeding-age heifers were the youngest identified BLV-positive age group, the median prevalence within the sampled heifers was 25.0% (range: 16.7 to 50.0%). Fifteen herds had 1 BLV-positive breeding-age heifer, 9 herds had 2 BLV-positive breeding-age heifers, and 5 herds had 3 BLV-positive breeding-age heifers.

Table 2. Summary of herd infection status for each of the 4 age groups tested, showing the youngest age group in which bovine leukemia virus (BLV)-positive animals were identified.

Age at first infection	Number of herds (%)
None[a]	1 (1.8)
Adults	18 (32.1)
Breeding-age heifers[b]	13 (23.2)
Weaned heifers	6 (10.7)
Pre-weaned heifers	18 (32.1)

[a] This herd was removed from analysis because no BLV-positive animals were identified.
[b] Two herds in this group were removed from analysis due to questionnaire nonresponse.

Table S1 (available online from: www.canadianveterinarians.net) displays the univariable analyses for all 59 variables from the questionnaire that were analyzed, and Table 3 displays the variables retained for model-building after univariable analyses. Table 4 displays the variables included in the final multivariable model. After forward selection model-building, the final model incorporated 4 variables: the number of times weaned heifers were regrouped, use of fly control in weaned heifers, the use of foot-trimming implements in breeding-age heifers; and whether the farm purchased bred heifers.

Herds that regrouped heifers after weaning had higher odds of older age groups being the youngest in which BLV was identified, compared to younger age groups. The highest odds were identified in herds where heifers were regrouped twice before they were old enough to enter the breeding-age heifer group (OR: 30.06), followed by herds that regrouped heifers 3 or more times (OR: 6.05), then by herds that regrouped heifers

Table 3. Variables retained for forward selection model-building using the youngest age group where bovine leukemia virus (BLV) was identified as the outcome variable in ordinal logistic regression. For variables that met the proportional-odds assumption, the *P*-value displayed is the *P*-value for each age group comparison.

Variable	Levels	OR	95% CI	*P*-value
Treatment of milk fed to calves	Pasteurized or acidified	N/A		
	Not treated	3.299	0.845, 12.880	0.086
Type of calf housing, pre-weaned calves	Individual pens or hutches	N/A		
	Group pens or hutches	2.051	0.705, 5.965	0.187
Fly control in pre-weaned calves	None	N/A		
	Environmental or topical	3.327	0.935, 11.841	0.063
	Environmental and topical	5.287	0.654, 42.711	0.118
	Overall *P*-value			0.133
Number of times weaned heifers are regrouped	Not regrouped	N/A		
	Regrouped once	2.791	0.502, 15.529	0.241
	Regrouped twice	9.644	1.791, 51.931	0.008
	Regrouped three times	2.146	0.384, 12.001	0.384
	Overall *P*-value			0.033
Fly control in weaned heifers	None	N/A		
	Environmental or topical	3.431	1.207, 9.760	0.021
	Environmental and topical	5.286	0.386, 72.452	0.213
	Overall *P*-value			0.051
Age when heifers enter the breeding group		0.809	0.590, 1.110	0.190
Breeding-age heifer housing related to adults	> 200 m away	N/A		
	< 200 m away	1.420	0.372, 5.416	0.608
	Same building	3.181	0.836, 12.105	0.090
	Overall *P*-value			0.177
Foot-trimming in breeding-age heifers[a]	No	N/A		
	Yes	2.753	0.808, 9.381	0.105
	Overall *P*-value			0.011
Fly control in breeding-age heifers	None	N/A		
	Environmental or topical	4.645	1.1493, 14.454	0.008
	Environmental and topical	4.439	0.660, 29.877	0.125
	Overall *P*-value			0.019
Farm buys bred heifers	No	N/A		
	Yes	3.169	0.810, 12.403	0.098
Farm buys mature cows	No	N/A		
	Yes	2.142	0.680, 6.749	0.193
Any other method of contact with other herds	No	N/A		
	Yes	0.315	0.080, 1.239	0.098

N/A — Not applicable.
[a] For the variable that did not meet the proportional-odds assumption, the estimate and *P*-value displayed are the one for the relevant age group comparison, along with the overall *P*-value.

once (OR: 5.35). Herds using some type of fly control for their breeding-age heifers were more likely to have older age groups as the youngest in which BLV was identified, compared to younger age groups, with the highest odds for herds using either environmental or topical fly control methods (OR: 13.06); however, the OR for herds using both environmental and topical fly control methods was only slightly lower (OR: 12.98). Herds where foot-trimming instruments were used on breeding-age heifers had 2.70× higher odds of adult cows being the youngest group in which BLV was identified, compared to younger age groups. The final predictor associated with older age groups being the youngest in which BLV was identified was the farm purchasing bred heifers; this resulted in odds 12.75× higher that older age groups were the youngest in which BLV was identified, compared to younger age groups.

Discussion

This study showed that, on dairy farms in Atlantic Canada, the most common age groups in which BLV infection is present are pre-weaned heifer calves and the adult milking herd. Also, BLV was commonly identified in breeding-age heifers, with the weaned heifers being the least likely in this study to be the youngest age group in which BLV was identified. The fact that BLV was identified in all age groups of calves, heifers, and cows suggests that there is a range of management practices on dairy farms in this region that may influence BLV transmission. However, on most farms where BLV-positive calves or heifers were identified, the apparent prevalence (*i.e.,* the number of BLV-positive animals divided by the number of sampled animals) was low.

Table 4. Final multivariable ordinal logistic regression model investigating management factors associated with the youngest age group of calves and heifers in which bovine leukemia virus (BLV)-positive animals were identified.

Variable	Levels	OR	95% CI	*P*-value
Number of times weaned heifers are regrouped	Not regrouped			
	Regrouped once	5.348	0.724, 38.542	0.096
	Regrouped twice	30.058	3.686, 245.136	0.001
	Regrouped three times	6.049	0.785, 46.591	0.084
	Overall *P*-value			0.013
Fly control in breeding-age heifers	None			
	Environmental or topical	13.057	2.666, 63.937	0.002
	Environmental and topical	12.978	1.544, 109.069	0.018
	Overall *P*-value			0.003
Foot-trimming in breeding-age heifers[a]				
All older age groups *versus* pre-weaned calves	No			
	Yes	0.338	0.071, 1.607	0.173
Adult cows + breeding-age heifers *versus* younger age groups	No			
	Yes	0.180	0.037, 0.861	0.032
Adult cows *versus* all calf and heifer age groups	No			
	Yes	2.704	0.519, 14.077	0.237
	Overall *P*-value			0.035
Farm buys bred heifers	No			
	Yes	12.745	1.908, 85.132	0.009

[a] For the variable that did not meet the proportional-odds assumption, all estimates and *P*-values for the 3 difference comparisons are shown, as well as the overall *P*-value.

The results of this study revealed 4 management practices that were statistically significantly associated with identifying BLV-positive animals in different age groups of calves and heifers on dairy farms in Atlantic Canada: if weaned heifers were regrouped before entering the breeding-age heifer group, if fly control was used for breeding-age heifers, if foot-trimming implements were used on breeding-age heifers, and if the farm purchased bred heifers.

For farms where weaned heifers were regrouped after weaning — where heifers did not remain solely with the heifers they were grouped with after weaning, but either were intermingled with other heifers or had their original group merged with another small group of heifers — the odds of older age groups being the youngest in which BLV was identified was higher than in herds where weaned heifers stayed in their original group until entering the breeding-age heifer group. This finding is contrary to what was initially expected. Moving animals and introducing them to new groups is stressful and can influence immune function, theoretically making heifers that experience more movement or regroupings more susceptible to infection, including BLV. Additionally, having more animals in a pen or more frequent mixing of animals will result in more direct contact between animals, and has the potential to allow more frequent blood transfer from a BLV-infected heifer to a BLV-negative one. However, the results of this study suggest that farms where heifers are moved more often have higher odds of adult cows being the youngest group in which BLV was identified, compared to any of the calf and heifer age groups. One possible explanation could be that farms regrouping heifers more often are doing so to optimize their feed intake and average daily gain, and so in general are implementing a larger proportion of more intensive heifer management practices. Another possibility is that these farms have implemented infection control practices in pre-weaned calves (*e.g.,* pasteurizing/freezing colostrum, feeding milk replacer or milk from BLV-negative cows), and so there is no risk to regrouping heifers, as none (or very few) are infected with BLV.

Fly-control practices in breeding-age heifers were also significantly associated with the youngest age group in which BLV was identified. Herds in which environmental or topical fly control was used had 13.06× higher odds of older age groups being the youngest in which BLV was identified, and herds using both types of fly control had 12.98× higher odds compared to herds using no fly control for breeding-age heifers. Univariable analysis also suggested that use of one or both methods of fly control in pre-weaned calves and weaned heifers resulted in higher odds of older age groups being the youngest in which BLV was identified, with the use of both methods of fly control having the highest odds. These findings suggest that the use of fly control may help to prevent spread of BLV *via* biting flies in calf and heifer groups and may result in BLV first becoming prevalent in adult cows. It is also interesting to note the link between fly control practices and the lower odds of BLV being first identified in younger age groups, as multiple studies have investigated the role of flies in BLV transmission in adult cows (16–21).

The practice of foot care in breeding-age heifers resulted in higher odds of adult cows being the youngest group in which BLV was identified, compared to any of the younger age groups. This could be explained by the fact that foot-trimming implements can become contaminated with blood, especially if foot infections such as strawberry foot rot are present. If not properly disinfected between animals, the implements could serve as fomites to transmit BLV. It is unlikely that farms would use a separate set of implements for heifers and adult cows, and so BLV-positive adult cows could be the infection source for naïve breeding-age heifers. Although the use of foot-trimming implements in breeding-age heifers resulted in higher odds of adult cows being the youngest group in which BLV was identified, it

did not result in higher odds of breeding-age heifers being the youngest group. This could be due to timing of foot trimming in breeding-age heifers; a possibility is that producers may be performing foot trimming within a few weeks or months of the heifer's anticipated entry into the adult herd at the time of calving, and so any infections that occur in these heifers may not be apparent until they are in the adult milking herd.

The final management factor that was associated with the youngest age group where BLV was identified was whether the farm purchased bred heifers. Farms purchasing bred heifers had 8.65× higher odds of older age groups being the youngest in which BLV was identified, compared to herds that did not purchase bred heifers. An explanation for this finding is that these farms could be practicing good disease-control measures in young age groups but purchasing bred heifers from farms where BLV infection occurs in the younger age groups, thus introducing BLV into their herds *via* older heifers. In this case, it would appear as though good infection control practices are in place until heifers enter the adult milking herd, when actually the BLV pressure on the farm is very low until BLV-infected heifers are purchased from an outside source and determined to be BLV-seropositive as adult cows.

None of the other management factors investigated for the 3 calf and heifer age groups was statistically significantly associated with the youngest age group in which BLV was identified. This included management factors previously associated with BLV transmission in adult cows; for example, the reuse of hypodermic needles and syringes (1). A possible explanation is the small dataset in this project prevented some important management factors from showing statistical significance, and a larger sample size of farms may allow for identification of further management factors associated with the youngest age group in which BLV is identified.

There were several limitations of this study. We did not manage to recruit the anticipated number of herds, and 3 herds that were sampled were excluded from analysis either due to being BLV-negative or due to an inability to complete the questionnaire. This limited the number of data points available for analysis and may have affected the overall results. In terms of the number of herds recruited compared to the total number of dairy herds present in Atlantic Canada, $<$ 10% of herds were enrolled in this study. This could have resulted in a sample size too small to detect significant results for other management factors included in the survey. Due to the small number of herds and the large number of questions asked on the questionnaire, there was also the risk of overfitting the available data points if too many predictors remained statistically significant in the final model.

The number of individual animals sampled on each farm was also a limitation, for several reasons. Regardless of herd size, the same maximum number of calves or heifers was sampled on each farm. In some small herds, this resulted in 100% of an age group being included in the study; in the largest herds, this resulted in 2% of an age group being included in the study. It is possible that some of the age groups sampled that were classified as BLV-negative may have been BLV-positive if a larger proportion of animals had been sampled. For example, there were several herds where BLV-positive pre-weaned calves were identified but all the sampled weaned heifers and breeding-age heifers were BLV-negative. These herds were still classified as having pre-weaned calves as the youngest group in which BLV was identified, as, presumably, if a larger subset of each age group was sampled, BLV-positive weaned heifers or breeding-age heifers would have been identified. This could have effects on the overall validity of the model in terms of using management factors to predict the odds of an age group being BLV-positive. In addition, the adult herd was only sampled using the results of bulk tank milk samples collected as part of a concurrent surveillance program, and no individual adult blood samples were collected. However, studies have shown that ELISA testing of bulk tank milk is both sensitive and specific for identifying BLV-infected herds, as well as for estimating the within-herd prevalence of BLV infection in lactating animals (13,22–23).

The literature is sparse in terms of prevalence studies of BLV in pre-lactating dairy animals, but 1 study (24) described a prevalence of 11.5% in naturally infected calves $<$ 12 mo old in a dairy herd with very high adult within-herd prevalence, and another study (12) reported 10.8% of calves born to BLV-infected mothers were BLV-positive at birth. Both these studies used nested PCR to test calves, whereas our study used qPCR on calves of the same ages. Although this estimate of prevalence in calves may be too high for dairy herds in Atlantic Canada, it illustrates that, in herds where a very small percentage of an age group was sampled, age group may have been falsely classified as BLV-negative, especially if the prevalence of BLV was low in that herd.

The calves and heifers sampled on each farm were intended to be randomly sampled, to prevent veterinarian or producer bias and the preferential inclusion of "BLV-suspect" animals. However, practicality on farms sometimes necessitated convenience sampling of whichever calves or heifers were available; *e.g.,* some farms had breeding-age heifers at a separate location, or loose in a pasture, or pastured with a bull. This reduced the proportion of herds where animals were sampled randomly and could have introduced sampling bias. Also, as noted above, the sampling fraction was 100% in some age groups due to the small size of the herd, and so sampling fraction was not consistent across all herds.

The different age groups of calves and heifers were also assessed with different tests for BLV. Due to the potential presence of maternal anti-BLV antibodies, the initial plan was to test the pre-weaned calves with RT-qPCR for viral RNA and the older calves and heifers with indirect ELISA for anti-gp51 antibodies. However, as the age range of weaned heifers was 2 to 14 mo, some of the younger heifers could have still retained maternal antibodies in their serum due to colostrum ingestion. After inconsistent results were obtained with the first 2 herds tested, where a higher prevalence was identified in weaned heifers compared to pre-weaned calves or breeding-age heifers, all of the pre-weaned and weaned calves/heifers were tested using qPCR, and only the breeding-age heifers were tested with ELISA. As these tests were investigating different measures of infection; *i.e.,* the qPCR was directly looking for viral genetic material after reverse transcription and the ELISA was looking

for the host response to BLV infection through antibodies, it is difficult to directly compare prevalence in the different age groups. There is currently no evidence that BLV-infected animals can clear the infection, and so the BLV-seropositive heifers presumably would also be BLV PCR-positive if tested with qPCR. However, seroconversion after infection can take up to 57 d (25), and so there may have been false negative results in the breeding-age heifer group. Additional false negatives could result from diagnostic test performance; however, the ELISA test used has a reported sensitivity of 99% and specificity of 99.4%, and the qPCR test has a reported sensitivity to detect $<$ 100 copies of the target DNA, making the presence of false negatives unlikely.

In conclusion, on dairy farms in Atlantic Canada, BLV was identified in all age groups of calves and heifers sampled, with pre-weaned calves and the adult milking herd most often the youngest age groups in which BLV was identified; however, there was no clear pattern of infection seen in all participating herds. In BLV-positive herds, management factors involving all age groups of calves and heifers were associated with the youngest age group in which BLV was identified. These results can be used by producers to identify the youngest age group where BLV is most likely to be identified, and tailor disease-control methods accordingly. CVJ

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Article

Anticoagulant rodenticide toxicity in dogs: A retrospective study of 349 confirmed cases in Saskatchewan

Mathieu Victor Paulin, Samantha Bray, Tanarut Laudhittirut, Jeneva Paulin, Barry Blakley*, Elisabeth Snead

Abstract

Objective

To evaluate the signalment and clinical, laboratory, treatment, and outcome features of dogs diagnosed with anticoagulant rodenticide (AR) intoxication in Saskatchewan.

Animals

We studied 349 dogs.

Procedure

Medical records from the Veterinary Medical Centre (Saskatoon, Saskatchewan) between 1999 and 2022 were reviewed. Cases were included if they met at least 1 of the following criteria: owner witnessed the dog ingesting an AR; AR was seen in the vomitus when emesis was induced; the dog had clinical signs of coagulopathy, with elevation of PT ± aPTT that normalized after vitamin K1 therapy, in the presence of appropriate clinical and paraclinical data and the absence of other causes of hypocoagulable state determined by the primary clinician.

Results

Fifty-three percent of cases were seen between July and October. Most dogs (61%) came from an urban setting. Ninety-two percent of dogs ingested a 2nd-generation AR and the most frequent toxin was bromadiolone. Clinical signs were reported in 30% of AR intoxications and included lethargy (86%), dyspnea (55%), and evidence of external hemorrhage (44%). The most common site of hemorrhage was the pleural space, accounting for 43% of hemorrhage sites. Consumptive thrombocytopenia was reported in 24% of dogs with evidence of AR-induced hemorrhage, with moderate (platelet count < 60 K/μL) and marked (< 30 K/μL) thrombocytopenia in 7/12 and 2/12 dogs, respectively. Blood products were administered to 84% of dogs with AR-induced hemorrhage; the most common product administered was fresh frozen plasma (56% of cases). Among dogs with AR-induced hemorrhage, those that received blood products were more likely to survive to discharge (81%) compared to those that did not (19%) ($P = 0.017$). Eighty-six percent of dogs with AR-induced hemorrhage survived to discharge.

Conclusion and clinical relevance

The pleural space was the most common site of hemorrhage. Moderate thrombocytopenia was a common finding. Eighty-six percent of dogs with AR-induced hemorrhage survived to discharge.

Résumé

Toxicité des rodenticides anticoagulants chez les chiens : étude rétrospective de 349 cas confirmés en Saskatchewan

Objectif

Évaluer le signalement et les caractéristiques cliniques, de laboratoire, de traitement et de résultats des chiens diagnostiqués avec une intoxication par un rodenticide anticoagulant (AR) en Saskatchewan.

Animaux

Nous avons étudié 349 chiens.

Department of Small Animal Clinical Sciences (MV Paulin, Laudhittirut, Snead) and Department of Veterinary Biomedical Sciences (Blakley), Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan S7N 5B4; Alpine Animal Hospital, 10 H Keenleyside Street, Winnipeg, Manitoba R2L 2B9 (Bray); VCA Central Animal Hospital, 106 103 Street East Saskatoon, Saskatchewan S7N 1Y7 (J Paulin).

Address all correspondence to Mathieu Victor Paulin; email: mvp784@usask.ca

* Deceased. The authors dedicate this article to the memory of their co-author, Dr. Barry Blakley.

Procédure

Les dossiers médicaux du *Veterinary Medical Centre* (Saskatoon, Saskatchewan) entre 1999 et 2022 ont été examinés. Les cas ont été inclus s'ils répondaient à au moins 1 des critères suivants : le propriétaire a vu le chien ingérer un AR; de l'AR a été observée dans les vomissures lorsque des vomissements ont été provoqués; le chien présentait des signes cliniques de coagulopathie, avec une élévation du PT ± aPTT qui s'est normalisée après un traitement par la vitamine K1, en présence de données cliniques et paracliniques appropriées et en l'absence d'autres causes d'état hypocoagulable déterminées par le clinicien initial.

Résultats

Cinquante-trois pour cent des cas ont été observés entre juillet et octobre. La plupart des chiens (61 %) venaient d'un milieu urbain. Quatre-vingt-douze pour cent des chiens ont ingéré un AR de 2e génération et la toxine la plus fréquente était la bromadiolone. Des signes cliniques ont été rapportés dans 30 % des intoxications par AR et incluaient de la léthargie (86 %), de la dyspnée (55 %) et des signes d'hémorragie externe (44 %). Le site d'hémorragie le plus fréquent était l'espace pleural, représentant 43 % des sites d'hémorragie. Une thrombocytopénie de consommation a été rapportée chez 24 % des chiens présentant des signes d'hémorragie induite par l'AR, avec une thrombocytopénie modérée (nombre de plaquettes < 60 K/μL) et marquée (< 30 K/μL) chez 7 chiens sur 12 et 2 chiens sur 12, respectivement. Des produits sanguins ont été administrés à 84 % des chiens présentant une hémorragie induite par l'AR; le produit le plus fréquemment administré était le plasma frais congelé (56 % des cas). Parmi les chiens présentant une hémorragie induite par l'AR, ceux qui ont reçu des produits sanguins étaient plus susceptibles de survivre jusqu'à leur congé (81 %) que ceux qui n'en ont pas reçu (19 %) (P = 0,017). Quatre-vingt-six pour cent des chiens présentant une hémorragie induite par l'AR ont survécu jusqu'à leur sortie.

Conclusion et pertinence clinique

L'espace pleural était le site d'hémorragie le plus fréquent. Une thrombocytopénie modérée était fréquente. Quatre-vingt-six pour cent des chiens présentant une hémorragie induite par l'AR ont survécu jusqu'à leur sortie.

(Traduit par Dr Serge Messier)

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Introduction

Anticoagulant rodenticides (ARs) are pest-control products that represent a common cause of intoxication in dogs (1–4). In recent years, legislation has been passed in North America to control and limit the use of 2nd-generation ARs (SGARs) yet, despite these actions, ARs remain one of the most commonly reported toxicoses in companion animals. Intoxication in dogs typically results from accidental ingestion of rodent baits and, in rare cases, from malicious poisoning (1,3,4). Secondary or relay intoxication of dogs due to ingestion of intoxicated rodents or non-target animals (such as birds and invertebrates) is also possible but uncommon (1,5).

Anticoagulant rodenticides induce coagulopathy and subsequent hemorrhage by impairing hepatic synthesis of certain coagulation factors. Anticoagulants are generally well-absorbed through the intestinal epithelium within a few hours (6). In addition, absorption through the respiratory tract and skin has also been reported; however, is less frequently relevant in dogs (6). In blood, warfarin is almost entirely protein-bound, primarily to albumin. After intestinal absorption, ARs travel to the liver *via* the portal vein system or chylomicrons, and are metabolized by the liver largely through the action of cytochrome P450 (6). Both 1st-generation ARs (FGARs) and SGARs accumulate rapidly in the liver until the microsomal binding sites are saturated (6). Anticoagulant rodenticides inhibit the hepatic enzyme vitamin K1 epoxide reductase (1). This enzyme is required for the recycling of vitamin K1 into its active form, which is a necessary cofactor for the γ-carboxylation of glutamic acid residues on clotting factors II, VII, IX, and X (7). Anticoagulant rodenticides therefore cause depletion of the active form of vitamin K1, leading to a progressive decrease in plasma concentrations of functional forms of vitamin K1-dependant clotting factors, and eventually leading to a hypocoagulable state and hemorrhage (8). Anticoagulant rodenticides then exit the liver *via* the hepatic vein (where they can be measured in circulation) and are eliminated through urine or bile. In the case of biliary elimination, some ARs are subject to enterohepatic recirculation and can remain in the liver tissues for weeks, even after successful treatment (1,5).

Anticoagulant rodenticides can be divided into 2 subcategories based on their potency and activity: FGARs and SGARs. Warfarin was the first AR developed and was widely used until rodents began to develop resistance. Warfarin is derived from dicoumarol, a naturally occurring chemical found in some plants, including sweet clover *(Melilotus* spp.*)* (9). Resistance to warfarin prompted the development of SGARs, sometimes referred to as superwarfarins (1,3,5). These superwarfarins (*i.e.,* brodifacoum, bromadiolone, difethialone) are more potent and longer-acting than FGARs. This is attributed to their greater affinity for vitamin K1 epoxide reductase, ability to disrupt the vitamin K1 epoxide cycle at more than one location, hepatic accumulation, and unusually long biological half-lives due to high lipid solubility and enterohepatic recirculation. For example, the plasma elimination half-lives of the SGARs bromadiolone and brodifacoum are 6 d, compared to warfarin's half-life of only 14 h (3,10). The toxic dose and median lethal dose depend on the AR product, but SGARs have median lethal doses significantly lower than those of FGARs and generally

require only a single feeding to result in the death of the target species (5).

Clinical signs of AR toxicosis usually develop 2 to 5 d after exposure, depending on the dose and type (FGAR *versus* SGAR) of AR product ingested (1,3,11,12). The impaired coagulation can be assessed by measurement of blood coagulation parameters including prothrombin time (PT) and activated partial thromboplastin time (aPTT). Among the 4 vitamin K1-dependant clotting factors (Factors II, V, VII, IX), Factor VII has the shortest half-life, and so an increase in PT is expected before an increase in aPTT (1,3). Therefore, PT testing is considered more sensitive than aPTT testing for early detection of AR intoxication (1,3). Advanced and confirmation testing are sometimes needed, especially in medicolegal cases, since results are generally not received in time to affect treatment decisions. Antemortem confirmatory tests include measurement of AR in stomach contents or serum/blood, whereas postmortem samples also include liver and kidneys for liquid chromatography (1).

Treatment of dogs with AR intoxication involves the administration of vitamin K1. The appropriate length of treatment required depends on the dose ingested (most often unknown) and, more importantly, the generation of the AR ingested. Treatment with vitamin K1 is normally given for 2 to 4 wk, depending on the specific AR (1,3). This highlights the importance of recognizing, if possible, which form of rodenticide was ingested.

To the authors' knowledge, only a few studies on dogs diagnosed with AR intoxication are available in the literature, and these reported comparatively small numbers of cases (8,13–15). The purpose of this study was to evaluate the signalment, epidemiology, clinical and clinicopathologic features, medical management, and outcomes of a large number of cases of AR intoxication (N = 349) presented to the Veterinary Medicine Centre (VMC), Western College of Veterinary Medicine (Saskatoon, Saskatchewan), over a 23-year period. Our aim is that the information presented in this report will aid clinicians in the diagnostic evaluation and treatment of suspected AR intoxications.

Materials and methods

Database

Medical records from the VMC from January 1999 to December 2022 were reviewed for confirmed cases of dogs with AR intoxication. Cases were included in the study if they met at least 1 of the following criteria: i) the owner witnessed the dog ingesting an AR; ii) a formed product matching common characteristics of AR products was present in the vomitus when emesis was induced; or iii) the dog had clinical signs of coagulopathy in which an elevated PT ± aPTT was documented, along with evidence of normalization of PT/aPTT measurements following vitamin K1 therapy, in the presence of appropriate clinical and paraclinical data and the absence of other causes of hypocoagulable state identified by the primary clinician.

Signalment and epidemiologic data were extracted from the medical record when the information was available. These included breed, age, sex, weight, urban *versus* rural setting, year and month when AR intoxication occurred, rodenticide active ingredient, and generation of AR. Sites of hemorrhage were recorded based on the clinical signs; physical examination findings at presentation (the hair coat was not routinely clipped); diagnostic imaging studies performed [thoracic and abdominal radiographs, abdominal- (AFAST) and thoracic- (TFAST) focused assessment with sonography for trauma (FASTVet, Spicewood, Texas, USA)]; any interventional procedures performed (*e.g.,* thoracocentesis, abdominocentesis); and necropsy findings, if intoxication was fatal or the owner requested euthanasia. The hospitalization time, if any, was also recorded.

The PT and aPTT testing were completed with 1 of 2 machines, depending on whether samples were processed within normal business hours (MLA Electra 750 photo-optical plasma coagulation timing instrument; Beckman Coulter, Mississauga, Ontario: RR 10 to 13 sec for PT and 9.6 to 45 sec for aPTT), or outside of normal business hours (patient-side Idexx Coag Dx Analyzer; Idexx, Markham, Ontario: RR 11 to 17 sec for PT and 72 to 102 sec for aPTT). For the complete blood (cell) count (CBC) data, the Cell Dyn 3500 (Abbott Laboratories, Mississauga, Ontario) automated hematology analyzer was used until the end of 2013, and the ADVIA 2120i (Siemens Healthineers, Oakville, Ontario) was used thereafter. As part of the CBC, blood samples from dogs classified as anemic were smeared and stained with new methylene blue dye, to calculate the percentage of reticulocytes present and to further characterize the anemia as regenerative or non-regenerative. In all cases, the automated platelet count was also confirmed as adequate by a clinical pathologist or clinical pathology technician based on manual evaluation of a blood smear.

Blood serum biochemistries were measured using a Hitachi 911 chemistry analyzer (Roche Diagnostics, Laval, Quebec) until mid-2011, and thereafter using a Cobas C311 (Roche Diagnostics). If full serum biochemical analysis was not undertaken, the total protein (TP) concentration was measured from plasma using a refractometer, as part of an emergency panel on admission, along with the dog's packed cell volume (PCV), blood glucose, and Azostix (Siemens Medical Solutions, Oakville, Ontario) blood urea nitrogen.

The treatment regimen for each animal was also reviewed, and data were extracted from the record, including induction of emesis, administration of activated charcoal or intravenous fluids, symptomatic treatments, blood product transfusions, and vitamin K1 supplementation. The dose, route, and duration of administration of vitamin K1 prescribed in hospital and prescribed at discharge were recorded. In addition, the times to recheck at the VMC, if any, for retesting of PT, aPTT, and other blood parameters were noted. The dog's outcome (alive, euthanized, deceased) was recorded, and necropsy reports, when available, were reviewed for any additional information to confirm AR intoxication.

Statistics

Qualitative descriptive data were presented as percentage, mean ± standard deviation, and range. For nonparametric contingency tables, the Fisher exact test was used. A P-value of < 0.05 was considered statistically significant. Statistical analyses were completed, and graphs were produced, using a commercial software package (GraphPad Prism 9 version 9.5.1; GraphPad Software, La Jolla, California, USA).

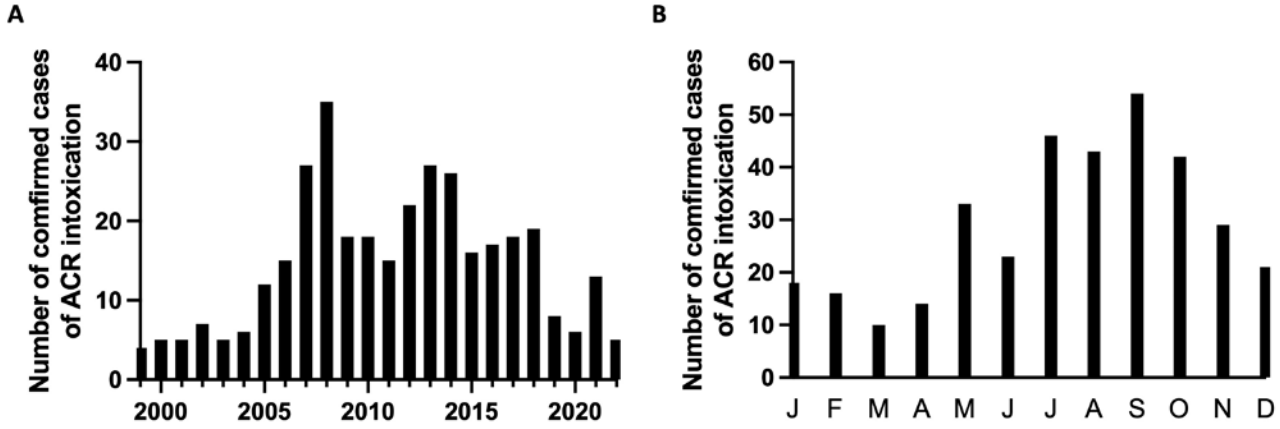

Figure 1. A – Distribution of yearly confirmed cases of anticoagulant rodenticide (ACR) intoxication in dogs between 1999 and 2022. B – Number of confirmed cases of ACR intoxication by month between 1999 and 2022.

Results

Study population epidemiology and rodenticide products

A total of 349 confirmed cases of dogs with AR intoxication were identified between January 1999 and December 2022, with a mean of 15 ± 9 cases/y (range: 4 to 35/y) (Figure 1 A). Most cases were seen between July and October, with 185/349 cases (53%) seen during this period (Figure 1 B). No sex predisposition was noticed: 161/349 dogs (46%) were males (69 intact and 92 castrated) and 188/349 (54%) were females (70 intact and 118 spayed). Age of dog at presentation was available for 342 dogs and median age was 42 mo (3.5 y) ± 42 mo (range: 1 mo to 17 y). Weight was available for 331 dogs and median weight was 16.6 ± 13 kg (range: 1.1 to 66.2 kg). There were 103 dogs (30%) in the small-breed category (< 10 kg), 126 dogs (36%) in the medium-breed dog category (10 to 25 kg), 86 dogs (25%) in the large-breed category (25.1 to 45 kg), and 11 dogs (3%) in the giant-breed category (> 45.1 kg). The most common breeds affected were Labrador retriever (66/349, 19%), German shepherd (29/349, 8%), and border collie (22/349, 6%), followed by golden retriever (5%), shih tzu (5%), beagle (4%), cocker spaniel (3%), Pomeranian (3%), and 60 other breeds with frequency < 3% for each.

Dog and owner lived in a rural setting (acreage or a farm) in 136/349 cases (39%) and in an urban setting in 213/349 cases (61%). In 240/349 cases (69%), the owner claimed to have witnessed their dog ingesting the rodenticide. The type of AR was recorded and provided by the owner in 157/349 cases (45%): 13 dogs (8%) ingested warfarin, an FGAR; the remaining 144 dogs ingested an SGAR, including bromadiolone (*n* = 51), brodifacoum (*n* = 40), difethialone (*n* = 20), diphacinone (*n* = 16), chlorophacinone (*n* = 15), and indandione (*n* = 2) (Figure 2).

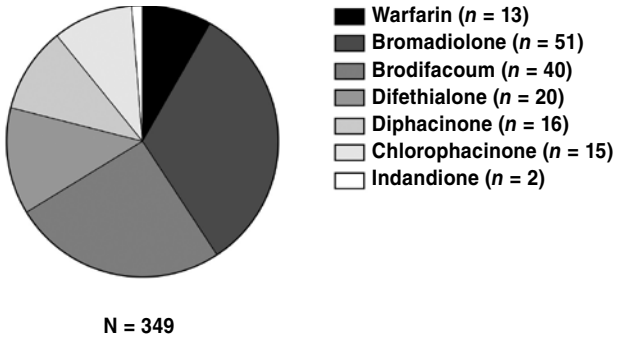

Figure 2. Distribution of active ingredient in 157 of 349 dogs with confirmed anticoagulant rodenticide intoxication.

Clinical signs and sites of hemorrhage

Clinical signs were reported in 104/349 (30%) of AR intoxications and included lethargy (*n* = 89), dyspnea (*n* = 57), evidence of external hemorrhage (ocular, oral, nasal, cutaneous or subcutaneous, vulvar or penile hemorrhages; melena/hematochezia; or hematemesis) (*n* = 46), anorexia (*n* = 26), cough (*n* = 23), vomiting (*n* = 19), diarrhea (*n* = 6), and neurological signs (seizures, paraparesis) (*n* = 3).

As part of the diagnostic evaluation, radiographs were obtained for 58/104 dogs with clinical signs of AR intoxication (thoracic radiographs in 54 dogs, abdominal radiographs in 19). No radiographs were obtained for animals that were asymptomatic. Point-of-care TFAST and AFAST became available in 2015 and were done in 33/104 dogs with clinical signs of AR intoxication and in only 7/245 asymptomatic dogs.

Sites of hemorrhage were reported in 81 cases (Figure 3). The most common site of hemorrhage was the pleural space (*n* = 35); thoracocentesis was performed in 12/35 cases and confirmed hemothorax in all cases. The other locations of hemorrhage were cutaneous or subcutaneous (ecchymosis) (*n* = 21); respiratory tract, including in the lungs (*n* = 19), based on radiographic changes or necropsy findings, epistaxis (*n* = 13), tracheal submucosal hemorrhage (*n* = 1) (identified on necropsy); oral cavity (*n* = 19), with evidence of bleeding from the tongue or gingiva; gastrointestinal tract, with melena (*n* = 15); peritoneal cavity (*n* = 11); mediastinum (*n* = 8); conjunctiva and sclera (*n* = 7); and urogenital tract, with hematuria (*n* = 6) or bleeding from the vulva or penis (*n* = 4). Suspected CNS involvement was

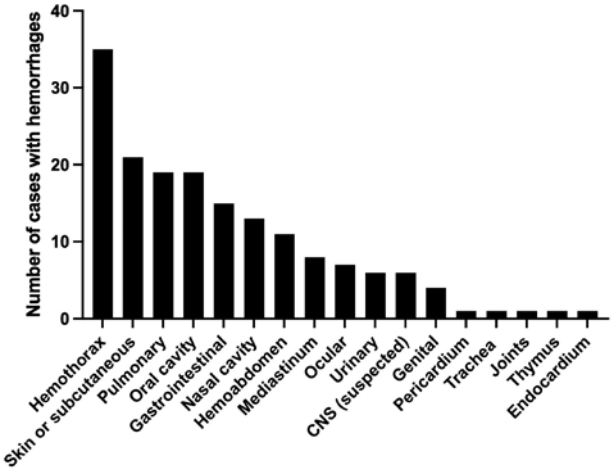

Figure 3. Site of hemorrhage for the 81 cases of dogs with anticoagulant rodenticide-induced hemorrhage.

also reported in 6 dogs but was not confirmed by advanced diagnostic imaging. Two dogs had acute onset of seizures, and another dog had extensor rigidity of the front limbs and paralysis of the back limbs, which was suspicious for multifocal hemorrhage into the cerebellum and spinal cord. Anecdotal sites of hemorrhage included eyelid hematoma (n = 1). In this case, the dog was bleeding from a surgical wound on the upper eyelid following removal of a benign mass 2 d before admission for AR intoxication. One dog also had hemarthrosis in multiple joints, confirmed by arthrocentesis; and another had thymic hemorrhages identified on necropsy. Of note, no dog had petechial hemorrhage despite severe thrombocytopenia in some cases, though hair coats were not clipped and petechia could have been missed. However, all dogs with cutaneous or subcutaneous hemorrhages had ecchymosis. Pericardial effusion was also not reported in any dog, but postmortem examination revealed multifocal hemorrhages in the endocardium and on the outer layer of the parietal pericardium in 1 dog.

Coagulation times

Of the 349 dogs with confirmed AR intoxication, PT was measured in 111 dogs and aPTT was measured in 107. Of 111 dogs with available PT measurements, 30 had increased PT on the MLA analyzer (including 19 above the limit of detection: $>$ 60 s) and 50 had increased PT on the Idexx analyzer (including 29 above the limit of detection: $>$ 100 s). Of 107 dogs with available aPTT measurements, 22 had increased aPTT on the MLA analyzer (including 19 above the limit of detection: $>$ 60 s) and 44 had increased aPTT on the Idexx analyzer (including 15 above the limit of detection: $>$ 350 s) (Table 1).

Of the 81 dogs with clinical signs of hemorrhage, PT was measured in 62 dogs and aPTT was measured in 60. Of 62 dogs with available PT measurements, 27 had increased PT on the MLA analyzer (including 18 above the limit of detection) and 28 had increased PT on the Idexx analyzer (including 24 above the limit of detection). The remaining dogs with clinical signs of hemorrhage but normal PT had received vitamin K1 before testing (usually by the referring veterinarian). Of 60 dogs with available aPTT measurements, 21 had increased aPTT on the MLA analyzer (including 17 above the limit of detection) and 26 had increased aPTT on the Idexx analyzer (including 11 above the limit of detection).

Of 303 dogs that received vitamin K supplementation, follow-up PT and aPTT results were available for 67 and 46 dogs, respectively, and were measured 48 to 72 h after discontinuation of oral vitamin K supplementation. On average, PT and aPTT were rechecked 21.9 $\pm$ 13 d after initial presentation. All recheck PT/aPTT measurements were with the MLA analyzer and none of the values were increased. The mean values at follow-up were 8.0 $\pm$ 1.3 s for PT (reference range: 10 to 13 s) and 13.7 $\pm$ 11.6 s for aPTT (reference range: 9.6 to 45 s).

Hematological analysis

Of the 73 emergency panels obtained, the mean PCV was 35 $\pm$ 14% and the mean TP was 6.1 $\pm$ 1.3 g/dL. A CBC was done in 65/349 dogs with AR intoxication and in 58/104 dogs with clinical signs of AR intoxication on initial examination. Based on the 65 CBCs, mean hematocrit was 0.30 $\pm$ 0.11 L/L (reference interval: 0.36 to 0.56 L/L), mean RBC count was 4.61 $\pm$ 1.8 $\times$ 10^{12}/L (reference interval: 5.2 to 8.2 $\times$ 10^{12}/L), and mean hemoglobin was 108 $\pm$ 40 g/L (reference interval: 133 to 197 g/L) (Table 1). Anemia, defined as PCV $<$ 36% or hematocrit $<$ 0.36 g/L, was present in 62 dogs out of 73 that had either a CBC or a PCV/TP measured. Based on reticulocyte count, the anemia was considered regenerative in 40/62 anemic dogs and non-regenerative in 22/62 anemic dogs, suggesting acute blood loss and a pre-regenerative state for the latter category. Of the 62 dogs with anemia, mean TP was 4.6 $\pm$ 1.5 g/L (reference interval: 5.5 to 7.1 g/L), consistent with blood loss.

Of the 81 dogs with evidence of hemorrhage, 49 dogs had a CBC analysis, with thrombocytopenia reported and confirmed on blood smear review in 12 cases (24%) and normal or increased platelet levels in the remaining 37 cases (76%). The mean platelet count was 120 236 $\pm$ 83 251/μL (range: 12 to 467 K/μL) in these 81 dogs, and 65 750 $\pm$ 39 269/μL (range: 12 to 126 K/μL) in the 12 dogs with thrombocytopenia, with 7/12 dogs having a platelet count $<$ 60 K/μL and 2/12 dogs having a platelet count $<$ 30 K/μL (1 dog had a platelet count of 12 K/μL and the other, a count of 20 K/μL) (Table 1). Of 46 dogs with anemia and with a CBC reported, 12 dogs had thrombocytopenia and 34 had normal-to-increased platelet counts. However, all dogs with thrombocytopenia were anemic.

Treatment

Among the 349 cases with confirmed AR intoxication, emesis was attempted with intravenous apomorphine in 189 dogs and with oral hydrogen peroxide in 61 dogs. Activated charcoal was given to 163 dogs as part of decontamination treatment on admission. Intravenous fluid therapy was administered to 73 dogs, and 34 dogs received oxygen therapy. Blood products were administered to 68/81 dogs (84%) with evidence of hemorrhage: 38 received fresh frozen plasma transfusions, 25 received fresh whole blood transfusions, 11 received packed RBC transfusions, and 8 received stored whole blood transfusions (these 8 dogs all had anemia with a mean PCV of 29 $\pm$ 9%). No

Table 1. Coagulation times (prothrombin time and activated partial thromboplastin time) and results of hematological analysis in dogs with anticoagulant rodenticide intoxication.

Parameters	Results	Reference intervals (RI)
On presentation		
PT (n = 111)		
MLA (n = 51)	• 19 dogs > UDL • 30 dogs had increased PT	10 to 13 s
POC (n = 62)	• 29 dogs > UDL • 50 dogs had increased PT	11 to 17 s
aPTT (n = 107)		
MLA (n = 48)	• 19 dogs > UDL • 22 dogs had increased aPTT	9.6 to 45 s
POC (n = 61)	• 15 dogs > UDL • 44 dogs had increased aPTT	72 to 102 s
PCV (n = 73)	• 35 ± 14%	36 to 56%
Hematocrit (n = 65)	• 0.30 ± 0.11 L/L	0.36 to 0.56 L/L
RBC count (n = 65)	• 4.61 ± 1.8 × 10^{12}/L	5.2 to 8.2 × 10^{12}/L
Hemoglobin (n = 65)	• 108 ± 40 g/L	133 to 197 g/L
Total proteins (n = 73)	• 6.1 ± 1.3 g/dL	5.6 to 7.4 g/dL
Platelet count (n = 65)	• 147 K ± 101 K/μL	117 K to 418 K/μL
Platelet count in dogs with AR-induced hemorrhages (n = 49)	• 120 K ± 83 K/μL • 66 K ± 39 K/μL in the 12 dogs with thrombocytopenia	117 K to 418 K/μL
Follow-up after treatment		
PT (n = 67), MLA	• Mean ± SD: 8.0 ± 1.3 s • All values were within RI	10 to 13 s
aPTT (n = 46), MLA	• Mean ± SD: 13.7 ± 11.6 s • All values were within RI	9.6 to 45 s

aPTT — Activated partial thromboplastin time; AR — Anticoagulant rodenticide; MLA — MLA Electra 750 photo-optical plasma coagulation timing instrument (Beckman Coulter, Mississauga, Ontario); PCV — Packed cell volume; POC — Patient-side Idexx Coag Dx Analyzer (Idexx, Markham, Ontario); PT — Prothrombin time; RBC — Red blood cell; SD — Standard deviation; UDL — Upper detection limit.

blood products were administered to any dog not having clinical signs. Among dogs with evidence of hemorrhage that survived to discharge, the percentage of dogs that received blood products (81%) was significantly higher than that of dogs that did not receive blood products (19%) (P = 0.017).

In 223/349 confirmed AR intoxication cases (64%), vitamin K1 supplementation was started in hospital, at a mean dose of 3.6 ± 1.3 mg/kg (range: 1.2 to 5.5 mg/kg), administered SC in 187 dogs, PO in 34 dogs, and IM in 2 dogs. Vitamin K1 supplementation was started in hospital for 70/81 dogs (70%) with AR-induced hemorrhage. At the time of discharge, oral vitamin K1 supplementation was prescribed for 310/349 dogs (89%), and 68/81 dogs (84%) with AR-induced hemorrhages (the remaining 13 dogs were euthanized or died before discharge). The mean dose of oral vitamin K supplementation was 2.6 ± 0.9 mg/kg. The frequency of oral administration of vitamin K1 was available for all cases and was q24h (n = 70), q12h (n = 235), and q8h (n = 5). The mean vitamin K1 treatment duration at home for all AR intoxications was 25.8 ± 7.5 d (means of 20 d for FGARs and 27 d for SGARs). Of 81 dogs with evidence of hemorrhage, combination treatment with intravenous fluid therapy, blood product transfusion, and vitamin K1 supplementation was reported in 58 dogs (72%).

Outcome

Of 104 dogs with clinical signs of AR toxicity, 78 dogs were hospitalized, with a mean length of hospitalization of 3.0 ± 1.5 d (range: 1 to 8 d). Of the 245 asymptomatic dogs, only 12 were hospitalized, with a mean length of hospitalization of 1.8 ± 0.6 d.

Of the 104 cases with clinical signs of AR intoxication on presentation, 91 dogs survived to discharge and 13 did not survive (7 dogs were euthanized). Of the 81 cases of AR intoxication with hemorrhage, 70 dogs (86%) survived to discharge and 13 dogs (14%) did not survive (7 dogs were euthanized). All dogs without clinical signs on admission survived to discharge.

Discussion

To the authors' knowledge, this is the first study evaluating the signalment and clinical, laboratory, treatment, and outcome features of a large population of dogs with AR intoxication in western Canada. Cases of AR intoxication in dogs are frequently seen at our institution, possibly because of the intensive nature of agriculture in our geographic region and the large rodent populations associated with our climate. Most cases (53%) were seen between mid-summer and early fall (July to October), when dogs have increased access to barns, farms, lake cottages, and even parks or wildlife areas.

The active AR ingredient was unknown in 55% of the cases in this study. When the active ingredient was known, SGARs accounted for 92% of cases, with bromadiolone, brodifacoum, and difethialone being the most common toxins. This first reflects that consumers have moved away from buying FGARs such as warfarin and preferably purchase SGARs that are more toxic and more efficient at killing rodents (1,3). It may also

reflect a change in which products are available for sale through hardware and pest extermination supply stores. A similar overrepresentation of SGARs has been documented in terrestrial birds of prey from western Canada with AR intoxication (16). In that study, brodifacoum, bromadiolone, and difethialone were detected in 58%, 54%, and 34% of raptor livers, respectively (16).

The clinical signs of AR intoxication vary depending on the site of hemorrhage. In this study, hemorrhage occurred at multiple sites, including the pleural space, peritoneal cavity, oral cavity, cutaneous or subcutaneous tissues, lungs, gastrointestinal tract, upper respiratory tract, mediastinum, conjunctiva and sclera, urogenital tract, CNS, eyelids, and joints. The most common site of documented hemorrhage in our study was the pleural space, accounting for 43% of hemorrhage sites (1,3,17). In a recent study, hemothorax was also the most prevalent single site of hemorrhage, and about 1/2 of the dogs had evidence of intracavitary hemorrhage (2). However, AR intoxication may cause hemorrhage at any site within the body, as seen in our study and supported by other retrospective studies, case reports, and case series, which reported hematometra (18), gastric wall hemorrhage (19), ureteral hemorrhage (20), extradural hematoma (21), tracheal mucosal hemorrhage (22), and others. Interestingly, conjunctival and scleral hemorrhages were not uncommon in the current study and were seen in 7/81 dogs with AR-induced hemorrhage. A previous study similarly reported 6 dogs with suspected or confirmed AR toxicity that presented with predominantly ocular manifestations, including subconjunctival hemorrhage, exophthalmos, and orbital pain (23). Interestingly, no pericardial effusion was reported in the present study, though the pericardium is a reported site of hemorrhage in cases of AR toxicity (2).

Thrombocytopenia was documented after blood smear review in 12 cases (24%) of the 49 cases of AR intoxication with hemorrhage that had a CBC, and 2 dogs had marked thrombocytopenia (< 30 K/μL). In another study, thrombocytopenia was documented in 8/11 dogs with AR-induced hemorrhage and was also marked in 2 cases (< 30 K/μL) (13). In both studies, petechial hemorrhage was not noted in any case. This highlights that AR intoxication should be part of the differential diagnosis for dogs with hemorrhage accompanied by mild-to-severe thrombocytopenia, and especially should be distinguished from disseminated intravascular coagulation.

Treatment of dogs with AR intoxication involves administration of vitamin K1 and transfusion therapy to provide active clotting factors, and in some cases, RBCs, if moderate-to-severe anemia is evident (1,3). Blood products were administered in 84% of dogs with evidence of hemorrhage; a recent study involving 62 dogs with hemorrhage secondary to AR intoxication similarly reported 77% of dogs received a transfusion (2). Among dogs with evidence of hemorrhage that survived to discharge, the percentage of dogs that received blood products (81%) was significantly higher than that of dogs that did not receive blood products (19%). Similar results were reported for another recent study, where dogs that received blood transfusions had a significantly higher rate of survival to discharge (64%) *versus* dogs that did not receive blood products (36%) (2). Of the 81 cases of AR intoxication with hemorrhage, 70 dogs (86%) survived to discharge and 13 dogs (14%) did not survive; these results are similar to those in previous reports with survival-to-discharge rates of 87% (2) and 83% (8). However, of the 13 dogs that did not survive, 7 dogs were euthanized shortly after admission, and financial constraints were reported in 6/7 cases. This may have decreased the survival rate of dogs with AR-induced hemorrhage seen in our institution.

Limitations of this study include the retrospective nature of the study design, and thus the lack of standardization in diagnosis and treatment. Due to the broad time frame of the study (1999 to 2022), single blood analyzers were not used for all samples; however, reference ranges for the analyzer used at the time of presentation have been used to avoid any overinterpretation of the data. One of the inclusion criteria was that the owner witnessed the dog ingesting a formed product matching common characteristics of AR products in the vomitus when emesis was induced; however, no laboratory verifications were done to confirm these products were ARs. Another inclusion criterion was that the dog had clinical signs of coagulopathy in which an elevated PT ± aPTT was documented, along with evidence of normalization of PT/aPTT measurements following vitamin K1 therapy. The authors cannot exclude the possibility that the increase followed by normalization on PT/PTT was secondary to liver failure; however, this is unlikely given the absence of other clinical and paraclinical data (*i.e.,* hyperbilirubinemia) to support hepatic insufficiency in these cases.

This study highlights the wide spectrum of clinical signs and sites of hemorrhage in dogs with AR intoxication, despite the classical association of AR intoxication with cavitary hemorrhages. The most common site of hemorrhage was the pleural space, accounting for 43% of the sites of hemorrhage. Thrombocytopenia was not uncommon and was reported in 24% of dogs. Blood products were administered to 84% of dogs with AR-induced hemorrhages, most commonly fresh frozen plasma (56% of cases). The prognosis for AR intoxication is good, with 86% of dogs with AR-induced hemorrhages surviving to discharge. CVJ

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Student Paper Communication étudiante

Resolution of necrotizing cellulitis in a dog using basic wound management

Jacalyn Normandeau

Abstract — An 8-month-old intact male golden retriever dog was seen as a case requiring urgent attention 2 d after an altercation with a cat. The dog was febrile, anorexic, and reluctant to move. There was soft-tissue swelling on the left ventral abdomen that progressed to necrotizing cellulitis. Despite the severity of the wound, client financial constraints necessitated management on a low-cost, outpatient basis using empirical antibiotics and raw-honey bandages. The wound resolved fully in 5 wk.

Résumé — Résolution de cellulite nécrosante chez un chien grâce à la gestion de base des plaies. Un chien golden retriever mâle intact de 8 mois a été considéré comme un cas nécessitant une attention urgente 2 jours après une altercation avec un chat. Le chien était fébrile, anorexique et hésitait à bouger. Il y avait une enflure des tissus mous sur l'abdomen ventral gauche qui a évolué vers une cellulite nécrosante. Malgré la gravité de la blessure, les contraintes financières des clients ont nécessité une prise en charge ambulatoire à faible coût, utilisant des antibiotiques empiriques et des bandages au miel cru. La plaie s'est complètement résolue en 5 semaines.

(Traduit par Dr Serge Messier)

Can Vet J 2024;65:504–506

Case description

An 8-month-old intact male golden retriever dog was brought into a rural mixed-animal clinic in Nova Scotia on July 15, 2023. The dog weighed 28.1 kg. He had been chased and attacked by a cat on July 13, 2023, and had since become lethargic, pyrexic (40.0°C), anorexic, and reluctant to move, with a heart rate of 156 beats per minute. To further the examination, the dog was administered subcutaneous (SC) atropine (Atro-SA 0.5 mg/mL; Rafter 8 Products, Calgary, Alberta), 0.011 mg/kg of body weight; butorphanol (Torbugesic 10 mg/mL; Zoetis, Parsippany-Troy Hills, New Jersey, USA), 0.062 mg/kg; and acepromazine (Acevet 25 mg/mL; Vétoquinol, Lavaltrie, Quebec), 0.015 mg/kg; followed by an intravenous (IV) injection of an equal parts ketamine/diazepam combination (Narketan 100 mg/mL; Vétoquinol and Diazepam 5 mg/mL; Sandoz Canada, Boucherville, Quebec), 0.15 mL/kg. No superficial or puncture wounds were detected: however, cellulitis with a central focal redness was seen on the left ventral abdomen, dorsal to the prepuce extending from mid-prepuce to xiphoid process. The area was painful, firm, and not exuding any fluid at the time of initial examination. No fluid was observed on ultrasound or aspirated from the tissue. A complete blood (cell) count showed leukopenia [2.5×10^9/L, reference range (RR): 6.0 to 17.0 $\times 10^9$/L], lymphopenia (0.36×10^9/L, RR: 1.0 to 4.8 $\times 10^9$/L), monocytopenia (0.09×10^9/L, RR: 0.2 to 1.5 $\times 10^9$/L), and thrombocytopenia (78×10^9/L, RR: 165 to 500 $\times 10^9$/L). Biochemistry analysis results indicated increased ALP (193 U/L, RR: 20 to 150 U/L) and ALT (232 U/L, RR: 10 to 118 U/L) and decreased potassium (3.4 mmol/L, RR: 3.7 to 5.8 mmol/L), but no other abnormalities were present. The dog was given a single dose of IV cefazolin (Cefazolin 334 mg/mL; Fresenius Kabi Canada, Toronto, Ontario), 22 mg/kg; SC meloxicam (Metacam 5 mg/mL; Boehringer Ingelheim, Ingelheim, Germany), 0.2 mg/kg; and 100 mL of SC fluids (0.9% saline); and was prescribed meloxicam (Metacam 1.5 mg/mL; Boehringer Ingelheim), 0.1 mg/kg, PO, q24h and enrofloxacin (Baytril 150 mg; Elanco Animal Health, Greenfield, Indiana), 150 mg, PO, q24h. The presumptive diagnosis was sepsis and cellulitis due to a cat scratch or bite. Bacterial culture and sensitivity analyses were not completed due to client financial constraints and the necessity for immediate empirical treatment. The client contacted the clinic *via* email the next day (3 d after the cat altercation) reporting increased reddening with the cellulitis (Figure 1).

At 6 d after the cat altercation, the dog was reexamined at another clinic under the same management but closer to the client's residence. The dog's mentation had improved and he exhibited less pain. The dog had a temperature of 39.0°C, but the inappetence persisted and the cellulitis had developed a necrotic center. Enrofloxacin was discontinued and amoxicillin/clavulanic acid (Clavaseptin 250 mg; Vétoquinol), 375 mg, PO, q12h, was initiated. The wound was bandaged with chlorohexidine

Ontario Veterinary College, University of Guelph, 50 Stone Road, East, Guelph, Ontario N1G 2W1.

Please address all correspondence to Jacalyn Normandeau; email: jacalyn@uoguelph.ca

Figure 1. Necrotizing cellulitis in an 8-month-old intact male golden retriever dog, 3 d after an altercation with a cat.

Figure 3. Purulent material had resolved and fresh blood was present in the wound after 2 d of honey bandaging (10 d after the cat altercation).

Figure 2. A hole 8 cm × 3 cm × 2 cm deep with purulent debris developed after the necrotic center of the necrotizing cellulitis wound dehisced and sloughed away 8 d after the cat altercation.

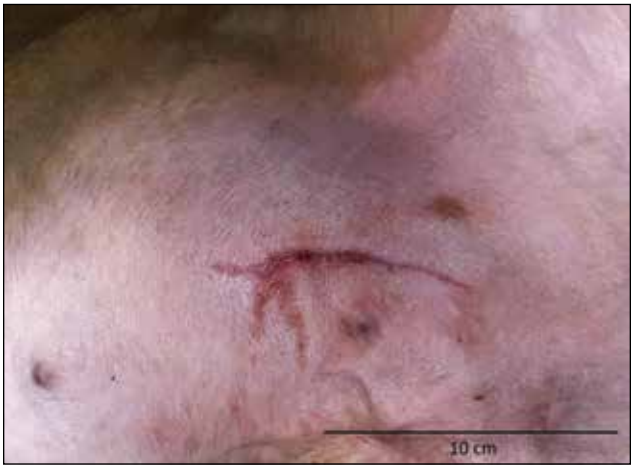

Figure 4. Second-intention healing of the necrotizing cellulitis wound took 5 wk in total, including 15 d of antibiotic treatment and 12 d of raw-honey bandaging with bandage changes every 2 d.

impregnated gauze (Bactigras; Smith and Nephew, Watford, UK) in a wraparound bandage consisting of stretch gauze and Vetwrap (3M Deutschland GmBH, Neuss, Germany). The dog was prescribed gabapentin (Teva-Gabapentin 300 mg; TEVA Canada, Toronto, Ontario), 300 mg, PO, q8h as needed, and fitted with a Funcol E-collar (Genia, New York, New York, USA). Referral options were discussed with the client but, due to financial constraints and distance to referral centers, the client opted to manage the wound and bandage changes on an outpatient basis.

The dog was brought to the clinic for a recheck examination 8 d after the cat altercation. He was bright, alert, and responsive; his temperature was 38.7°C; and the cellulitis had partially receded. However, the necrotic center of the wound had dehisced to a hole 8 cm × 3 cm × 2 cm deep with purulent debris present (Figure 2). The E-collar was replaced with a stretch shirt (Medical Pet Shirts, Prismalaan, Netherlands) and the dog continued receiving meloxicam and amoxicillin/clavulanic acid. The wound was packed with raw-honey-covered gauze and bandaged with honey-covered Bactigras gauze and a wraparound bandage, as described. The client declined a tie-over bandage due to financial constraints associated with the sedation necessary for suture placement. The dog was brought for rechecks 2 and 5 d later. The wound was clear of purulent material with fresh blood present after 2 d of honey bandaging (10 d after the cat altercation; Figure 3), and had contracted in size with granulation tissue forming after 5 d of honey bandaging (13 d after the cat altercation). The dog continued receiving amoxicillin/clavulanic acid and meloxicam until 18 d after the cat altercation, and the client was advised to continue honey-bandage changes every 2 d. The dog was rechecked 1 wk later and showed marked wound contracture with granulation tissue filling in the dead space. Given a lack of exudate from the wound, honey bandaging was discontinued but bandaging with Bactigras and stretch gauze continued until the wound fully closed at 5 wk after the altercation with the cat (Figure 4).

Discussion

Necrotizing cellulitis with pain, soft-tissue swelling, and redness of the skin has been documented following a cat scratch or

bite (1) and is often caused by the Gram-negative coccobacillus *Pasteurella multocida* (2,3). However, necrotizing cellulitis unrelated to cat altercations often involves multiple bacterial species or *Streptococcus* spp. (4). In a retrospective study of 47 dogs with severe soft tissue infections, an overall mortality rate of 53% was observed, in which 85% of those infections involved necrotizing cellulitis (5). Hallmarks of necrotizing cellulitis include pain disproportionate to the apparent lesion and rapid progression of the wound (4), both of which were noted in this case. Treatment typically involves selection of broad-spectrum antibiotic therapy following culture and sensitivity, but a retrospective study identified that 81% of empirical antibiotic choices were appropriate (5). One other paper reported a similar case of necrotizing cellulitis in a dog following an altercation with a cat in which a superficial wound was lacking (6). In that case, the resulting wound was intensively managed for 6 d in a referral setting, including biopsy of the wound with bacterial culture and sensitivity, IV antibiotics, surgical debridement, and full-thickness skin grafts harvested from the flank regions (6). This case was seen in a rural general practice setting. Ideally, the necrotizing cellulitis would have been managed with a tie-over bandage, considering the anatomical location (leading to difficulty maintaining proper bandage placement) and the potential need for surgical debridement, drain placement, and delayed primary closure. However, financial limitations precluded additional diagnostic tests, such as bacterial culture and sensitivity testing, as well as further procedures involving sedation. Although surgery typically involves a larger up-front cost, frequent bandage changes can incur a higher cost over a lengthy wound healing period. Given that the client was willing and able to complete most of the bandage changes at home, the cost of supplies and 8 brief rechecks was less than the cost of surgical tie-over bandage placement and/or delayed primary closure would have been in this case. Therefore, the wound was left to heal by second intention, managed with empirical antibiotics and honey bandaging on an outpatient basis, and took 5 wk to resolve.

When necrotic cellulitis developed and the center of the wound sloughed away, the open wound was managed using a raw-honey bandage. This was due to the well-documented use of unrefined honey for necrotic, exudative, purulent open wounds (7) and its ready availability and low cost. Unrefined honey has anti-inflammatory, antibacterial, and antioxidant properties due to its natural acidity and hyperosmolarity (which prevent bacterial growth), local immune stimulation (leukocyte cytokine release and antibody production), continuous enzymatic hydrogen peroxide production at noninflammatory levels, and other unidentified compounds (7–10). There is evidence that honey bandaging leads to reduced wound-healing time, reduced wound exudate and edema, and a clean wound bed that may increase success of surgical closure and skin grafts (9,10). Certain types of honey (*i.e.,* Manuka honey) are reported to have additional antibacterial properties beyond those present naturally in all unrefined honey (9,10). Medihoney (Comvita, Te Puke, New Zealand) is a product available for use in veterinary medicine that contains Manuka honey sterilized *via* gamma irradiation (11). However, raw honey from the local pharmacy was used in this case, as the clinic did not carry Medihoney and Manuka honey was not locally available. Nevertheless, raw honey improved the wound dramatically between Day 8 and Day 10 by debriding purulent necrotic tissue and leaving relatively healthy vitalized tissue underneath. Continued use of raw-honey bandages while the wound was exudative resulted in formation of a healthy bed of granulation tissue and secondary closure of the dead space in < 2 wk, suggesting that, in this case, food-grade raw honey had sufficient properties to aid wound healing.

Management of this case was shared by all 4 veterinarians in the practice: The dog was first brought in as an emergency to the on-call veterinarian on a Saturday at 1 clinic (where 24-hour mixed-animal care is provided to all clients), and then brought for rechecks on weekdays at the other clinic (where all veterinarians in the practice rotate through each day). This case report highlights the importance of communication and cooperation between veterinarians for case continuity when care at a single location by 1 veterinarian is not possible due to challenges presented in rural, mixed veterinary medicine. Here, as the closest referral hospitals are located 4 to 6 h away by car in Halifax, Nova Scotia; Charlottetown, Prince Edward Island; and Moncton, New Brunswick, referral is an impractical option for many clients in the area. These factors necessitate local management of complicated cases, often within financial constraints. This case is an example of successfully overcoming the challenges of rural veterinary medicine to resolve a case of necrotizing cellulitis in a dog using basic wound management.

Acknowledgments

I thank the veterinarians and staff at my externship clinic for hosting me as a 4th-year student. Additional thanks to Kaitlin Doering for reviewing this case report. CVJ

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Answers to Quiz Corner
Corrigé du test éclair

1. A) It is most appropriate to remove the umbilical remnant and aspirate and flush the stifle under general anesthesia. Also, administer systemic broad-spectrum antimicrobials.

Omphalophlebitis is one of the more common conditions in neonatal foals. Heat, pain, swelling, and/or discharge of the umbilical stump or a patent urachus is seen.

In some foals, only the internal remnants are affected and the foals have a fever, high white blood cell count, hyperfibrinogenemia, and increased serum amyloid A. Ultrasound examination is necessary to evaluate the internal remnants (1 umbilical vein, 1 urachus, and 2 umbilical arteries).

Treat uncomplicated cases with long-term systemic antimicrobials (ideally based on culture and sensitivity). Cases with evidence of sepsis (*i.e.,* another body system is affected, such as a swollen joint or pneumonia) or infections that extend to the liver should be treated surgically.

Figure 2 (below) is a photo of this foal's umbilical remnant that was removed at surgery. In the figure, the arrow is pointing to the enlarged umbilical artery adjacent to the enlarged urachal stump. The scissors are placed for size reference.

1. A) La meilleure option est de retirer le moignon ombilical, de ponctionner et de rincer le grasset sous anesthésie générale, et d'administrer des antimicrobiens systémiques à large spectre d'action.

L'omphalophlébite est l'une des affections les plus courantes chez les poulains nouveau-nés. On observe de la chaleur, de la douleur, un gonflement et/ou un écoulement du moignon ombilical ou une persistance du canal de l'ouraque.

Dans certains cas, seuls les vestiges internes sont touchés, et les poulains présentent de la fièvre, une leucocytose, une hyperfibrinogénémie et une augmentation du taux d'amyloïde A sérique. Un examen échographique est nécessaire pour évaluer les vestiges internes (la veine ombilicale, le canal de l'ouraque et les deux artères ombilicales).

Les cas non compliqués peuvent être traités par l'administration d'un antimicrobien systémique durant une longue période (idéalement en fonction des résultats d'une culture et d'un antibiogramme). Les cas qui présentent des signes de septicémie (c'est-à-dire lorsqu'un autre système est affecté, par exemple si le poulain a une articulation enflée ou une pneumonie) ou une infection qui s'étend au foie devraient être traités chirurgicalement.

La figure 2 est une photo du moignon ombilical de ce poulain qui a été retiré par une intervention chirurgicale. La flèche pointe vers l'artère ombilicale distendue adjacente au volumineux vestige du canal de l'ouraque. Les ciseaux servent de référence pour la taille.

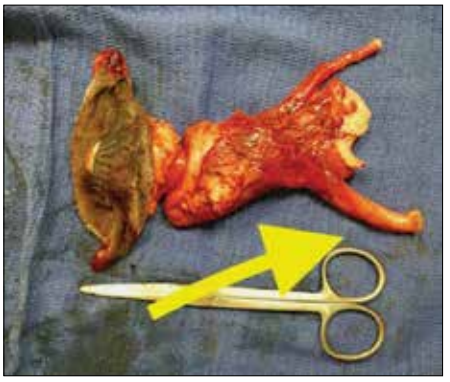

Figure 2.
Image courtesy of Nora Grenager, VMD, DACVIM.
Image de Nora Grenager, D.M.V., DACVIM.

Stifle effusion in a foal of this age (especially with other evidence of infection, such as an enlarged umbilicus) should be rapidly investigated and treated as a septic joint until proven otherwise.

Specific diagnostic tests and treatments include aseptic aspiration with culture and cytology of the aspirated fluid, joint lavage, and intra-articular antimicrobials, ± regional limb perfusion with antimicrobials.

Reference

1. Smith BP, Van Metre DC, Pusterla N, eds. Large Animal Internal Medicine, 6th ed. North York, Ontario: Elsevier Canada, 2019:267–280.

2. E) This is atrial standstill, characterized by an absence of P-waves and a slow heart rate, usually < 60 bpm. In this case, atrial standstill is caused by hyperkalemia due to hypoadrenocorticism (Addison's disease).

Along with hyperkalemia, the serum biochemistry shows other classic changes commonly seen in Addisonian patients (*e.g.,* hyponatremia, hypercalcemia, hypoglycemia, azotemia) and lack of a stress leukogram in a sick patient.

Other causes of hyperkalemia include acute oliguric/anuric kidney injury, urinary obstruction, or uroperitoneum due to a ruptured bladder.

Hyperkalemia severe enough to produce atrial standstill is a medical emergency. Correct blood potassium levels with aggressive IV fluid therapy and IV bicarbonate and dextrose ± insulin. Intravenous calcium gluconate is recommended for its cardioprotective effects, but it does not correct the hyperkalemia.

Hypercalcemia does not cause atrial standstill but it can cause ventricular arrhythmias if ionized calcium levels are severely increased. Hypovolemia can cause tachycardia, but it should not cause a bradyarrhythmia.

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The questions and answers are provided by Zuku Review, online veterinary test prep.

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Un épanchement du grasset chez un poulain de cet âge (en particulier en présence d'autres signes d'infection, comme une augmentation de volume du moignon ombilical) devrait être examiné sans délai et considéré comme indiquant une articulation septique jusqu'à preuve du contraire.

Les tests de diagnostic et les traitements spécifiques comprennent la ponction aseptique avec culture et cytologie du liquide aspiré, le rinçage de l'articulation, et l'administration intra-articulaire d'antimicrobiens, avec ou sans perfusion régionale du membre au moyen d'antimicrobiens.

Référence

1. Smith BP, Van Metre DC, Pusterla N, eds. Large Animal Internal Medicine, 6th ed. North York, Ontario: Elsevier Canada, 2019:267–280.

2. E) Il s'agit d'une paralysie auriculaire, caractérisée par l'absence d'ondes P et une fréquence cardiaque lente (généralement < 60 bpm). Dans ce cas-ci, la paralysie auriculaire est causée par une hyperkaliémie due à l'hypoadrénocorticisme (maladie d'Addison).

Outre l'hyperkaliémie, la biochimie présente d'autres changements classiques couramment observés chez les patients atteints de la maladie d'Addison (hyponatrémie, hypercalcémie, hypoglycémie, azotémie) et l'absence d'un leucogramme de stress chez un patient malade.

Les lésions rénales aiguës causant une oligurie ou une anurie, l'obstruction urinaire, et l'uropéritoine dû à une rupture de la vessie sont d'autres causes possibles d'hyperkaliémie.

Une hyperkaliémie suffisamment grave pour provoquer une paralysie auriculaire est une urgence médicale. Il faut corriger le taux de potassium sanguin par une fluidothérapie intraveineuse énergique et l'administration de bicarbonate et de dextrose (± insuline) par voie intraveineuse. Le gluconate de calcium intraveineux est recommandé pour ses effets cardioprotecteurs, mais il ne corrige pas l'hyperkaliémie.

L'hypercalcémie ne provoque pas de paralysie auriculaire, mais elle peut entraîner des arythmies ventriculaires si le taux de calcium ionisé est très élevé. L'hypovolémie peut provoquer une tachycardie, mais elle ne devrait pas entraîner une bradyarythmie.

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Veterinary Practice Management
Gestion d'une pratique vétérinaire

Keeping revenues ahead of inflation

Maintenir une croissance des revenus supérieure à l'inflation

Darren Osborne

Despite recent media criticism, companion animal veterinary fees have barely kept up with inflation in the last 2 y. Revenues have increased over that time, but the source of higher revenues was more clients — not higher fees. After adjusting for inflation, revenues were flat.

The increased number of clients in the last 2 y can be directly attributed to the pandemic. According to the 2022 Ontario Veterinary Medical Association (OVMA) Pet Owner's Survey, stay-at-home orders and an increase of people working from home was responsible for a 25% jump in pet ownership. There was a 9% increase in 2021 and another 14% increase in 2022. Those outside the veterinary profession saw this as a windfall, but staffing shortages and social distancing restrictions made it difficult for veterinary hospitals. Most veterinary hospitals were unable to take on new clients and existing clients had to wait weeks for access to elective care. To make matters worse, the average companion animal practice had fewer staff in 2023 than they did in 2022 or 2021 (1).

The number of pets was not the only thing increasing at unprecedented rates; inflation shot up to a 40-y high in 2022 (2), which led to increased veterinary expenses. Although most costs went up with inflation, labour costs soared. Last year, according to the Canadian Veterinary Medical Association (CVMA) Provincial Reports on Compensation and Benefits for Associate Veterinarians, 8 of 10 provinces reported double-digit increases in the cost of veterinary labor and 5 provinces reported

Malgré les récentes critiques publiées dans les médias, les coûts des soins vétérinaires pour les animaux de compagnie ont à peine suivi l'inflation au cours des deux dernières années. Les revenus des pratiques vétérinaires ont augmenté au cours de cette période, mais cette hausse s'explique par l'augmentation du nombre de clients, et non par l'augmentation des tarifs. En fait, si on tient compte de l'inflation, les revenus sont restés stables.

L'augmentation du nombre de clients au cours des deux dernières années peut être directement attribuée à la pandémie. D'après un sondage réalisé en 2022 par l'Ontario Veterinary Medical Association (OVMA) auprès de propriétaires d'animaux de compagnie, le confinement et le télétravail durant la pandémie ont entraîné une croissance de 25 % du nombre de propriétaires d'animaux, ce nombre ayant augmenté de 9 % en 2021 et de 14 % en 2022. De l'extérieur, cette situation a été perçue comme une bénédiction, mais le manque de personnel et les restrictions liées à la distanciation sociale ont créé des difficultés pour les équipes vétérinaires. La plupart ont dû refuser des nouveaux clients, et les clients existants ont dû attendre des semaines pour avoir accès à des soins non urgents. Pour compliquer davantage les choses, les cliniques vétérinaires pour animaux de compagnie avaient en moyenne moins de personnel en 2023 qu'en 2022 ou 2021 (1).

Il n'y a pas que le nombre d'animaux de compagnie qui a connu une flambée sans précédent : l'inflation a atteint son plus

Darren Osborne has been the Director of Economic Research for the Ontario Veterinary Medical Association for over 20 years. He completed an MA (Economics) from York University and has worked as an economic analyst in veterinary medicine, dentistry, human medicine, and the transport industry.

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Address all correspondence to the CVMA (admin@cvma-acmv.org).

Darren Osborne est directeur de la recherche économique pour l'Ontario Veterinary Medical Association depuis plus de 20 ans. Il a obtenu une maîtrise en économie de l'Université York et a travaillé comme analyste économique en médecine vétérinaire, en médecine dentaire, en médecine humaine et dans l'industrie des transports.

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Veuillez adresser toute correspondance à l'ACMV (admin@cvma-acmv.org).

Table 1/Tableau 1. Growth observed in 2022 and 2023.
Croissance observée en 2022 et 2023.

	2023	2022
Revenue/Revenus	4%	10%
Active clients/Clients actifs	0%	10%
Average fees/Tarifs moyens	4%	7%
Inflation	4%	7%
Inflation adjusted Ajustement en fonction de l'inflation		
Revenue/Revenus	0%	3%
Fees/Tarifs	0%	0%

increases that were 2 or more times the rate of inflation. Given veterinary labor accounts for a quarter of expenses in the average veterinary hospital, overall expenses went up higher than the rate of inflation. That meant revenue growth had to exceed the rate of inflation to cover increased costs.

In 2022, the average companion animal hospital in Canada saw revenue grow 10% (Table 1) whereas inflation was running at 7%. The same year the number of active clients grew 10% and the average hospital raised their fees 7% to match inflation. In a perfect world, revenues in 2022 could have gone up 17%–10% from increased clients and 7% from higher fees, but the average growth in revenue was lower due to inefficiencies in managing more clients, staffing shortages and productivity challenges. A higher number of clients helped propel revenue.

Last year, companion animal hospitals saw revenue growth match inflation at 4%, with zero growth in the number of active clients. Therefore, all the growth in revenue came from the 4% average fee increase. At first glance, zero growth in clients may appear alarming but for most practices, zero growth in clients made their existing client base manageable. Not growing for them was like an airplane leveling off; just because an airplane stops gaining altitude does not mean it's going to crash. The downside to zero client growth in 2023 was that fee increases only matched inflation and since labor costs were running higher than inflation, many hospitals will be worse off financially then they were the year before.

Inflation is expected to get back to less than 3% which will provide an opportunity for veterinary hospitals to get ahead of inflation with modest fee increases. With client numbers leveling off, veterinary hospitals can take a step back and refocus on getting more productive with existing clients and continue to drive revenue forward.

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haut niveau depuis 40 ans en 2022 (2), ce qui a fait grimper les dépenses pour les pratiques vétérinaires. L'inflation a fait bondir la plupart des postes de dépenses, et les coûts de la main-d'œuvre ont explosé. L'année dernière, selon les rapports provinciaux de l'Association canadienne des médecins vétérinaires (ACMV) sur la rémunération et les avantages sociaux des vétérinaires salariés, le taux d'augmentation du coût de la main-d'œuvre vétérinaire a été à deux chiffres dans 8 provinces sur 10 et au moins deux fois plus élevé que le taux d'inflation dans 5 provinces. Étant donné que la main-d'œuvre vétérinaire représente le quart des dépenses d'une pratique vétérinaire moyenne, la hausse globale des dépenses a été supérieure au taux d'inflation. Par conséquent, la croissance des revenus devait dépasser l'inflation pour contrebalancer la hausse des dépenses.

En 2022, la clinique moyenne pour animaux de compagnie au Canada a vu ses revenus augmenter de 10 % (tableau 1), alors que l'inflation était de 7 %. La même année, le nombre de clients actifs a grimpé de 10 % et la clinique moyenne a majoré ses tarifs de 7 % pour suivre l'inflation. Dans un monde idéal, les revenus en 2022 auraient pu croître de 17 % (10 % grâce à l'augmentation du nombre de clients et 7 % grâce à la hausse des tarifs), mais la croissance moyenne des revenus a été plus faible en raison de problèmes d'efficacité liés à la gestion d'un plus grand nombre de clients, de la pénurie de personnel et d'une productivité non optimale. C'est la hausse du nombre de clients qui a contribué à accroître les revenus.

L'année dernière, les pratiques pour animaux de compagnie ont connu une croissance de leurs revenus égale à l'inflation, soit 4 %, sans aucune augmentation du nombre de clients actifs. Ainsi, la croissance des revenus était attribuable en totalité à l'augmentation moyenne des tarifs de 4 %. À première vue, l'absence de croissance du nombre de clients peut sembler alarmante, mais pour la plupart des pratiques, elle a rendu leur clientèle existante gérable. Cette situation est comparable à l'atteinte de l'altitude de croisière d'un avion – ce n'est pas parce qu'un avion cesse de prendre de l'altitude qu'il va s'écraser. L'inconvénient d'une croissance nulle de la clientèle en 2023 est que les augmentations de tarifs n'ont fait que suivre l'inflation, et comme l'augmentation des coûts de la main-d'œuvre a été supérieure à l'inflation, de nombreuses pratiques se retrouvent maintenant en moins bonne posture financière qu'elles ne l'étaient l'année précédente.

L'inflation devrait revenir à moins de 3 %, ce qui donnera l'occasion aux pratiques vétérinaires de la dépasser en augmentant modestement leurs tarifs. Le nombre de clients s'étant stabilisé, les équipes vétérinaires pourront prendre du recul, se recentrer sur l'amélioration de la productivité auprès des clients existants et continuer à faire progresser les revenus.

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Diagnostic Ophthalmology
Ophtalmologie diagnostique

Lynne S. Sandmeyer, Marina L. Leis

History and clinical signs

A 2-year-old domestic longhair cat was examined by the ophthalmology service at the Western College of Veterinary Medicine (Saskatoon, Saskatchewan). This cat was presented for evaluation of a red right eye. Treatment before referral included topical tobramycin antibiotic eye drops (Tobrex 0.3%; Novartis Canada, Montreal, Quebec), with no improvement. The menace response and dazzle reflex were absent in the right eye. The pupil was miotic in the right eye, the direct pupillary light reflex was absent in the right eye, and the consensual pupillary light reflex was absent in the left eye. The palpebral and oculocephalic reflexes were normal bilaterally. Schirmer tear test (Schirmer Tear Test Strips; Alcon Canada, Mississauga, Ontario) values were 19 and 20 mm/min in the right and left eyes, respectively. The intraocular pressures were estimated with a rebound tonometer (Tonovet; Tiolat, Helsinki, Finland) and were 69 and 24 mmHg in the right and left eyes, respectively. Fluorescein staining (Fluorets; Bausch & Lomb Canada, Markham, Ontario) of the cornea was negative bilaterally. On direct examination using a transilluminator (Welch Allyn Finoff Transilluminator; Welch Allyn, Mississauga, Ontario), abnormalities in the right eye included moderate scleral congestion, mild diffuse corneal edema, and an abnormal appearing iris. No abnormalities were noted in the left eye. Following application of 0.5% tropicamide (Mydriacyl; Alcon Canada), mydriasis was achieved in the left eye but not in the right eye. Examination of the right eye using a handheld biomicroscope (Kowa SL-17 Portable Slit Lamp; Kowa, Tokyo, Japan) revealed moderate aqueous flare, 360-degree posterior synechia of a miotic pupil, *rubeosis iridis,* and a shallow anterior chamber due to the iris bulging forward in all quadrants. No abnormalities were noted in the left eye. Indirect ophthalmoscopic (Heine Omega 500; Heine Instruments Canada, Kitchener, Ontario) examination was completed for the left eye and revealed no abnormalities, but was not possible for the right eye due to miosis and posterior synechia. A complete physical examination was otherwise unremarkable. A photograph of the right eye at presentation is provided for your assessment (Figure 1).

Department of Small Animal Clinical Sciences, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan S7N 5B4.

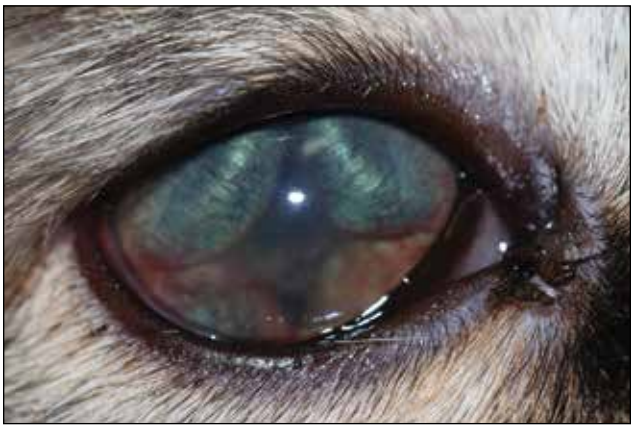

Figure 1. Photograph of the right eye of a 2-year-old domestic longhair cat.

What are your clinical diagnoses, differential etiologic diagnoses, therapeutic plan, and prognosis?

Discussion

The ophthalmic diagnosis was unilateral anterior uveitis and secondary glaucoma of the right eye. Uveitis is inflammation of the uveal tract, and the clinical manifestations of uveitis occur due to breakdown of the blood-ocular barriers and the effects of inflammatory mediators within the eye. Uveitis may present unilaterally or bilaterally, and common clinical signs include conjunctival hyperemia, miosis, aqueous flare, and a reduction of intraocular pressure. Other clinical signs include hypopyon, hyphema or fibrin in the anterior chamber, inflammatory cell adhesion to the endothelium (keratic precipitates), and iris hyperemia. Chronicity can lead to development of lymphoid nodules in the iris; pre-iridal fibrovascular membrane formation (clinically referred to as *rubeosis iridis*); and sequelae such as synechiae, secondary glaucoma, and lens luxation (1). The bulging appearance of the iris in this case is typical of iris bombé, which occurs when there is 360-degree posterior synechia that obstructs aqueous humour passage through the pupil. Aqueous humor thus builds up in the posterior chamber, causing the iris leaflets to be pushed forward, resulting in compression of the iris base against the peripheral cornea and closure of the iridocorneal angle. The obstruction of aqueous humor outflow at the pupil and the iridocorneal angle leads to intraocular pressure elevation, secondary glaucoma, and eventual blindness (1).

Uveitis may have exogenous or endogenous causes. Exogenous causes include blunt or penetrating ocular trauma and corneal ulceration (1). Endogenous causes include systemic infectious

disease [feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), feline infectious peritonitis (FIP), toxoplasmosis, bartonellosis, cryptococcosis, histoplasmosis, blastomycosis, and coccidioidomycosis], primary ocular neoplasia (uveal melanoma, ciliary body adenoma, or sarcoma), metastatic neoplasia (carcinoma) or systemic neoplasia (lymphosarcoma), and immune-mediated or idiopathic conditions (2–4).

Etiological studies of uveitis are usually retrospective in nature and are limited by variability of diagnostic testing completed. However, the average age of cats diagnosed with uveitis is ~8 y, and idiopathic uveitis is most common, reported in 37.5 to 45.7% of cases (2–5). The most common infectious cause of uveitis in cats is FIP, reported in ~15% of cases and with a median age of cats diagnosed with FIP of just ~1.5 y (3,4,6).

The diagnostic workup for a cat with uveitis should include not only a complete ocular examination but also a thorough physical examination. Additional diagnostic testing may include a CBC, serum chemistry, urinalysis, thoracic radiographs, abdominal ultrasound imaging, and select serological titres for infectious agents. Referral to an ophthalmologist for aqueous humour cytology may be helpful in diagnosis of neoplasia such as intraocular lymphosarcoma but has been shown to have little diagnostic utility in cats with non-neoplastic uveitis (7,8). The extent of the workup applied to each case will vary depending on many factors, including clinical and physical examination findings, geographic location, and client finances.

The general physical examination was normal in this cat. A CBC and serum chemistry analysis, as well as serology for FIV and FeLV, were completed by the primary care veterinarian before referral. No significant abnormalities were noted and the FIV/FeLV test was negative.

The main differential diagnosis for a bulging appearance of the iris is intraocular neoplasia. Ocular ultrasound can be performed to evaluate the iris structure and differentiate an iris mass from iris bombé. We completed an ocular ultrasound to evaluate the intraocular structures and confirmed the presence of iris bombé in addition to cataract, hyperechoic perilenticular and vitreous debris, and retinal detachment. As there was significant and painful intraocular disease and the eye was irreversibly blind based on the neuro-ophthalmic examination and retinal detachment, we recommended enucleation, which was completed at the referring clinic. Histopathologic assessment of the enucleated globe revealed rupture of the anterior lens capsule and extensive cataract. A pre-iridal fibrovascular membrane was present, causing adhesion of the iris to the lens capsule (posterior synechia) and the iris was ballooning forward into the anterior chamber (iris bombé) with adhesion of the peripheral iris to the corneal endothelium, causing closure of the iridocorneal angle (peripheral anterior synechia). The vitreous contained predominantly neutrophilic inflammation, and there was lymphoplasmacytic inflammation of the iris, ciliary body, choroid, and retina. Areas of exudative retinal detachment and subretinal haemorrhage were present, as well as retinal degeneration and optic nerve atrophy, consistent with chronic glaucoma. The histologic diagnosis was phacoclastic uveitis, endophthalmitis, retinal detachment, and secondary glaucoma. Special immunohistochemical staining to screen for intralesional bacteria was negative. Based on the histologic findings, the most likely etiology for the uveitis was trauma.

Both blunt and penetrating trauma may cause severe uveitis. Blunt-force trauma to the globe results in ocular distortion and globe compression, often leading to uveal haemorrhage, lens luxation, retinal detachment, and even scleral rupture (1). Penetrating ocular trauma occurs due to penetration of a claw, sharp foreign body, needle, or surgical instrument through the cornea or sclera (9–12). Penetration through the cornea usually presents with a corneal laceration or obvious penetration site. However, scleral penetration sites are not as obvious. There are several examples in the literature of unintentional scleral penetration with needles used to administer local anaesthesia for dentistry and rhinoscopy (11–14). In this cat, there was no clinically or histologically identifiable penetration site on the globe, and no history of a medical or surgical procedure before the ocular disease. Therefore, the cause of trauma was unknown.

In cases of trauma-induced lens rupture, the lens penetration can be overlooked, as the miosis that occurs with uveitis may obscure direct examination of the lens. Hence, dilation of the pupil is an essential component of the ocular examination to facilitate visualization of the lens. Lens capsule rupture leads to focal cataract formation, and often, extrusion of lens material in the anterior chamber. Exposure of lens material causes severe inflammation, termed phacoclastic uveitis (6). Unless appropriate therapy is initiated effectively and promptly, this inflammation commonly leads to secondary glaucoma (1). If lens capsule rupture is diagnosed early, treatment includes topical and systemic anti-inflammatory and antimicrobial treatments and topical mydriatic/cycloplegic therapy. In most cases of lens capsule rupture, treatment requires lens extraction *via* phacoemulsification in addition to medical therapy. Therefore, referral to an ophthalmologist is recommended when a penetrating ocular trauma is suspected.

There is additional concern following ocular trauma in cats due to the phenomenon of feline ocular post-traumatic sarcoma development (15,16). This is an invasive malignant neoplasia thought to occur secondary to malignant transformation of the lens epithelium following traumatic lens rupture (15). This neoplasia may take months to years to develop after initial injury but can be lethal. The tumour is locally invasive within the globe, and eventually may extend outside the sclera and along the optic nerve to invade the brain. Therefore, early enucleation is recommended in cases of severe ocular trauma in cats, and histopathologic examination is essential following removal of any feline eye when there is a history of trauma. In addition, if lens removal surgery is completed for a lens rupture, long-term follow-up will monitor for evidence of neoplasia development (16).

The cause of uveitis was initially unknown in this cat; however, development of glaucoma secondary to the inflammation warranted enucleation. Histopathologic assessment revealed the most likely etiology was trauma. Early enucleation may have prevented development of potentially fatal feline ocular post-traumatic sarcoma. This case highlighted the diagnostic and therapeutic importance of enucleation and histopathology in blind and painful eyes.

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Veterinary Dermatology
Dermatologie vétérinaire

Nasal planum diseases in dogs

Tim Chan, Andrea T.H. Lam

Introduction

A broad range of diseases can affect the nasal planum in dogs, alone or in addition to other cutaneous manifestations; these include keratinization, infectious, immune-mediated, and neoplastic disorders (Table 1). The initial (primary) lesion and distribution, breed predisposition, and presence of other cutaneous signs are helpful in determining the etiology. This article describes key clinical features of diseases that primarily or frequently target the nasal planum in dogs.

Diagnostic procedures

The initial lesion is crucial to understanding the pathogenesis but is easily missed due to rapid progression to chronic, secondary lesions that offer less diagnostic value. A thorough history aids in establishing the type and distribution of the initial lesion. For instance, depigmentation of the nasal planum can occur at the onset of disease (*e.g.,* vitiligo), before secondary lesions, such as ulceration or crusting, appear. Conversely, in other cases, secondary depigmentation may develop as a result of healing following erosion or ulceration.

A physical examination warrants careful attention to the lesion type and location within the nasal planum. Lesions include hypopigmentation or depigmentation, erythema, erosions, ulcers, crusts, loss of architecture ("cobblestone" appearance), fissures, and nodules. Certain diseases also have characteristic anatomical distributions of lesions on the nasal planum, including the dorsal ridge, alar folds, and philtrum; or are multifocal to diffuse.

Cytology helps confirm the presence of microorganisms and may reveal neoplastic cells or acantholytic keratinocytes. Microbial culture and susceptibility testing can further support an infectious cause and guide antimicrobial selection. However, detection of microorganisms does not always indicate an infectious etiology, as bacteria and *Malassezia* are common secondary occurrences to any inflammatory process affecting the nose. Primary infections typically do not present exclusively as nasal

Dr. Chan is a dermatology resident, Cummings School of Veterinary Medicine, Tufts University, North Grafton, Massachusetts, USA.

Dr. Lam is a Board-certified veterinary dermatologist, University of California, Davis, Davis, California, USA.

Address all correspondence to Dr. Tim Chan; email: imchnt@gmail.com

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Table 1. Diseases that affect the nasal planum in dogs.

Disease	Initial (primary) lesion and distribution on nasal planum	Other clinical features	Signalment
HNPK	Hyperkeratosis — dorsal aspect	May progress to crusts, bleeding ulcers from secondary infection or trauma	Young dogs (1 to 2 *y*) Labrador retriever and greyhound
INH	Hyperkeratosis — dorsal aspect	May progress to crusts, bleeding ulcers from secondary infection or trauma	Middle-aged to older dogs No strong breed predisposition American cocker spaniel, English bulldog, miniature poodle, miniature schnauzer, and Doberman pinscher are overrepresented
MCP	Erythema, swelling, crust — alar folds and perinasal junction	Chronic lesions are fissures, erosions, depigmentation, and loss of nasal planum architecture Other mucocutaneous locations may be affected (lips, perioral) Antibiotic-responsive	German shepherd
Cryptococcosis	Nodule — nasal planum and bridge of nose	Progresses to ulcers, draining tracts Spread to other organs and systemic disease common	Active dogs (*e.g.,* hunting)
Vitiligo	Noninflammatory depigmentation with intact nasal planum architecture ("cobblestone") — multifocal	Multifocal lesions Considered a cosmetic condition in dogs	Young dogs (median: 2 *y*) Rottweiler, Doberman pinscher, and collie appear to be overrepresented
UDS	Depigmentation, erythema, swelling — diffusely affecting entire nasal planum	Progresses to crusts, erosions/ulcers, alopecia Other areas include periocular skin and lips Ocular disease precedes dermatologic signs in vast majority	Middle-aged dogs Akita, Samoyed, Siberian husky
DLE	Diffuse depigmentation, erythema — dorsal half of the nasal planum common	Progresses to erosions, ulcers, crusts Atrophic scarring and dyspigmentation are chronic Nasal planum commonly the only area affected	Generally, large-breed dogs German shepherd and its crosses
PF	Pustule, purulent crust — dorsal aspect and bridge of nose	Progresses to healing depigmentation and loss of nasal planum architecture Other areas of both commonly affected	Middle-aged to older dogs Chow chow, shar-pei, and Akita are overrepresented
MCLE	Erosions and ulcers — symmetrical ± perinasal	Progresses to crusts, secondary depigmentation, and loss of nasal planum architecture > 1 perimucosal region often affected	German shepherd
AISBD	Erosions and ulcers (blisters) — no clear distribution, multifocal	Progresses to crusts (hemorrhagic), healing depigmentation, and loss of nasal planum architecture > 1 region often affected	German shepherd (MMP)
Alar fold arteriopathy	Linear ulcer/fissure — rostrolateral alar folds	Crust (hemorrhagic), healing (secondary) depigmentation	German shepherd
Dermal arteritis of the nasal philtrum	Deep, V-shaped ulcer — center of the nasal philtrum	Progresses to crust (hemorrhagic), healing (secondary) depigmentation, and loss of nasal planum architecture	Saint Bernard
Cutaneous histiocytosis	Nodule, swelling — no predilection site	May develop crusts, erosion/ulcers, loss of nasal planum architecture on nodule/swelling Lesions are commonly multifocal Systemic histiocytosis is described	Bernese mountain dog is predisposed to systemic histiocytosis
ECTL	Variable clinical phenotype — depigmentation may extend beyond border of nasal planum	Nasal planum seldom the only area affected	Older dogs
SCC	Nodule — usually asymmetrical, no known predilection site	May develop crusts, erosions/ulcers, draining tracts on nodule	Older dogs

HNPK — Hereditary nasal parakeratosis; INH — Idiopathic nasodigital hyperkeratosis; MCP — Mucocutaneous pyoderma; UDS — Uveodermatologic syndrome; DLE — Discoid lupus erythematosus; PF — Pemphigus foliaceus; MCLE — Mucocutaneous lupus erythematosus; AISBD — Autoimmune subepidermal blistering diseases; MMP — Mucous membrane pemphigoid; ECTL — Epitheliotropic cutaneous T-cell lymphoma; SCC — Squamous cell carcinoma.

lesions, except in specific instances such as certain dermal and subcutaneous mycoses.

Physical examination and cytology or culture are often inadequate for definitively diagnosing most nasal planum diseases, except for those with a distinct clinical phenotype, such as dermal arteritis of the nasal philtrum. Therefore, the authors recommend skin biopses for histopathological evaluation. Ideally, 2 punch biopsies (4 to 6 mm) of early lesions should be taken to capture the pathomechanistic role of the disease. Older lesions often lack primary histological changes of the underlying process and are more likely to have microbial overgrowth. For the best chance of a definitive diagnosis, it is imperative to sample fully affected skin rather than attempt to straddle the margin between normal and abnormal skin, especially if using a small biopsy punch. Similarly, sampling solely from an ulcer may not provide a conclusive answer due to the absence of epidermis. Key features for each disease are presented in Table 1.

Keratinization disorders

Hereditary nasal parakeratosis

Hereditary nasal parakeratosis is an autosomal recessive disorder of the *SUV39H2* gene in Labrador retriever and greyhound dogs that causes aberrant keratinocyte terminal differentiation of the nasal planum (1,2). Lesions usually appear in puppies and young dogs and consist of adherent scale, hyperkeratosis, and fissures on the dorsal ridge of the nasal planum, which may predispose to secondary infections (1,2). Affected dogs are otherwise healthy.

Idiopathic nasal or nasodigital hyperkeratosis

Idiopathic nasal or nasodigital hyperkeratosis causes proliferative hyperkeratosis on the dorsal ridge of the nasal planum, with or without paw pad involvement (3). Lesions are considered cosmetic and asymptomatic, but occasional deep fissuring can predispose to secondary *Malassezia* or bacterial infections. Overrepresented breeds include American cocker spaniel, English bulldog, miniature poodle, miniature schnauzer, and Doberman pinscher (3).

Infectious causes

Mucocutaneous pyoderma (MCP)

Mucocutaneous pyoderma is a bacterial dermatitis commonly affecting German shepherd dogs and their crosses (4). Early lesions are erythema, swelling, purulent exudate, and crusts, followed by ulcers, fissures, hypopigmentation/depigmentation, and loss of normal architecture. Predilection sites include the alar folds and perinasal areas. The nasal planum can exclusively be affected, but other mucocutaneous junctions, *e.g.,* lip margins, periocular region, and vulva, may be involved (4). Cytological examination of erosions or exudate typically reveals numerous bacteria and inflammatory cells. Mucocutaneous pyoderma responds to topical or systemic antibiotics (*e.g.,* cephalexin, 25 to 30 mg/kg, PO, q12h), empirically or based on culture and susceptibility testing. Mucocutaneous pyoderma shares clinical and histopathological features with discoid lupus erythematosus, but the latter has cytotoxic interface dermatitis that MCP lacks. As such, distinguishing between these 2 diseases often relies on clinical differentiation first involving an antibiotic trial.

Subcutaneous and cutaneous mycoses

Nasal planum lesions as the sole clinical sign is rare for most mycotic infections except cryptococcosis (5). *Cryptococcus neoformans* and *C. gattii* are dimorphic fungi present in pigeon feces, soil, and plant matter (5). These are considered primary fungal pathogens as they can cause infection in immunocompetent individuals through inhalation of blastospores from the environment (5). Clinically, cryptococcosis presents as deep nodules that form a characteristic "beak-shaped" bump encompassing the nasal planum and bridge of the nose. Ulceration and draining tracts may develop on the nodules. The condition is more common in cats than dogs (5). Yeast or spherules of *Cryptococcus* are typically numerous on cytological and histological evaluation (5).

Immune-mediated or autoimmune diseases

Vitiligo

Vitiligo is putatively an acquired autoimmune disease that targets melanocytes in the skin, resulting in depigmented macules and patches without affecting the nasal planum architecture (6) (Figure 1 A). Leukoderma and leukotrichia are commonly seen in other areas of the body. This is considered a cosmetic disease in dogs. The few reports of canine vitiligo report overrepresentation in rottweiler, Doberman pinscher, and collie dogs (6). Early signs are usually noticed in young dogs (median age: 24 mo) (6). The clinical features are normally sufficient for diagnosis, but skin biopsies of areas of early depigmentation for histopathological examination can provide a definitive diagnosis (6).

Uveodermatologic syndrome (VKH-like syndrome)

Akita, Samoyed, and Siberian husky dogs are believed to be predisposed to uveodermatologic syndrome, an immune-mediated disease that targets melanocytes or antigens in both skin and ocular tissues (6). The median age of onset is 3 y (6). Ocular disease (uveitis, vision loss) precedes dermatologic signs in most cases (6,7). Diffuse depigmentation of the entire nasal planum is the first cutaneous change, often with erythema and swelling, followed by erosions/ulcers, crusts, and alopecia of the surrounding haired skin (6). The nasal planum is most commonly affected but the periocular skin and lips are also common predilection sites (6). Skin biopsies for histopathological examination should be done on early, active inflammatory lesions (depigmentation, erythema, swelling) (6).

Facial discoid lupus erythematosus (DLE) and mucocutaneous lupus erythematosus (MCLE)

Facial discoid lupus erythematosus is the most common form of chronic cutaneous lupus erythematosus (8). The nasal planum is typically the sole predilection site, which helps distinguish DLE from other diseases. Diffuse depigmentation is usually the first sign, due to cytotoxic interface dermatitis resulting in pigmentary incontinence. Later, depigmented lesions progress to ulcers that form a thick layer of crusts, followed by atrophic

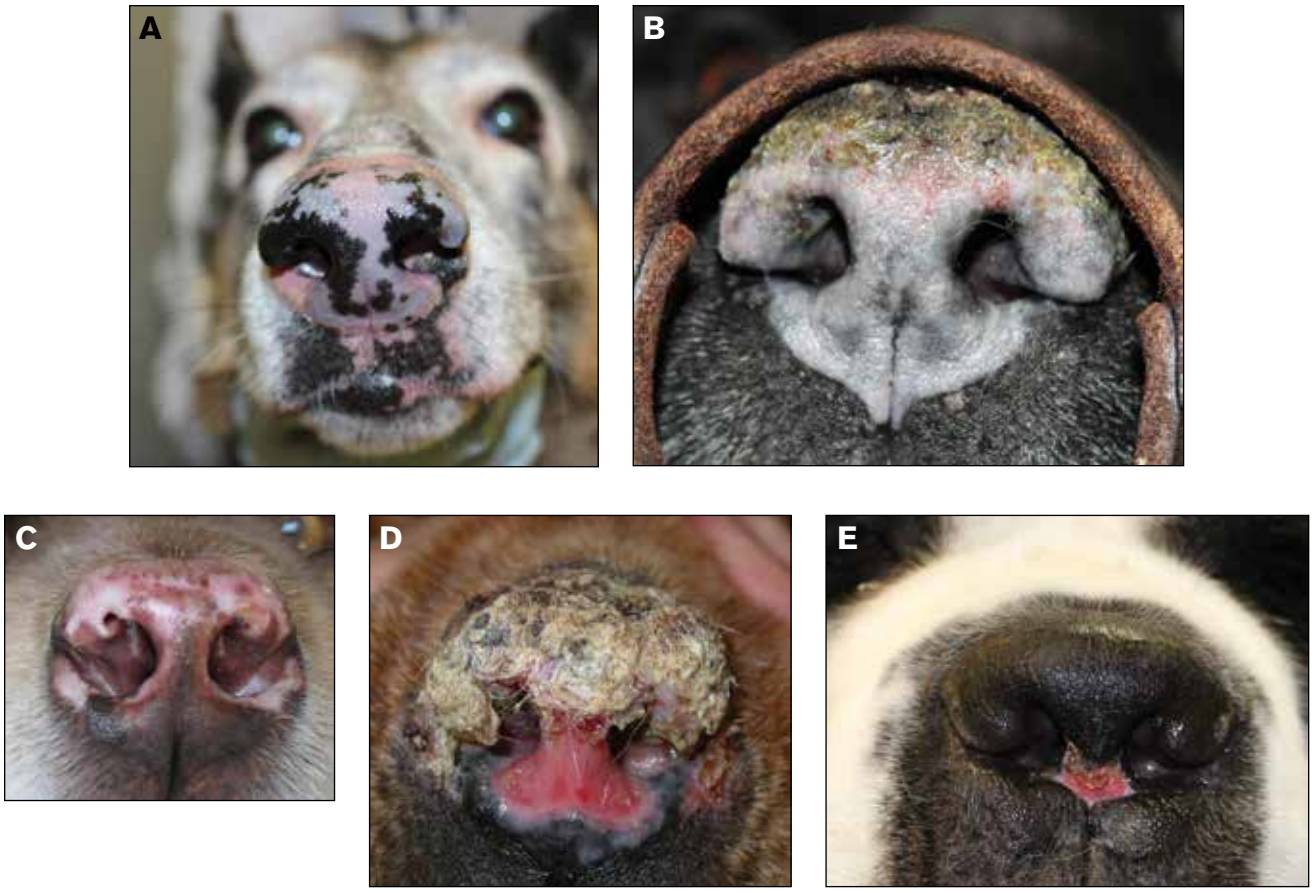

Figure 1. Clinical lesions of diseases that commonly target the nasal planum in dogs. A – Vitiligo in a German shepherd. Varying degrees of hypopigmentation to complete depigmentation are noted. In contrast to discoid lupus erythematosus (DLE), the normal "cobblestone" architecture is entirely intact. B – Early DLE with secondary infection in a Labrador retriever. Note the loss of pigment affecting the nasal planum. Architecture has not yet been entirely effaced, but a layer of crust has formed over the dorsal ridge, along with mild erosion. C – Chronic DLE in a mixed-breed dog. Depigmentation is well-established. There is almost complete loss of normal "cobblestone" appearance to the nasal planum and expansion of the nares due to prior destruction of the nasal tissues during the active phase of disease. D – Pemphigus foliaceus in a Labrador retriever cross. Note the heavy crusts on the nasal planum, with erosions and ulcers affecting the rostral aspect of the nose. E – Nasal arteritis in a Saint Bernard. Note the well-demarcated, wedge-shaped ulcer located directly over the nasal philtrum.

Photographs courtesy of Dr. Andrea Lam (A, B, D, and E) and Dr. Jennifer Schissler (C).

or hypertrophic scarring and further dyspigmentation in more chronic stages (8) (Figure 1 B, C). Facial DLE tends to affect middle-aged to older, large-breed dogs, with German shepherd dog and its crosses being overrepresented (8). A rare subtype that may or may not involve the nasal planum, generalized DLE, has been described (9). Mucocutaneous lupus erythematosus is another type of chronic cutaneous lupus erythematosus that also primarily affects middle-aged German shepherd dogs (10). Nasal lesions are not the predominant area affected, accounting for only 20% of cases in one study of 21 dogs (10). In addition, more than half of affected dogs exhibit involvement in $>$ 1 perimucosal site (10). Skin biopsies of areas of early depigmentation are recommended.

Pemphigus foliaceus (PF)

Pemphigus foliaceus is an autoimmune, superficial pustular dermatosis that classically targets the dorsal aspect of the nasal planum, bridge of the nose, pinnae, periocular area, and foot pads (11). However, a truncal variant of PF may spare the nasal planum. Many dog breeds have been reported to develop PF, but the chow chow, shar-pei, and Akita are overrepresented (11,12). This condition usually occurs in middle-aged to older dogs, though dogs of any age may be affected. Intact pustules are fragile and therefore transient, and rarely appreciated grossly, especially on the nasal planum. It is more common to observe crusts with underlying purulent exudate and erosions (Figure 1 D). Cytological examination of intact pustules or purulent exudate underneath a crust may reveal acantholytic keratinocytes, suggestive of PF. However, as staphylococcal pyoderma and pustular dermatophytosis can also cause acantholysis, it is important to rule out both types of infection first. Skin biopsies for histopathology are highly recommended to definitively diagnose PF once infectious etiologies have been ruled out, as treatment involves immunosuppression. Punch biopsies (4 to 6 mm)

should encompass intact pustules (when present) or adherent crusts. To optimize preservation of lesions and improve chances of definitive diagnosis, refrain from scrubbing skin or clipping hair before biopsy recovery.

Autoimmune subepidermal blistering diseases (AISBD)

Autoimmune subepidermal blistering diseases are a rare group of diseases driven by autoantibody-mediated disruption of proteins involved in maintaining structural integrity of the basement membrane zone, resulting in vesicle (blister) formation. Autoimmune subepidermal blistering diseases that commonly affect the nasal planum include mucous membrane pemphigoid, bullous pemphigoid, and junctional epidermolysis acquisita (13). The primary lesion is a vesicle, which is fragile and transient, quickly evolving into erosions and ulcers. Hemorrhagic crusts can form over ulcers, but purulent crusting is not a feature. Other body sites are almost always affected. The German shepherd dog is overrepresented for mucous membrane pemphigoid (14). The authors suggest using an appropriately sized punch biopsy or performing a wedge biopsy to extract an intact vesicle whenever feasible. Alternatively, sampling the inflamed edge of a ruptured vesicle for histopathological examination is recommended for definitive diagnosis.

Nasal alar arteriopathy

Nasal alar arteriopathy is a vasculopathy observed primarily in middle-aged German shepherd dogs, characterized by a linear ulcer/fissure on the rostrolateral alar fold (15). Its etiology is unknown but it is suspected to be an immune-mediated vasculopathy with a strong breed predisposition. Profuse arteriolar bleeding can be seen and presents a challenge during tissue biopsy sampling. As a result, empirical treatment with topical and/or systemic immunomodulatory drugs can be considered.

Dermal arteritis of the nasal philtrum

Dermal arteritis of the nasal philtrum presents clinically as a distinct, deep, V-shaped or wedge-shaped ulcer on the nasal philtrum, most commonly affecting Saint Bernard dogs (Figure 1 E). Similar to nasal alar arteriopathy, the clinically distinct phenotype, strong breed predisposition, and challenge in performing biopsies often necessitate a clinical diagnosis and empirical treatment with immunomodulatory drugs.

Histiocytosis

Cutaneous reactive histiocytosis is an inflammatory, lymphohistiocytic, proliferative disorder of unknown etiology, likely involving antigen-driven immunoregulatory disruption (16). Nodules with or without ulcerations on the nasal planum and other areas of the body are common and may wax and wane. Multiple nodules are more common than solitary lesions (16). Systemic reactive histiocytosis is a generalized histiocytic disorder with a known familial occurrence in the Bernese mountain dog (16). Cutaneous nodules in both cutaneous and systemic forms are similar, and hence diagnostic imaging and sampling of internal viscera are warranted to differentiate them. Diagnosis involves deep-tissue biopsies of nodules for cultures to exclude infectious causes and for histopathological evaluation.

Neoplasia

Cutaneous epitheliotropic T-cell lymphoma

The clinical presentation of cutaneous epitheliotropic T-cell lymphoma is highly diverse. Older dogs are predisposed (17). The nasal planum is commonly affected (~28%) but seldom the only location involved (17). Due to the phenotypic variability, initial lesions may include depigmentation, erythema, loss of architecture, crusts, and plaques. Interestingly, depigmentation may not follow a symmetrical pattern, and may extend beyond the border of the nasal planum to cause hypopigmentation/depigmentation of the rostral muzzle, lips, and in some cases, mucosa. Impression smear cytology of affected sites may reveal numerous lymphocytes, but the authors recommend skin-punch biopsies to demonstrate epitheliotropism.

Squamous cell carcinoma

Squamous cell carcinomas make up 5% of cutaneous tumors in dogs and usually appear as a single nodule on the nose that may ulcerate and bleed (18). Older dogs are more commonly affected. Punch or excisional biopsy or debulking surgery are recommended.

Conclusion

Many diseases can cause lesions of the nasal planum in dogs, but very few affect the nasal planum only, without involving other areas of the body. The initial lesion type and location within the nasal planum are useful clinical clues that help highlight unique features of these diseases. Cytological evaluation of lesions is recommended as an initial diagnostic step to rule out secondary infections; however, most cases warrant histopathological evaluation to reach a definitive diagnosis.

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