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CONTENTS

MESSAGE FROM THE EDITOR
C Mansfield

What’s your diagnosis
Beck C

AROUND THE JOURNALS

PAPER
Vascular transformation of a serosal lymph node in a cat
Wyatt SR, King JB

COMMENTARY
Review of CBD for treatment of OA in dogs

RETROSPECTIVE STUDY
Adverse events associated with cerebellomedullary cistern cerebrospinal fluid collection in 150 dogs with suspected intracranial disease: a retrospective case series

WHAT’S YOUR DIAGNOSIS
The answer and some more questions

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Message from the Editor

I hope that you enjoy the first volume of 2023 for the Australian Veterinary Practitioner, the format and content is slightly different from previously. This is due to several factors, but mainly due to where the journal sees itself now and where it is planning to be in the next 2-5 years. As this is a journal for members of ASAV, it needs to be relevant and useful for many stakeholders. ASAV has tasked me as the new Editor to steer the journal towards being indexed on Medline, so that residents and others in training can have a journal where their submissions are approved for credentialling purposes. Additionally, we want to improve member value and make the journal clinically useful for Australian practitioners. So, now is the opportunity for you to let ASAV Executive know what you would like to see in this journal. Please send through your suggestions or ideas to: editor.avp@ava.com.au, we are keen to listen.

In this edition we have a mix of some learning material, reviews and original articles- taste of some different options. The process for me getting to this first edition has been a steep learning curve and I am thankful for the wonderful assistance of the AVA staff and ASAV Executive in helping me. I am also thankful for the hard work and groundwork that Professor Bruce Parry, as the previous scientific editor has put in. Again, without his expertise this journal would not even be possible.

I look forward to working with you all in the future and to seeing what exciting heights the AVP can reach.

Kind regards,

Professor Caroline Mansfield
What is your diagnosis?

Dr Cathy Beck, BVSc FANZCVSc

Case History

A 4-year-old female entire border collie was presented for investigation of lethargy and vomiting for 3 days with increasing frequency.

On physical examination, the dog had a heart rate of 160 beats/min (reference interval 70-120 beats/min), pale and tacky mucous membranes and a capillary refill time of > 2 sec. Respiratory rate was 28 breaths/min (reference interval 15-30 breaths/min) and the dog was normothermic. Cardiac and thoracic auscultation was normal, but there were reduced gut sounds. On abdominal palpation there was no pain, and no masses were identified. Rectal examination was normal, there were no faeces present.

Haematology:

• Mild neutrophilia (12.6 x 10^9/L, reference interval 3.0-11.6 x 10^9/L) with total WBC count 14.1 x 10^9/L (reference interval 5.5-16.8 x 10^9/L)
• Mild lymphopenia 0.8 x 10^9/L (reference interval 1.1-5.1 x 10^9/L)
• Mild thrombocytosis 106 x 10^9/L (reference interval 148-484 x 10^9/L)
• Rest within normal reference intervals

Serum biochemical analysis:

• Mild hypokalaemia 3.3 mmol/L (reference interval 3.5-5.8 mmol/L)
• Selective chloride loss (Na-Cl difference 49; reference interval 29-42)
• Mild metabolic alkalosis HCO₃⁻ 26 mmol/L (reference interval 18-25 mmol/L).

The dog received methadone (0.2 mg/kg IV) and orthogonal abdominal radiographs were performed (see images over page).
What's your diagnosis?
Describe the radiographic findings on the next page. What’s your diagnosis?
Around the journals...

This section aims to highlight some open access papers that have been published in major veterinary journals in the last 6 months of 2022, with a brief summary and link.

Enjoy!

Evaluation of a long-acting injectable formulation of omeprazole in healthy dogs (wiley.com)

This paper, out of Tennessee, evaluates an IM injectable omeprazole product (developed in Australia for horses) and measured gastric pH using a capsule device in healthy dogs. The goal for acid reduction in dogs is poorly defined, but in people, maintenance of gastric pH at set levels for certain percentages of the day promotes healing of duodenal ulcers and gastro-oesophageal reflux. One injection (4 mg/kg IM) of this new product met this goal in most study dogs; with the pH being $\geq 3$ for 75% of the time for a mean of 5.5 days, and $\geq 4$ for 67% of the day for a mean of 5.25 days. The onset of action was quick (within 98 minutes). There were no adverse effects.

Take home message: This new product may be a suitable alternative for acute settings where GI bleeding is suspected/confirmed.

Ultra-long-acting recombinant insulin for the treatment of diabetes mellitus in dogs (wiley.com)

This pilot study out of Davis, California assessed 5 dogs with pre-existing diabetes mellitus that had been treated with a standard insulin regime every 12 hours to good clinical effect. The rationale for the study is that as this new insulin has an ultra-long plasma half-life [it is protected from degradation due to fusion with an immunoglobulin-fragment-crystallisable (Fc)], fewer injections will be required for management. The study authors transitioned dogs from the old regime to a prototype of the new insulin formulation (which is injected once weekly), adjusting the dose according to mean interstitial glucose. No adverse events or changes in clinical signs were documented in any dog. However, one of the five dogs required supplementation with lente insulin to maintain glycaemic control.

Take home message: This was a pilot study, but there was some data to suggest that good response to the insulin may not be maintained longer term in all dogs. The addition of meal-time short acting insulin may be necessary in some dogs to optimise glycaemic control using this product. Additionally, it is unknown how effective this insulin will be in poorly controlled diabetic dogs. However, more predictable absorption and therefore few dose adjustments (along with the need for fewer injections) make this an exciting area to keep an eye on.
Relationship between hearing, cognitive function, and quality of life in aging companion dogs (wiley.com)

There have been a lot of interesting developments in the field of cognitive dysfunction in dogs recently, mainly focused on recognition and treatment (see papers regarding ketogenic diets +/- medium chain triglyceride supplementation). This paper, however, looks at whether a physical impairment will potentially affect the way owners perceive their dog's cognitive ability and their dog's quality of life. Unsurprisingly, owner assessment of quality of life was significantly associated with hearing loss. This is likely due to a reduction in the quality of interaction between pets and their owners. Despite this, the dogs in the study all responded well to pointing cues and non-auditory social interactions, suggesting a good sense of vitality. More interestingly, the authors suggest that presbycusis (age-related hearing loss) may be a risk factor for canine cognitive dysfunction syndrome (however this may be difficult to prove, as much of the assessment criteria for CDDS rely on hearing).

Effect of standard-dose and high-dose pimobendan on select indices of renal and cardiac function in dogs with American College of Veterinary Internal Medicine stage B2 myxomatous mitral valve disease (wiley.com)

In this study, 30 dogs with stage B2 mitral valve disease were given either placebo (n=6), standard dose pimobendan (0.2-0.3 mg/kg q 12 hours) or high-dose pimobendan (0.5-0.6 mg/kg q 12 hours). At baseline, 7 and 10 days echocardiographic values, glomerular filtration (as determined by iohexol clearance), quality of life assessment and a cardiac biomarker (NT-proBNP) were measured. There were no significant differences in GFR or QoL between groups at any time point. Both pimobendane-treated groups showed improved echocardiographic parameters and reduction in NT-proBNP.

**Take-home message:** In dogs without pre-existing renal issues, pimobendan does not appear to affect GFR. In dogs with stage 2b MMVD, there is no short-term clinical advantage in administering high-dose pimobendan over standard doses.

Anticoagulant effects of rivaroxaban, prednisone, alone and in combination, in healthy dogs (wiley.com)

This randomised cross-over study (from Mississippi State) assessed 9 healthy colony dogs. Dogs were either given prednisone (2 mg/kg PO q 24 hours), rivaroxaban (1.5 mg/kg PO q 24 hours) or both for 8-day periods with a 21-day washout period between dosing. Coagulation status was assessed using prothrombin time and anti-Xa activity.

**Take home message:** The concurrent use of prednisone did not appear to impact the anti-coagulant effects of rivaroxaban. Prothrombin time may be used as a surrogate marker for anti-Xa activity.
ACVIM consensus statement on diagnosis and management of acute canine thoracolumbar intervertebral disc extrusion (wiley.com)

This is a useful reference for decision making and treatment recommendations for management of TL disc disease in dogs.

Comparison of biochemical and hematologic values obtained via jugular venipuncture and peripheral intravenous catheters in dogs (wiley.com)

Obtaining blood from IV catheters is a desirable technical procedure in hospitalised dogs to try to minimise venipuncture. This study assessed paired samples in 61 client-owned dogs obtained from the jugular vein and cephalic IV catheter. Overall, most parameters showed no difference between the two collection sites. However, AST, bilirubin, potassium, bicarbonate and haematology indices showed the most discrepancy.

Effect of oral or injectable supplementation with cobalamin in dogs with hypocobalaminemia caused by chronic enteropathy or exocrine pancreatic insufficiency (wiley.com)

In this study from the GI lab at Texas A&M, dogs with documented hypocobalaminaemia (due to either chronic enteropathy or exocrine pancreatic insufficiency) were given either an oral or injectable course of cobalamin.

The following dosage was administered:

<table>
<thead>
<tr>
<th>BW &lt; 4.5 kg</th>
<th>BW 4.5-10 kg</th>
<th>BW 10-15 kg</th>
<th>BW 16-20 kg</th>
<th>BW 20-35 kg</th>
<th>BW 35-44 kg</th>
<th>BW ≥ 45 kg</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Oral</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>treatment</td>
<td>250 µg</td>
<td>250 µg</td>
<td>500 µg</td>
<td>500 µg</td>
<td>1000 µg</td>
<td>1000 µg</td>
</tr>
<tr>
<td>(dosage/day)</td>
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<td></td>
<td></td>
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<td></td>
</tr>
<tr>
<td><strong>Injectable</strong></td>
<td>250 µg</td>
<td>400 µg</td>
<td>600 µg</td>
<td>800 µg</td>
<td>1000 µg</td>
<td>1200 µg</td>
</tr>
<tr>
<td>(dosage/week)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>1500 µg</td>
</tr>
</tbody>
</table>

There was no difference in serum cobalamin concentration (or methylmalonic acid concentration) between either form of supplementation.

**Take-home message:** oral supplementation (even with concurrent GI disease) may be a suitable alternative to weekly injections. Dosages should be adjusted according to body weight.

2022 AAFP/EveryCat Feline Infectious Peritonitis Diagnosis Guidelines - Vicki Thayer, Susan Gogolski, Sandra Felten, Katrin Hartmann, Melissa Kennedy, Glenn A Olah, 2022 (sagepub.com)

This paper has an extensive review of the diagnostic tips (and pitfalls) for diagnosing FIP in cats and is an excellent resource for veterinary practice. There is a focus on diagnosing the ‘dry’ form, as this is the most difficult to determine. There are some excellent ocular images as well.
2022 ISFM Consensus Guidelines on Management of the Inappetent Hospitalised Cat - Samantha Taylor, Daniel L Chan, Cecilia Villaverde, Linda Ryan, Franck Peron, Jessica Quimby, Carolyn O’Brien, Serge Chalhoub, 2022 (sagepub.com)

Another excellent open access resource for use in practices where cats may be hospitalised (albeit with a Northern hemisphere focus). The guidelines also have links to guides and videos to aid clients in tube feeding at home, as well as links for feeding tube placement and management for veterinary staff. These guidelines were developed by the International Society of Feline Medicine and have been endorsed by the American Association of Feline Practitioners.

Comparison of the effect of three intramuscular sedation protocols on packed cell volume and total protein in cats - Alicia M Skelding, Alexander Valverde, 2022 (sagepub.com)

This paper addresses a common problem in veterinary practice- obtaining samples from stressed or fractious cats in hospital. This project assessed the impact of three sedation protocols on basic blood parameters (PCV/TS, electrolytes, glucose and lactate).

Thirty cats (10 in each group) received one of the following protocols at standard IM doses:

- Methadone + acepromazine (MA)
- Methadone + dexmedetomidine (MD)
- Methadone + Midazolam + Alfaxalone (MMA)

Level of sedation was best in the MMA group, whilst performed least effectively in the MA group. At 25 minutes post sedation, PCV and haemoglobin was significantly decreased in all groups, total protein reduced in MA and MMA, and glucose decreased in MD group. Although there were some differences in sodium and chloride these weren’t of clinical significance.

Take-home message: Care should be taken in over interpreting low PCV and total protein results in samples obtained from cats sedated with one of the above protocols.


In this study, five healthy cats were given a single dose of gabapentin at 20 mg/kg PO and 25 cats with IRIS stage 2 (n=14) and 3 (n=11) CKD were given a dose of 10 mg/kg PO. The cats with CKD had higher serum gabapentin concentrations at 3 and 8 hours following administration; which was correlated with SDMA concentration. Despite this, there was no over sedation observed in any cat.

Take-home message: Consideration to dose reduction for gabapentin administration should be given for cats with CKD.
Evaluation of a nutritional supplement for the alleviation of pain associated with feline degenerative joint disease: a prospective, randomized, stratified, double-blind, placebo-controlled clinical trial - Rachael Cunningham, Margaret E Gruen, Andrea Thomson, B Duncan X Lascelles, 2022 (sagepub.com)

In this study, 59 cats with osteoarthritic pain were recruited. Thirty received a placebo, whilst 29 received a glucosamine/chondroitin sulfate supplement after 2 weeks of placebo. Response was assessed by at home accelerometry, client surveys and veterinary assessment. Both groups showed significant improvement in all aspects at all time points, with no differences in activity between the two groups. The cats receiving placebo had the most improvement in pain, quality of life and client-specific outcomes.

**Take-home message:** there is no evidence that this nutritional supplementation was effective. The strong placebo effect seen in this study should be considered when assessing other studies of osteoarthritis treatment.

Randomised clinical trial comparing the perioperative analgesic efficacy of oral tramadol and intramuscular tramadol in cats - Sébastien H Bauquier, 2022 (sagepub.com)

This was study performed in young cats undergoing ovariohysterectomy. Twelve cats received oral tramadol (6 mg/kg) 60 minutes prior to anaesthetic induction along with an IM placebo injection, whilst 12 cats received intramuscular tramadol (4 mg/kg) plus a placebo capsule 30 minutes prior to anaesthetic induction. Two cats receiving IM tramadol required additional analgesia post recovery, however overall, there was no difference between the two groups in pain scores post-operatively.

**Take-home message:** pre-operative oral tramadol appears to provide adequate analgesia for 6 hours post OVH surgery.

Risk factors for neonatal mortality prior to hospital discharge in brachycephalic and nonbrachycephalic dogs undergoing cesarean section (wiley.com)

This retrospective study came from Georgia, covering a nine-year period. They reviewed the records for a total of 480 puppies born from 90 bitches undergoing 106 caesarean sections. Overall neonate survival to discharge was 93.11% (447/480), with no difference in survival between brachycephalic and non-brachycephalic. However, it was found that puppies part of a large litter requiring caesarean had a survival rate compared to smaller litters (but not overall litter size).

**Take-home message:** In bitches at high risk of dystocia, elective caesareans should be considered to maximise puppy survival.
Preoperative skin asepsis protocols using chlorhexidine versus povidone-iodine in veterinary surgery: A systematic review and meta-analysis (wiley.com)

This study used literature review methods to assess the difference in skin prep protocols. Following PRISMA guidelines, the authors were only able to identify 9 appropriate papers in the veterinary literature. Formulation, concentration and application methods between these papers were not consistent. Either because of the scarcity of reports, or due to a true lack of difference, the authors were unable to find any significant difference in surgical site infection or skin bacterial colonisation between the use of chlorhexidine or povidone-iodine solutions.

**Take-home message:** As this is a vital part of daily veterinary practice, further controlled prospective studies are required in this area.

Advances in the pharmaceutical treatment options for canine osteoarthritis (wiley.com)

This Petsavers review paper is from a group based at the University of Liverpool. There is an obvious UK bias in terms of product availability, but the review provides an excellent synopsis of osteoarthritis pain pathways, standard anti-inflammatory medications, monoclonal antibodies, adjunctive analgesia and regenerative treatments. It is long, but comprehensive. One of the useful points is that in people, the use of monoclonal antibodies against nerve growth factor along with NSAIDs showed a rapid progression in OA, but this has not been investigated in dogs.

Identification of canine osteoarthritis using an owner-reported questionnaire and treatment monitoring using functional mobility tests (wiley.com)

Sticking with the osteoarthritis theme, this paper (with authors affiliated to Zoetis), report a questionnaire to detect early signs of OA in dogs. Dogs that were identified in the preliminary survey as possibly having OA (yes to at least one of the questions) were then eligible for further assessment and an OA treatment trial, with the questionnaire assessed to see if a valid monitoring tool.

Questions to be completed were simple yes/no questions and could easily be incorporated into general health check-ups:

1. Does your dog limp or appear stiff after exercise?
2. Do you think your dog shows signs of pain?
3. Is your dog reluctant to climb stairs or jump?
4. Does your dog have difficulty in rising from a resting position?
5. Have you noticed a change in your dog\'s behaviour?
6. Does your dog tire easily or lag behind during walks?
7. Has your dog ever been injured?
8. Have you ever given your dog medication for pain?
9. Has your dog gained weight in the last year?
Vascular transformation of a serosal lymph node in a cat

SR Wyatt*
Veterinary Specialist Services, Jindalee, Queensland, Australia
swyatt@vss.net.au

JB King
Veterinary Specialist Services, Jindalee, Queensland, Australia
jking@vss.net.au

Abstract

Background
Vascular transformation of lymph nodes, or plexiform vasculopathy, is an uncommon condition, which has been reported in inguinal and cervical lymph nodes in cats, but not before in a serosal location.

Case report
An eight-year-old male neutered Domestic Shorthair Cat presented with vomiting, hyporexia, and abdominal pain of 24-hour duration. A mass lesion at the ileocolic junction was identified on abdominal ultrasound. A vascular mass arising from the serosal surface of the caecum was present on exploratory laparotomy. Histopathology was consistent with plexiform vasculopathy. There was concern over malignant transformation of the lesion based on histopathology, however at 98 days following surgery there was no evidence of recurrence on abdominal imaging.

Conclusion
To the authors' knowledge this is the first described case of plexiform vasculopathy in a serosal lymph node. This should be considered as a differential for vascular gastrointestinal mass lesions in cats.

Keywords: cat; plexiform vasculopathy; lymph node

Authors: SR Wyatt¹, JB King²

¹Veterinary Specialist Services, Jindalee, Queensland Australia

Introduction
Vascular transformation of lymph nodes is an uncommon and poorly described condition in both human and veterinary medicine; it was first reported in humans in 1971¹,², and has since been reported in cats in cervical and inguinal lymph nodes³,⁴. In cats it is referred to as plexiform vasculopathy. Plexiform vasculopathy is considered a benign entity, with previous studies reporting excellent prognosis and lack of recurrence when lesions are fully excised. Malignant transformation and association with intranodal haemangiosarcoma has, however, been reported in a small number of cervical lymph nodes in one cat case series⁴. This was associated with a more guarded prognosis and survival times ranging from less than one
month to over 30 months. It has also been suggested that such lesions may transform to lymphangiosarcoma.

The pathogenesis of plexiform vasculopathy is still unclear, however, it is suspected to occur secondary to lymphatic or venous obstruction, for example due neoplasia or thrombosis. In an experimental study, it was found that complete occlusion of lymphatic flow, or partial occlusion of lymphatic and venous flow, lead to vascular sinus transformation in cervical lymph nodes in rabbits. In contrast, when venous drainage alone was occluded, vascular transformation did not develop.

Histopathological features of plexiform vasculopathy include vascular proliferation, with well-defined endothelial cells and a fibrous stroma. Vascular channels may be venous or lymphatic in origin, and a recent study documented markers of both vascular and lymphatic origin. There is concurrent loss or compression of adjacent lymphoid tissue, but the capsule of the lymph node appears unaffected. This case report describes a case of plexiform vasculopathy in a serosal lymph node located at the ileocaecocolic junction in a cat, with no known malignant association. To the authors' knowledge this is the first described case of such a lesion at this location.

**Case description**

An eight-year-old, male neutered domestic short hair cat presented to the emergency after-hours service with acute onset inappetence, vomiting and lethargy. There was no significant prior medical history. On examination, the animal was pyrexic (39.9°C) with moderate abdominal pain. In-house haematology, biochemistry and urinalysis were performed. Haematology showed a mild leucocytosis with neutrophilia. Biochemistry showed mild, non-specific changes with mildly low urea, mild hypophosphataemia and mild hyperglycaemia. Urinalysis showed isosthenuria, however the sample was collected after starting intravenous fluids. There was also mild glucosuria, likely associated with transient stress related hyperglycaemia.

An abdominal ultrasound was performed shortly after admission to hospital and was reportedly unremarkable. The cat was treated with methadone (0.1-0.3mg/kg intravenously [IV] every four hours), antiemetics (maropitant at 1mg/kg IV every 24 hours), and intravenous crystalloid fluid therapy. Due to persistent pyrexia and worsening abdominal pain in hospital, the ultrasound was repeated four hours after admission. This revealed a focally dilated loop of small intestine, with distal acoustic shadowing concerning for an intestinal foreign body.

Following referral, an abdominal ultrasound was repeated by a Small Animal Internal Medicine Specialist and mildly enlarged ileocolic lymph nodes measuring up to 6mm in width with normal echogenicity were present. There was a 3.0 x 2.3cm heterogenous mass immediately adjacent to the serosal surface of a section of small intestine (see Figure 1). It was unclear if the mass was adjacent to, or arising from, the serosal surface. The surrounding mesenteric fat was hyperechoic and there was a small volume of free abdominal fluid. There were no other significant ultrasonographic findings. Due to the small volume of fluid, samples were unable to be obtained. After discussion with the owner, it was elected to proceed directly to exploratory laparotomy rather than performing fine needle aspirates or thoracic imaging.
At surgery, the vascular mass was found to be arising from the serosal surface of the caecum. The associated lymph node was mildly enlarged and dark red in colour. There were no other gross changes noted in the abdomen. An ileocaecocolic resection and anastomosis was performed, and the affected intestine and associated lymph node were removed en bloc.

Post-operatively, the cat was treated with ongoing maropitant, and analgesia. Parenteral cobalamin supplementation was commenced (0.5mg cyanocobalamin subcutaneously [SQ]) along with mirtazapine as an appetite stimulant (2mg applied as transdermal cream once daily). At day 2, due to concerns regarding prolonged anorexia, an oesophagostomy tube was placed under general anaesthesia. Feeding was commenced later that day as standard for oesophagostomy tubes. The cat was discharged Day 3 post operatively, with ongoing maropitant (2mg/kg orally once daily), mirtazapine (2mg applied transdermally once daily), and buprenorphine (0.015mg/kg transmucosally eight hourly).

Histopathology results confirmed the intestinal mass to be a serosal lymph node with evidence of atypical vascular proliferation. There were variably sized and anastomosing vascular spaces which contained blood and were lined by spindle cells. Spindle cells exhibited pleomorphism, nucleolar prominence and increased nuclear size, with occasional mitotic figures which raised concern for possible malignant transformation. The vascular proliferation did not extend to the examined sections of gastrointestinal tract. The associated lymph node showed reactive lymphoid hyperplasia, suppurative lymphadenitis and haemorrhage. Findings were considered similar to previously described reports of plexiform vasculopathy. The terminal ileum associated with the mass revealed mild increase in lamina propria lymphocytes and plasma cells consistent with mild lymphoplasmacytic enteritis. There were no significant lesions of the colon or caecum.

At day 6 post-operatively, the cat presented for review and was bright and alert, normothermic, with no abdominal discomfort. Weekly cobalamin injections were continued,
as per standard protocol as laboratory results confirmed low serum cobalamin (205pmol/L, reference range 214-1107pmol/L).

At day 15 post-operatively, a repeat ultrasound assessment identified mild mesenteric lymphadenomegaly (measuring up to 6.4mm), with hyperechoic mesentry and a small volume of free abdominal fluid in the region of the anastomosis. By day 22 post-operatively, follow-up abdominal ultrasound identified resolution of free abdominal fluid and normal mesenteric lymph nodes.

At follow up 98 days post-operatively, the cat was reportedly doing well with no ongoing gastrointestinal signs. The cat was subsequently lost to follow up.

**Discussion**

This is, to the authors’ knowledge, the first report of vascular transformation of a serosal, intra-abdominal lymph node in a cat. Vascular transformation of lymph nodes is generally considered a benign condition, but in this case, there were concerns over the potential for malignant transformation in this mass lesion (subsequently identified as lymph node). It has recently been suggested that malignant transformation may occur, and that nodal haemangiosarcoma or lymphangiosarcoma may develop. In this case, given the lack of recurrent lesions identified on ultrasound, the histopathological changes are thought most likely to be benign in nature.

Immunohistochemistry (IHC) can be performed on samples to determine lymphatic or vascular origin of cells but does not differentiate benign from malignant lesions. Markers that have previously been used include cluster of differentiation 31 (CD31), erythroblast transformation specific regulated gene-1 (ERG for both vascular and lymphatic endothelium), cluster of differentiation 31 (CD34, for vascular endothelium) and podoplanin (D2-40, for lymphatic endothelium). In this case, the cells were identified as of vascular rather than lymphatic origin on plain H&E stain, and as such IHC was not performed.

It is unclear why the cat had severe abdominal signs given the extraluminal location of the mass and lack of obvious peritonitis. At the time of surgery, no other gross abnormalities were detected however full gastrointestinal biopsies were unfortunately not obtained. The mild lymphoplasmacytic change noted in the ileum may reflect underlying inflammatory bowel disease, with acute exacerbation of a more chronic process however further diagnostics including IHC would ultimately be required to exclude small cell lymphoma. This cat had no prior history of gastrointestinal (GI) disease and had not developed signs following recovery from surgery until lost to follow-up. It is possible, however, that there were signs that were not perceived by the owner.

Hypocobalaminemia may support pre-existing GI disease and has been reported in 16.5%-61% of cats with gastrointestinal disease. The cause of hypocobalaminemia in this case may be related to mild lymphoplasmacytic enteritis. However, the detected intestinal inflammatory changes may have occurred secondary to the abnormal vascular proliferation in the draining lymph node. While the inflammatory changes in this case were only mild in nature, no association between severity of histopathological change or the duration of clinical signs and cobalamin concentrations has been reported in cats.

One limitation in this study is the lack of biopsy samples from the remainder of the GI tract and pancreas, along with no thoracic imaging. Given the acute nature of his signs, and the sonographic appearance of the mass, a neoplastic cause was suspected and thus collection of further samples was not deemed
necessary at the time of surgery. Obtaining further biopsies would have allowed a more thorough evaluation for inflammatory bowel disease or small cell lymphoma, which may have contributed to the lymph node changes.

Malignant transformation was suspected based on histopathology results, however, was not confirmed. Given the lack of recurrence on repeat imaging and the lack of clinical signs following surgery, a malignant process is considered less likely. Ongoing sonographic monitoring was recommended in this case, to detect evidence of recurrence.

**Conclusion**

In conclusion, this case study reports abnormal vascular transformation of a serosal ileocolic lymph node, resulting in acute abdominal signs in a cat. While an uncommon histopathological diagnosis, it should be considered as a possible cause of vascular intestinal masses in cats. Importantly, this represents a benign condition which can be cured with appropriate surgical margins.

**Acknowledgements**

We thank Dr Louise Sullivan at QML Vetnostics (Murrarie, QLD) for her assistance in interpretation of histopathology results.

**Conflict of interest statement**

The authors declare no conflicts of interest or sources of funding for work presented here.

**References**


Cannabinoid pain-relieving effects are linked to various interactions and modulation of the endocannabinoid, inflammatory and nociceptive systems (Vuckovic et al., 2018), with cannabidiol presenting high affinity for cannabinoid CB1 and CB2 receptors (antagonist), G-protein-coupled receptor 55 (antagonist) and many TRPV receptors (agonist) as well as peroxisome proliferator-activated receptor γ (Peng et al., 2022), the latter two being largely recognised for their role in chronic pain and OA-related joint degradation (Fahmi et al., 2011). Cannabinoids have shown promise in animal models with acute and chronic pain (Malan et al., 2003; Manzanares et al., 2006; Starowicz et al., 2013). A recent systematic review of cannabidiol in dogs indicated high-quality studies and good evidence of efficacy as part of a multi-modal treatment plan (Barbeau-Grégoire et al. 2022). Several clinical trials focussed on dogs have evaluated the safety and efficacy of CBD for the treatment of pain, discomfort, or improvement in mobility. These show conflicting results and the need for larger, multi centre studies.

Summary of recent studies

- **Canine Osteoarthritis Safety and Efficacy Study:** The objective of this study by Mejia et al., in 2022, was to provide preliminary data describing the safety and effect of cannabidiol (CBD) for symptom relief of canine osteoarthritis-associated pain in a clinical setting using objective outcome measures. Twenty-three client-owned dogs with naturally occurring OA completed this prospective, double-blinded, crossover, placebo-controlled study. Baseline data were acquired for 4 weeks, followed by random allocation to either placebo or CBD treatment for 6 weeks, followed by 6 weeks with the opposite treatment. Outcome measures included objective gait analysis, activity counts (via accelerometry) and clinical metrology instruments. There were no differences noted between groups at any time point for any of the recorded outcome measures. Adverse events associated with CBD administration included elevation in liver enzymes (n = 14) and vomiting (n = 2).

  The pilot data from this study do not support the use of CBD for canine OA.

- **Canine Osteoarthritis Efficacy Study:** A study by Kogan et al., in 2020, 32 dogs with OA completing a 90-day trial
Commentary

with no placebo control group. CBD dose escalations of 0.5 to 0.75 mg/kg approximately every 12 hours were prescribed at each reassessment until the dog’s pain score on palpation was zero to one, with zero indicating the least pain and ten indicating the greatest pain. In this study, dogs could receive concurrent gabapentin, but not NSAIDs, and concurrent acupuncture or electroacupuncture, nutraceuticals, polysulfated glycosaminoglycan, or a combination thereof. Physical examination by veterinarians and owner assessments were done every two weeks using the Cincinnati Orthopaedic Disability Index. This study reports that two of the 32 dogs did not respond during the 90-day study, with their overall pain scores remaining at one out of ten. CBD treatment appeared to be effective within the CBD dosing range of 0.3 to 4.12 mg/kg every 12 hours. Most of the dogs enrolled required 1 to 2 mg/kg of CBD-rich product to achieve a palpation score of zero to 1; 94% of owners felt the treatment positively impacted the dog’s quality of life. Of the 23 dogs that were also on gabapentin for pain mitigation, use was completely discontinued for ten dogs and daily dose was reduced for 11 dogs. With respect to CBC and serum biochemical analyses prior to and after the trial, only serum ALP concentrations showed a significant increase. Neither the owners nor veterinarians reported evidence of adverse effects from the treatment. Owners felt their dogs slept less and interacted more with family while on the CBD-rich product.

- **Canine Osteoarthritis Efficacy Study:** A study by Verrico et al., in 2020, investigated the therapeutic potential of CBD for OA, using both CBD in coconut oil and liposomal-encapsulated CBD in a four-week, randomized, placebo-controlled, double-blinded study in a spontaneous canine model of osteoarthritis. This pilot study included 20 dogs divided into four groups. They were given placebo (coconut oil), low dose (0.5 mg/kg/ day) CBD, or higher-dose (1.2 mg/kg/ day) CBD in coconut oil, or 0.5 mg/kg/ day of liposomal-encapsulated CBD. Before study initiation and at day 30, and using a five-point scale, a veterinarian assessed walking, running, and assuming a standing position from both a sitting and lying down position. Owners also evaluated dogs before treatment, at week four of treatment and then again at week six (two weeks after discontinuing the treatment) using the Helsinki Chronic Pain Index. Veterinary examinations and owner evaluations indicated no significant difference in response between dogs given a placebo or low dose (0.5 mg/kg/ day) CBD, but significant improvement was noted for all sitting to standing and lying to standing transitions for dogs that received the higher dose (1.2 mg/kg/ day) CBD and the liposomal-encapsulated CBD (0.5 mg/kg/day). Administration of the higher dose CBD or the liposome-encapsulated CBD was associated with significant improvements to quality of life as quantitated by both owner and veterinarian.

- **Canine Pharmacokinetics & Osteoarthritis Efficacy Study:** A study by Gamble et al., in 2018, determine the pharmacokinetics of a full spectrum CBD dominant, hemp-based oil infused formula in dogs. The elimination half-life was 4.2 hours at both the 2mg/kg and 8 mg/kg dose, with no observable side effects. Subsequently, a randomized, placebo-controlled, double blinded, cross-over study was conducted using the same formula at 2 mg/kg q12 hours for 4 weeks. Overall, assessments of pain relief by owners and veterinarians were positive. The results demonstrated positive efficacy for pain associated with osteoarthritis with no adverse side effects, although 9 out
of 16 dogs did develop increases in ALP values on serum chemistry analysis.

- **Efficacy of CBD as part of multimodal analgesia in canine osteoarthritis:**

  According to a study by Brioschi et al., 2020, pet owners reported (using the Canine Brief Pain Inventory) satisfactory improvements in pain management and quality of life in dogs receiving CBD oil (2 mg/kg every 12 h), as part of a multimodal pharmacological approach for the treatment of OA-related pain. Combined with a non-steroidal anti-inflammatory drug, gabapentin and amitriptyline, CBD appears to enhance osteoarthritic pain relief and quality of life improvement. Furthermore, its co-administration resulted in dose reduction of other administered drugs, minimizing the severity and incidence of associated side effects. The high tolerability, and the lack of adverse effects of oral CBD may represent potential benefits for long-term therapy.

Recent findings provide promising evidence to support the use of CBD for the treatment of OA related pain in dogs as part of a multimodal approach to chronic progressive disease. In dogs with OA related pain, efficacy was most frequently found between 1mg – 2mg/kg when administered orally every 12-hours. Further studies are warranted in other domestic species where evidence is lacking.

**References:**


Adverse events associated with cerebellomedullary cistern cerebrospinal fluid collection in 150 dogs with suspected intracranial disease: a retrospective case series.

A Storrera,b*, G Childc, TS Barnesd, S Waltons, T Kinga, BM MacKaya CM Chanaf

* Corresponding author’s present address and email: Queensland Veterinary Specialists, 53 Flinders Parade, North Lakes 4509, Brisbane, Queensland, Australia Anjie.storrer@qldvetspecialists.com.au

a Veterinary Specialist Services, Corner of Lexington and Logan Roads, Underwood 4119, Queensland, Australia

b Queensland Veterinary Specialists, 53 Flinders Parade, North Lakes 4509, Brisbane, Queensland, Australia

c Small Animal Specialist Hospital, Level 1, 1 Richardson Place, North Ryde 2113, New South Wales, Australia

d The University of Queensland, School of Veterinary Science, Gatton 4343, Queensland, Australia

e University of Florida Small Animal Hospital, 2015 SW 16th Avenue, Gainesville, Fl, 32608, USA

f The Pet Oncologist, 1A Norman Street, Fig Tree Pocket 4069, Brisbane, Queensland, Australia

Abstract

Objective: To document complications when performing cerebellomedullary cistern cerebrospinal fluid collection in dogs with clinical signs indicative of intracranial disease, estimate the frequency of these complications and identify risk factors associated with their occurrence.

Design: Retrospective case series.

Procedure: Medical records were reviewed for 150 dogs with clinical signs indicative of intracranial disease undergoing cerebellomedullary cistern cerebrospinal fluid collection. Signalment, neurological signs, anaesthesia information, cerebrospinal fluid analysis, adverse events, and diagnosis were recorded. Adverse events were classified based on clinically relevant complications and logistic regression was used to identify risk factors associated with these complications.

Results: Clinically relevant complications occurred in 10.0% of cases with the most common complication recorded as deterioration in neurological status following spinal fluid collection. Risk factors found to be associated with the development of complications were rapid progression of neurological signs, severe forebrain or caudal fossa disease, multifocal/diffuse central nervous system disease or diagnosis of pleocytosis.

Conclusion: Cerebellomedullary cistern cerebrospinal fluid collection is associated with a low risk of complication. An increased risk of complications was seen in dogs presenting with signs indicative of severe forebrain disease, caudal fossa disease, multifocal central nervous system disease or a rapidly deteriorating clinical course on presentation.

b Idexx Laboratories Pty Ltd
c QML Pathology Pty Ltd
d The University of Queensland School of Veterinary Science Department of Clinical Pathology;
e Stata®, Version 10. StataCorp, College Station, Texas USA.
**Keywords:** Canine, central nervous system, cerebrospinal fluid, neurology

**Abbreviations:** ANKC, Australian National Kennel Council; CNS, central nervous system; CMC, cerebellomedullary cistern; CMCCSF, cerebellomedullary cistern cerebrospinal fluid; CSF, cerebrospinal fluid; CRI, continuous rate infusion; CT, computed tomography; ICP, intracranial pressure; MRI, magnetic resonance imaging; MUO, meningitis of unknown origin; RBC, red blood cell; TNCC, total nucleated cell count; TP, total protein.

Cerebellomedullary cistern cerebrospinal fluid (CMCCSF) collection is an ancillary diagnostic test used in animals showing signs referable to central nervous system (CNS) or meningeal disease.\(^1\)\(^2\) Cerebrospinal fluid (CSF) analysis is commonly used to aid in categorising the underlying disease process and is most useful in helping to investigate possible inflammatory or infectious CNS diseases.\(^2\) Collection requires general anaesthesia and an aseptic technique, with fluid routinely collected from either the cerebellomedullary cistern (CMC) or lumbar subarachnoid space.\(^1\)\(^3\)\(^4\) While collection from the CMC carries a lower risk of blood contamination, it is considered to be associated with more severe potential complications.\(^5\)\(^7\) CMCCSF collection can be rapid and simple to perform. However, care must be taken to avoid injuring surrounding neural structures.\(^3\)\(^4\)\(^6\) Direct spinal cord trauma, brainstem trauma, CNS haemorrhage, herniation of cerebellum through the foramen magnum, and herniation of occipital lobe caudally through the tentorial notch,\(^1\)\(^3\)\(^4\)\(^6\) are recognised potential complications of performing CMCCSF collection.\(^3\)\(^5\)\(^8\)\(^9\)

Reports of adverse events after CMCCSF collection are lacking in the current veterinary literature and most of the information is based on human literature, case reports\(^5\) or is anecdotal.\(^3\) Generally, caution is advocated when performing CMCCSF collection in dogs displaying signs suggestive of elevated intracranial pressure (ICP), including dogs that present with heart rate and blood pressure parameters consistent with the Cushing reflex, severe neurological disease, decreasing level of consciousness, severely altered mentation, evidence of a large space occupying mass on neurocranial imaging, or suspected diffuse inflammatory CNS disease.\(^1\)\(^10\)\(^11\) These animals are believed to be at increased risk of brain herniation following CMCCSF collection due to elevated ICP.\(^5\)\(^11\) In addition, CMCCSF collection in smaller dogs is often considered to carry increased risk due to the large size of the needle compared to the relatively smaller area of the CMC.\(^12\) In theory, certain conditions such as Chiari like malformation or other cranio-cervical junction malformations may be considered to increase risk of CMCCSF sampling due to altered anatomy, such as risk of caudal displacement of the cerebellum, although this has not been demonstrated\(^9\)\(^11\).

Another proposed factor that may increase risk of complications is longer anaesthetic time. Prolonged anaesthesia has been associated with changes in cerebral perfusion which could potentially result in further deterioration of neurological status post CSF collection.\(^10\)\(^11\)

The aims of this retrospective study were to (i) describe the complications occurring when performing CMCCSF collection in dogs with clinical signs suggestive of intracranial disease in a referral practice setting, (ii) estimate the frequency of these complications, and (iii) identify risk factors associated with their occurrence.

**Materials and methods**

The medical record database for Veterinary
Specialist Services, Underwood, Queensland, Australia was searched to identify dogs that underwent CMCCSF collection between July 2008 and July 2010. Dogs were included in the study if they underwent a physical and neurological examination, had initial clinical signs or history suggestive of intracranial disease, had haematological and biochemistry tests performed with results not suggestive of extracranial disease, and a CMCCSF sample collected and submitted for cytological and biochemical analyses. Dogs were subsequently excluded from the study if myelography was performed, or if the dog was subsequently diagnosed with spinal cord disease. For each eligible animal, information extracted from the medical records included age (<3 years, 3-8 years or >8 years) and breed (categorised according to Australian National Kennel Council [ANKC] classification system) (group 1: toy, group 2: terrier, group 3: non-sporting or group 4: other), sex, body weight (<6kg, 6-20kg or >20kg).

Also recorded were duration of clinical signs (<48 hours, 48 hours to 1 week, 1 week to 1 month or >1 month), progression of neurological signs (static [non-progressive], slowly progressive [signs progressive over weeks to months] or rapidly progressive [signs progressive on a daily basis]), signs of forebrain disease (none, mild, moderate or severe), signs of caudal fossa disease (none, mild, moderate or severe) [Tables 1-3]. Dogs were categorised as having multifocal or diffuse CNS disease if neurolocalisation indicated CNS disease that could not be ascribed to a single neuroanatomical region. Dogs were also categorised based on alterations in level of consciousness (no, yes or yes but drug induced).

Cerebellomedullary cistern CSF collection was performed using aseptic technique following standard procedures.1, 3, 4 CSF findings were recorded and categorised into normal or abnormal. An abnormal CSF was considered if TNCC was ≥7 cells/μL, TP >30 g/L, or if unusual cells were identified via cytological analyses (such as neoplastic cells or white blood cells not normally found in CSF).4, 13-16 In the present study, the TNCC was corrected in samples with red blood cell contamination by subtracting 1 from the TNCC for every 500 X 106 RBC.4 Corrected TNCC was subsequently categorised as normal (<7 cells/µL) or abnormal (≥7 cells/µL).

Anaesthetic information recorded included premedication, anaesthetic induction agent and the length of time dogs were anaesthetised (<30 minutes versus ≥30 minutes).

Combining information from diagnostic tests (such as neurocranial imaging and CMCCSF findings) in conjunction with historical and clinical examination findings, a presumptive diagnosis was made for each case. The presumptive diagnoses were categorised into intracranial disease, extracranial disease, or an open diagnosis. The length of hospitalisation post CMCCSF collection and survival was also recorded. Survivors were defined as dogs that survived to discharge following CMCCSF collection. Dogs that were euthanised or died within 24 hours of CMCCSF collection were recorded as non-survivors. The length of hospitalisation was recorded in days from the day CSF collection was performed. If the dog was discharged from hospital the same day, then this was considered as 1 day.

All adverse events encountered within 24 hours after CMCCSF collection were recorded and categorised based on clinical relevance. Clinically relevant complications following CMCCSF collection included subjective worsening severity of existing neurological signs, development of new neurological signs, failure to recover from anaesthesia or development of apnoea requiring ventilation following CSF collection. Complications that were not considered clinically relevant...
included dogs displaying mild behavioural changes or dogs that subjectively appeared more ataxic following CMCCSF collection for less than 24 hours.

Descriptive statistics were used to describe the frequency distributions of the study population and those with clinically relevant complications across each variable of interest. Univariable logistic regression was used to estimate odds ratios for other categories of each putative risk factor relative to the category chosen as the reference category. These odds ratios reflect the crude association between the putative risk factor of interest and the occurrence of clinically relevant complications after CMCCSF collection within the study population. During the study design phase, additional multivariable analyses were planned to identify a set of risk factors that best explained the occurrence of complications. However, due to the small number of dogs with clinically relevant complications, this was not conducted. All analyses were conducted using Stata® Version 10.e Results were considered significant at \( P \leq 0.05 \).

**Results**

The initial record search identified 282 dogs that underwent CMCCSF collection between July 2008 and July 2010. One hundred and thirty-two were subsequently excluded from the study due to a diagnosis of spinal cord disease \( (n = 97) \), no index of suspicion of intracranial disease \( (n = 17) \), myelography \( (n = 13) \), lumbar CSF collection \( (n = 3) \) or incomplete medical records \( (n = 2) \). Thus, the study population consisted of 150 dogs.

The median age was 6.0 years (range 0.5-15 years). Fifty-one (34.0%) dogs were <3 years, 53 (35.3%) between 3-8 years and 46 (30.7%) were >8 years. There were 56 (37.3%) toy breed dogs, 25 (16.7%) terriers, 9 (6.0%) non-sporting dogs, and 60 (40.0%) dogs of other breeds included in the study. The most common breeds included Maltese or Maltese crosses \( (n = 27, 18\%) \) followed by Staffordshire bull terriers \( (n = 11, 7.3\%) \). There was an equal distribution of male and females in this study. Of the females, 62 (41.3%) were spayed, and of the males, 58 (38.7%) were neutered. Median body weight was 11.7kg (range 1.9 to 62.5kg). Heavier dogs (> 20kg) made up most cases in the study \( (n = 65, 43.3\%) \), followed by dogs < 6kg \( (n = 47, 31.3\%) \). (Table 1).

CSF results were abnormal in 57.3% of cases, with 58.7% of abnormal samples having pleocytosis and 41.3% of dogs having elevated total protein; 6.6% of CMCCSF samples had red blood cell contamination with values >500 RBC/µL (median 1035.5; range 508–5,073). Final diagnoses are detailed in Table Four.

One hundred and thirty-nine (92.7%) of 150 dogs survived and were discharged from hospital. Of the 11 non-survivors, 9 (81.8%) were euthanised and 2 (18.2%) died within 24 hours of CSF collection. Of those that were euthanized, four were euthanized due to significant neurological deterioration and five were euthanized based on MRI findings. Median length of hospitalisation for surviving animals was 2 days (range 1 to 12 days).

Clinically relevant complications occurred in 10.0% of cases \( (n = 15) \) following CMCCSF collection. Complications included neurological deterioration \( (n = 9) \), seizures \( (n = 2) \), apnoea requiring manual ventilation for >12 hours \( (n = 3) \), and bradyarrhythmia \( (n = 1) \).

Of the 15 dogs that experienced clinically relevant complications, 12 (80%) had a final diagnosis of structural intracranial disease \( (7 = \text{presumed non-infectious meningitis of unknown origin, } 3 = \text{brain tumour, } 1 = \text{brain lesion or mass, } 1 = \text{intracranial haemorrhage}) \). Of the remaining three cases, one was subsequently diagnosed with idiopathic epilepsy. In the remaining two cases, the diagnosis was not determined.
The frequency of complications associated with CMCCSF collection across each of the variables of interest are summarised in Tables 1 and 2. There was no evidence of a strong association between the occurrence of clinically relevant complications and variables used to describe signalment, including body size. There was evidence of an association between the development of complications and several of the variables used to describe history and clinical findings. There was an association between severity of forebrain disease signs and risk of complications. Those with moderate/severe signs were at increased risk compared to those with no signs of forebrain disease (moderate: OR 12.5, 95% CI: 1.3 – 116.8, p = 0.027, severe: OR: 105, 95% CI: 5.2 – 2135, p = 0.002). Dogs with rapidly progressing neurological signs were at much greater risk of complications than those with static signs (OR: 14.8, 95% CI: 2.1 – 104, p = 0.007), as were those with signs of multifocal/diffuse CNS disease rather than without (OR: 5.4, 95% CI: 1.2 – 24.7, p = 0.011). Dogs with signs of severe rather than no caudal fossa disease had a statistically significant increased risk of complications (OR: 15.3, 95% CI: 2.1 – 110, p = 0.007).

There was no evidence of a strong association between anaesthetic length and concurrent invasive procedures and the risk of complications following CMCCSF collection. There was evidence of an association between the occurrence of clinically relevant complications and pleocytosis (OR: 3.5, 95% CI: 1.1 – 11.7, p = 0.027).

### Table 1: Descriptive statistics and odds ratios for possible risk factors associated with complications of CMCCSF collection (signalment/presenting signs).

<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>Category</th>
<th>No. of animals (%)</th>
<th>No. with complications (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient size</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Small &lt;6kg</td>
<td>47 (31.3)</td>
<td>4 (8.5)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Medium 6-20 kg</td>
<td>38 (25.3)</td>
<td>6 (15.8)</td>
<td>2.01 (0.53-7.74)</td>
<td>0.307</td>
</tr>
<tr>
<td></td>
<td>Large &gt;20kg</td>
<td>65 (43.3)</td>
<td>5 (7.7)</td>
<td>0.90 (0.23-3.53)</td>
<td>0.875</td>
</tr>
<tr>
<td><strong>Breed</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Toy</td>
<td>56 (37.3)</td>
<td>6 (10.7)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Terrier</td>
<td>25 (16.7)</td>
<td>2 (8.0)</td>
<td>0.72 (0.14-3.87)</td>
<td>0.706</td>
</tr>
<tr>
<td></td>
<td>Non-sporting</td>
<td>9 (6.0)</td>
<td>1 (11.1)</td>
<td>1.04 (0.11-9.83)</td>
<td>0.972</td>
</tr>
<tr>
<td></td>
<td>Other</td>
<td>60 (40.0)</td>
<td>6 (10.0)</td>
<td>0.93 (0.28-3.06)</td>
<td>0.900</td>
</tr>
<tr>
<td><strong>Duration of clinical signs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>&lt; 48 hours</td>
<td>35 (23.3)</td>
<td>3 (8.6)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>48 hours to 1 week</td>
<td>50 (33.3)</td>
<td>6 (12)</td>
<td>1.45 (0.34-6.26)</td>
<td>0.615</td>
</tr>
<tr>
<td></td>
<td>1 week to 1 month</td>
<td>35 (23.3)</td>
<td>6 (17.1)</td>
<td>2.21 (0.51-9.64)</td>
<td>0.293</td>
</tr>
<tr>
<td></td>
<td>&gt; 1 month</td>
<td>30 (20.0)</td>
<td>0 (0.0)</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td><strong>Progression of neurological signs</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Static</td>
<td>76 (50.7)</td>
<td>7 (9.2)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Slowly Progressive</td>
<td>69 (46.0)</td>
<td>5 (7.2)</td>
<td>0.77 (0.23-2.55)</td>
<td>0.669</td>
</tr>
<tr>
<td></td>
<td>Rapidly progressive</td>
<td>5 (3.3)</td>
<td>3 (60.0)</td>
<td>14.79 (2.10-104.01)</td>
<td>0.007</td>
</tr>
</tbody>
</table>

continued
<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>Category</th>
<th>No. of animals (%)</th>
<th>No. with complications (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Signs of forebrain disease</strong></td>
<td>None</td>
<td>36 (24.0)</td>
<td>1 (2.8)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>91 (60.7)</td>
<td>6 (6.6)</td>
<td>2.47 (0.29-21.28)</td>
<td>0.410</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>19 (12.7)</td>
<td>5 (26.3)</td>
<td>12.50 (1.34-116.8)</td>
<td>0.027</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>4 (2.7)</td>
<td>3 (75.0)</td>
<td>105.00 (5.16-2134.80)</td>
<td>0.002</td>
</tr>
<tr>
<td><strong>Multifocal or diffuse CNS disease</strong></td>
<td>No</td>
<td>63 (42.0)</td>
<td>2 (3.2)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>87 (58.0)</td>
<td>13 (14.9)</td>
<td>5.36 (1.16-24.67)</td>
<td>0.031</td>
</tr>
<tr>
<td><strong>Significant level of altered consciousness</strong></td>
<td>No</td>
<td>142 (94.7)</td>
<td>13 (9.2)</td>
<td>Reference group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>7 (4.7)</td>
<td>2 (28.6)</td>
<td>3.97 (0.70-22.53)</td>
<td>0.120</td>
</tr>
<tr>
<td></td>
<td>Yes (drug induced)</td>
<td>1 (0.7)</td>
<td>0 (0.0)</td>
<td>N/A</td>
<td></td>
</tr>
<tr>
<td><strong>Signs of caudal fossa disease</strong></td>
<td>None</td>
<td>67 (44.7)</td>
<td>6 (9.0)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Mild</td>
<td>60 (40.0)</td>
<td>3 (5.0)</td>
<td>0.54 (0.13-2.24)</td>
<td>0.392</td>
</tr>
<tr>
<td></td>
<td>Moderate</td>
<td>18 (12.0)</td>
<td>3 (16.7)</td>
<td>2.03 (0.46-9.08)</td>
<td>0.353</td>
</tr>
<tr>
<td></td>
<td>Severe</td>
<td>5 (3.3)</td>
<td>3 (60.0)</td>
<td>15.25 (2.11-110.01)</td>
<td>0.007</td>
</tr>
</tbody>
</table>

**Table 2:** Descriptive statistics for possible risk factors associated with complications of CMCCSF collection. (During/following CMCCSF collection). Overall p values for each variable are likelihood ratio test p values and p values comparing each category of each variable to the reference category are individual Wald p values. OR: Odds ratio, CI: confidence interval.

<table>
<thead>
<tr>
<th>Explanatory variable</th>
<th>Category</th>
<th>No. of animals (%)</th>
<th>No. with complications (%)</th>
<th>Odds Ratio (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>General Anaesthetic length</strong></td>
<td>&lt; 30 minutes</td>
<td>48 (32.0)</td>
<td>7 (14.6)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>&gt; 30 minutes</td>
<td>102 (68.0)</td>
<td>8 (7.8)</td>
<td>0.50 (0.17-1.47)</td>
<td>0.206</td>
</tr>
<tr>
<td><strong>Invasive procedure(s) performed before CMCCSF collection</strong></td>
<td>No</td>
<td>139 (92.7)</td>
<td>14 (10.1)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>11 (7.3)</td>
<td>1 (9.1)</td>
<td>0.89 (0.11-7.5)</td>
<td>0.917</td>
</tr>
<tr>
<td><strong>Abnormal CSF</strong></td>
<td>No</td>
<td>64 (42.7)</td>
<td>3 (4.7)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>86 (57.3)</td>
<td>12 (14.0)</td>
<td>3.30 (0.89-12.22)</td>
<td>0.074</td>
</tr>
<tr>
<td><strong>CSF Total nucleated cell count (cells/μL)</strong></td>
<td>Normal (&lt;7)</td>
<td>80 (53.3)</td>
<td>4 (5.0)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abnormal (≥7)</td>
<td>70 (46.7)</td>
<td>11 (15.7)</td>
<td>3.54 (1.07-11.69)</td>
<td>0.038</td>
</tr>
<tr>
<td><strong>CSF Total Protein (g/dL)</strong></td>
<td>Normal (&lt;0.30)</td>
<td>88 (58.7)</td>
<td>7 (8.0)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Abnormal (&gt;0.30)</td>
<td>62 (41.3)</td>
<td>8 (12.9)</td>
<td>1.71 (0.59-5.00)</td>
<td>0.324</td>
</tr>
<tr>
<td><strong>Diagnosis of Intracranial disease</strong></td>
<td>No</td>
<td>30 (20.0)</td>
<td>1 (3.3)</td>
<td>Reference Group</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Yes</td>
<td>103 (68.7)</td>
<td>13 (12.6)</td>
<td>4.19 (0.53-33.42)</td>
<td>0.176</td>
</tr>
<tr>
<td></td>
<td>Open diagnosis (uncertain)</td>
<td>17 (11.3)</td>
<td>1 (5.9)</td>
<td>1.81 (0.11-31.97)</td>
<td>0.681</td>
</tr>
</tbody>
</table>
Discussion

To the authors' knowledge, this is the first study to describe the complications occurring when performing CMCCSF collection in dogs with clinical signs of intracranial disease and to estimate their frequency and risk factors for their occurrence. In the present study, 10.0% of cases suffered adverse events following CMCCSF collection. The reported adverse events, including progression of neurological signs, development of seizures and apnoea have been previously reported following CMCCSF collection.5, 10, 11 Our study found that increased risk of clinically relevant complications following CMCCSF collection was associated with dogs presenting with a rapid progression of neurological signs, signs indicative of severe forebrain or caudal fossa disease or evidence of multifocal brain disease. This agrees with what is clinically perceived in practice and what has been reported in human literature.1, 4, 10, 17 This study also identified several risk factors associated with the occurrence of complications.

On univariable analysis, the point estimate suggested a possible strong association between an eventual diagnosis of intracranial (structural) disease and increased risk of CSF complications. However, this estimate was very imprecise so no definitive conclusion could be reached as to whether structural intracranial disease was associated with adverse events following CMCCSF collection (OR: 4.2 [95% CI:0.5-33.4], P = 0.218). Larger studies, in which all cases undergo neurocranial MRI as well as CSF fluid collection would assist in further investigating this association.

Dogs diagnosed with pleocytosis were associated with increased risk of complications following CMCCSF sampling on univariable analysis (P = 0.027). This is consistent with the perception in veterinary practice that dogs with inflammatory CNS are considered at greater risk of CSF complications.1, 4, 10

In this cohort, body size was not associated with increased risk of CSF complications. While this does not support the theory that smaller patients may be at increased risk due to increased prevalence of craniocervical junction abnormalities in these patients9-11, assessment of the craniocervical junction was not performed in all cases. Furthermore, cases with these abnormalities detected may also not have undergone CSF collection due to a perceived increase in risk by the clinician. Therefore, these results must be interpreted with caution.

A longer anaesthetic was similarly not shown to increase the risk of complications following CSF collection in the present study. While theoretically, complications such as neurological deterioration could be expected following prolonged anaesthesia due to alterations in cerebral perfusion10-11, this could not be demonstrated. Previous studies have found a correlation between anaesthetic length and post-anaesthetic complications in dogs anaesthetised for various different reasons.18, 19 However, to the author’s knowledge, no veterinary studies have been performed to investigate anaesthetic length on prognosis in patients with neurological disease. As the present study only compared anaesthetic length over or under 30 minutes, future studies may be useful to breakdown the anaesthetic lengths further (e.g. <1 hour, 1-2 hours, >2 hours) to determine if there is increased risk with prolonged anaesthesia.

There were several limitations in the present study. Firstly, the presence of iatrogenic contamination of CSF with peripheral blood occurred in 10 dogs during CSF collection. While study results have been conflicting in the veterinary literature, some studies have shown that blood contamination may lead to higher TNCC and TP, which could affect the
interpretation of the results.\textsuperscript{4,20-24} Although correction can be performed, the accuracy reduces with higher contamination levels.\textsuperscript{4,22} One previous study investigated the effects of iatrogenic blood contamination on CSF analyses in 53 dogs.\textsuperscript{22} Blood contamination was not shown to significantly increase TP or TNCC in cases of mild to moderate blood contamination (RBC values <13,230 RBC/µL). In our study, of the cases which had peripheral blood contamination of CSF, none exceeded 5,073 RBC/µL, which is far below the reported upper limit in the previous study.\textsuperscript{22} Therefore, using the corrected results were considered reliable in the present study.

A second limitation was the lack of a validated scoring system to categorise the severity of forebrain or caudal fossa signs. At the time the present study was performed, such a scoring system (objective or subjective) for severity of neurological signs did not exist. Therefore, we developed our own scoring system to assess severity of neurological signs in dogs. This is a fundamental problem as the scoring system is very subjective in nature.

Selection bias is another limitation in this study. Dogs with a rapidly deteriorating clinical state, space-occupying brain lesions or with evidence of craniocervical junction abnormalities such as Chiari-like malformation or cerebellar herniation were possibly less likely to undergo CSF sampling due to perceived increased risk of the procedure. Therefore, these potentially ‘higher risk’ patients could not be included in the present study as they did not undergo CSF sampling and therefore the risk of CMCCSF sampling may be underestimated in the present study. Furthermore, 36% of patients underwent CSF sampling without prior imaging, making the true frequency of structural intracranial disease or craniocervical junction abnormalities in the study population unknown.

Being retrospective in nature, there were other limitations. Abnormalities in systemic blood pressure and heart rate, which may give an indication of ICP elevation, were not consistently reported in all cases. Therefore, conclusions cannot be drawn regarding increased risk of CMCCSF collection in patients who present with or develop signs of increased ICP. This would be very useful information as these values are readily assessable in practice. Larger, prospective clinical studies in which blood pressure measurements are recorded concurrently with heart rate alterations prior to and during anaesthesia for CMCCSF collection would be ideal to determine if these parameters are risk factors for adverse events.

Another limitation in the present study was that it could not be determined if the development of complications was due to CSF collection, anaesthesia, or disease progression. As CMCCSF collection must be performed under a general anaesthesia, the possible confounding variable of anaesthesia cannot be avoided. Similarly, patients with rapidly progressing clinical signs who had adverse events following CSF collection may have been more likely to have neurological deterioration simply due to disease progression, regardless of having the procedure performed.

In our case series, we could not accurately quantify the skill level of the clinician performing CSF collection. Cerebellomedullary cistern CSF collection was performed by specialist clinicians, registrars, residents, or interns under supervision of a specialist clinician. The number of CSF collection attempts performed, any technical difficulty encountered in performing CSF collection, and/or skill of the operator would have been interesting risk factors to assess in this study.

Further limitations of this study include the relatively small population size and small
number of complications. These features affected the power of the study and the ability to adjust for confounding. The study had limited power to detect associations. The small number of dogs experiencing the outcome of interest meant that it was not statistically appropriate to conduct multivariable analyses. As a result, it was not possible to identify whether the observed univariable associations were true associations or whether the observed effects were due to confounding by other variables.

The author acknowledges that the data used in this study dates from 2008-2010. With increased availability of high-field MRI, more recent data may have an increase in detection of more subtle intra-cranial lesions. However, as the technique for CMCCSF collection has not altered over this time, the author believes the results are useful and relevant.

In conclusion, in this population of dogs, CMCCSF collection is associated with a 10.0% (low but clinically important) risk of complications with the most common complication recorded as deterioration in neurological status following CSF collection. Risk factors for complications include dogs presenting with signs indicative of severe forebrain or caudal fossa disease, multifocal disease and rapidly progressing neurological signs. This study is useful for clinicians because it indicates that CMCCSF collection should be performed with caution in dogs presenting with signs indicative of severe or multifocal CNS disease or dogs with rapidly progressing neurological signs, regardless of the underlying aetiology. This study also allows clinicians to indicate the likelihood of adverse events when discussing CMCCSF sampling more confidently with owners. Further larger, prospective clinical studies are required to increase the strength of data presented here.

Conflict of interest
The authors declare no conflict of interest in this research, and no external funding was sought or obtained.

Acknowledgements
The authors would like to thank Veterinary Specialist Services for use of their data.

References
10. Child G. Treatment of CNS Inflammatory Disease (GME, MUA in dogs. Australian College of Veterinary Science Week, Gold Coast, Brisbane, Queensland, Australia, June 2010.


What is Your Diagnosis? The answer and some more questions

Radiographic findings:

There is good peritoneal and retroperitoneal serosal detail. The stomach is moderately distended with fluid and gas which distributes normally with changes in patient position. On both lateral projections, caudal to the stomach, within a loop of small intestine, there is a rectangular soft tissue opacity structure with a mixed striated to bubble gas pattern throughout. This structure is not definitively identified on the ventrodorsal projection. The remainder of the small intestines contain some gas and fluid, there is no evidence of small intestinal overdistension.

The liver, spleen, left and right kidneys are within normal limits. The urinary bladder is minimally distended. The included thorax and musculoskeletal structures are normal.

Radiographic diagnosis: Small intestinal foreign body without evidence of intestinal obstruction.

In case you missed it- here are zoomed in images of the foreign body. What is it most likely to be?
**Diagnosis:** This dog has a small intestinal foreign body, most likely a corn cob, which is not currently causing intestinal obstruction. The dog has a hypochloremia metabolic alkalosis consistent with the history of vomiting for three days and likely indicates that the foreign body was causing a gastric outflow obstruction. Given that the foreign body is within the small intestine without radiographic evidence of obstruction it may be that this foreign body will be self-resolving.

**Treatment plan:** The dog was admitted to hospital and placed on IV fluids. In cases such as these, consideration may be given to performing abdominal ultrasound however in this case there is no clinical reason to perform ultrasound at this stage given the radiographic findings.

Following fluid therapy, the dog was much brighter and there was no further vomiting. Repeat blood gas and electrolytes showed resolution of the alkalosis and hypokalaemia.

Repeat abdominal radiographs were performed 18 hours following the initial series. Describe the radiographic findings. What’s your diagnosis?
What’s your diagnosis?
There is good peritoneal and retroperitoneal serosal detail. The previously described moderately distended stomach has returned to normal size. The previously described small intestinal foreign body can now be seen within the gas dilated descending colon. The small intestines contain a small amount of gas and fluid, there is no evidence of overdistention. The liver, spleen, left and right kidneys are normal. The urinary bladder is very distended. The included thoracic and musculoskeletal structures and normal.

Radiographic diagnosis: The foreign body has progressed through the gastrointestinal tract and is now within the descending colon.

The dog started eating whilst in hospital, there was no vomiting and was discharged. When contacted 24 hours after discharge, the owner reported that the dog was completely back to normal.

Comments:
Radiographs remain an essential tool for the investigation of vomiting in dogs and cats. Radiographs provide a quick and relatively cheap assessment of the gastrointestinal tract for evidence of mechanical obstruction. For this dog, a small intestinal foreign body was present within the small intestine, however there was no radiographic evidence of mechanical obstruction.

In the dog, the normal small intestines are of a uniform diameter containing some fluid and often gas. Various measurements of the small intestines have been recommended, the most useful being the ratio of the maximum small intestine diameter to the narrowest height of the body of L5. If the ratio is <1.6 an obstruction is very unlikely, if the ratio is > 1.6 obstruction is likely and higher values, approaching 2, are significantly associated with obstruction. However linear foreign bodies may not lead to significant small intestinal distension so are an exception to this rule. In this patient’s initial radiographs the small intestines were not overdistended therefore obstruction was unlikely. Follow up radiographs confirmed that the foreign body had progressed to the colon, thus obstruction did not occur because of the foreign body in this patient.

The value of follow up radiographs in vomiting dogs should not be underestimated. On an initial series it may be that the patient has clear radiographic evidence of mechanical obstruction, thus surgery is indicated. In others, such as this patient, the small intestines are within normal limits, thus mechanical obstruction is not present, and medical management is indicated. However, there are some cases which are equivocal, these are the challenging cases. In the equivocal cases ultrasound examination may be indicated for further evaluation of the gastrointestinal tract and other abdominal organs but repeat radiographs in 12-24 hours can provide valuable information regarding the progression, or resolution of the gastrointestinal disease.

References
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