OPEN ACCESS

University of BRISTOL

Publisher's PDF, also known as Version of record

Link to published version (if available):
10.2460/javma.249.10.1180

Link to publication record in Explore Bristol Research
PDF-document

University of Bristol - Explore Bristol Research
General rights

Medical management of spinal epidural empyema in five dogs

Susana R. Monforte Monteiro DVM

Antonella Gallucci DVM, PhD

Nicolas Rousset BVSc

Paul M. Freeman MA, VetMB

Edward J. Ives MA, VetMB

Gualtiero Gandini DVM, PhD

Nicolas Granger DVM, PhD

An E. Vanhaesebrouck DVM

From the Department of Veterinary Medicine, University of Cambridge, Cambridge CB3 0ES, England (Monforte Monteiro, Rousset, Freeman, Ives, Vanhaesebrouck); the Department of Veterinary Medical Sciences, University of Bologna, Via Tolara di Sopra, 50 40064 Ozzano dell'Emilia BO, Italy (Gallucci, Gandini); and the School of Veterinary Sciences, University of Bristol, Langford, Bristol BS40 5DU, England (Granger). Dr. Rousset's present address is Western Veterinary Specialist and Emergency Center, 1802 10 Ave SW, Calgary, AB T3C 0J8, Canada. Dr. Ives' present address is Anderson Moores Veterinary Specialists, The Granary, Bunstead Barns, Poles Ln, Hursley, Winchester SO21 2LL, England. Dr. Vanhaesebrouck's present address is Nuffield Department of Clinical Neurosciences, Level 6, West Wing, John Radcliffe Hospital, Oxford OX3 9DU, England.

Drs. Granger and Vanhaesebrouck contributed equally to this study.

Address correspondence to Dr. Monforte Monteiro (susanamonfortemonteiro@gmail.com).

CASE DESCRIPTION
5 dogs were examined because of clinical signs of myelopathy, including signs of pain associated with the spinal region and rapidly progressive neurologic deficits.

CLINICAL FINDINGS
In all dogs, results of MRI were consistent with spinal epidural empyema. Concurrent infectious processes were identified at adjacent or distant sites in all dogs, including diskospondylitis, prostatitis, dermatitis, paraspinal infection following a penetrating injury, urinary tract infection, and pyothorax. Bacteria were isolated from 3 dogs; *Escherichia coli* was isolated from blood, urine, and prostatic wash samples from 1 dog; a *Pasteurella* sp was isolated from a percutaneous aspirate from an adjacent infected wound in a second dog; and a *Corynebacterium* sp was isolated from a thoracic fluid sample from a third dog. For the remaining 2 dogs, results of bacterial culture were negative.

TREATMENT AND OUTCOME
All dogs showed clinical improvement within 2 weeks after initiation of antimicrobial treatment, and all had an excellent long-term outcome.

CLINICAL RELEVANCE
In dogs, spinal epidural empyema has previously been regarded as a surgical emergency. Findings for dogs in the present report suggested that, as is the case for humans, selected dogs with spinal epidural empyema may be successfully managed with medical treatment alone. (*J Am Vet Med Assoc* 2016;249:1180–1186)

A 10-year-old neutered male Golden Retriever (dog 1), 7-year-old sexually intact male Labrador Retriever (dog 2), 10-year-old sexually intact male English Bulldog (dog 3), 11-week-old sexually intact female Boxer (dog 4), and 2-year-old sexually intact female Cocker Spaniel (dog 5) were examined by the veterinary neurology services of the University of Cambridge (dogs 1 and 2), University of Bologna (dog 3), and University of Bristol (dogs 4 and 5) between 2011 and 2013 because of signs of severe pain involving the spinal region and rapidly progressive neurologic deficits. Signs of pain had reportedly first developed between 1 and 10 days prior to initial examination, and neurologic deficits had reportedly first developed between 1 and 2 days prior to initial examination. Clinically relevant historical findings included a 3-day history of dysuria in dog 2, chronic skin disease and long-term corticosteroid administration in dog 3, routine vaccination 3 days previously in dog 4, and previous food intolerance in dog 5.

Dog 1 had a 7-day history of signs of lumbosacral pain and, on initial examination, had nonambulatory paraparesis and decreased withdrawal reflexes in both pelvic limbs. Dog 2 had a 1-day history of signs of cervical pain and, on initial examination, had nonambulatory tetraparesis and a decreased withdrawal reflex in the left thoracic limb. Dog 3 had a 2-day history of signs of lumbosacral pain and, on initial examination, had ambulatory paraparesis and decreased withdrawal reflexes in both pelvic limbs. Dog 4 had a 1-day history of pelvic limb ataxia, signs of thoracolumbar pain that progressed to cervical pain, and thoracic limb ataxia. On initial examination, dog 4 had paraplegia and decreased withdrawal reflexes and a loss of deep pain sensation in the thoracic limbs. Dog 5 had a 10-day history of lumbar pain and reluctance to jump that had progressed to ambulatory tetraparesis. On initial examination, dog 5 had a decreased withdrawal reflex in the right thoracic limb and was more severely affected on the right side than the left.

None of the dogs were febrile except dog 3 (39.5°C [103.1°F]). Dog 2 was lethargic, and dog 3 had diffuse alopecia with variable erythema and pustules on the limb extremities and muzzle. On the basis of physical examination findings, lesions were localized to the L6-S3 spinal cord segments in dog 1; the C6-T2 spinal cord segments in dogs 2, 4, and 5; and the L4-S3 spinal cord segments in dog 3. Given the signs of pain involving the spinal region and rapidly progressive neurologic signs, differential diagnoses for all dogs included spinal epidural empyema, diskospondylitis, meningomyelitis, neoplasia, and intervertebral disk herniation.

Hematologic and serum biochemical testing was performed in all 5 dogs. Results of hematologic
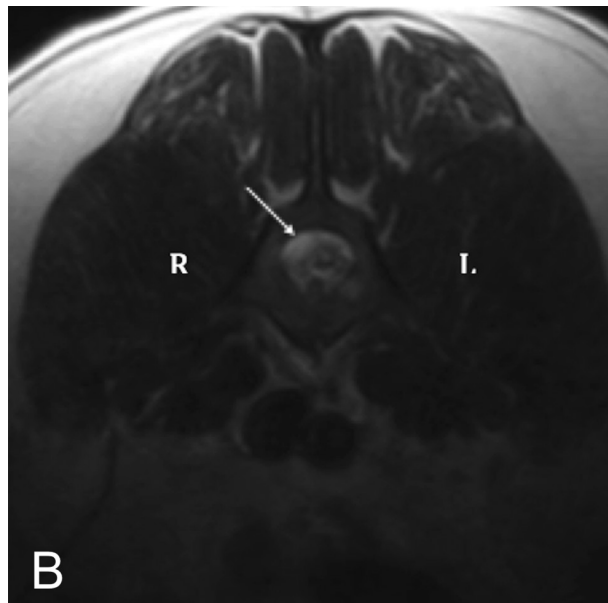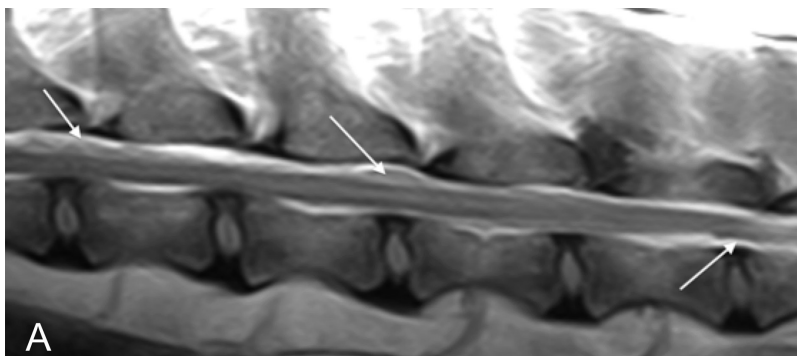
testing were unremarkable in dogs 1 and 3; dogs 2, 4, and 5 had leukocytosis characterized by neutrophilia (14.0×10^9 neutrophils/L [dog 2] and 47.2×10^9 neutrophils/L [dog 5]; reference range, 3.0×10^9 neutrophils/L to 11.5×10^9 neutrophils/L [data were missing for dog 4]). Results of serum biochemical analyses were unremarkable, other than high serum alkaline phosphatase activity in dog 3 (1,181 U/L; reference range, 42 to 180 U/L).

Radiography of the vertebral column was performed in dogs 1, 2, and 3. Results were unremarkable for dog 1, but dogs 2 and 3 had evidence of diskospondylitis at C7-T1 and L7-S1, respectively. Computed tomography of the entire vertebral column was performed in dog 4. A focal hypoattenuating lesion was seen surrounding the spinous process of T2. This lesion was suspected to represent a pocket of fluid, such as an abscess, cyst, or hematoma, although a primary or metastatic tumor could not be ruled out.

Magnetic resonance imaging of the spinal region of interest was performed with a 0.25-T MRI unit[a] in dogs 1 and 2, a 0.22-T MRI unit[b] in dog 3, and a 1.5-T MRI unit[c] in dogs 4 and 5. Imaging revealed multiple extradural lesions from L1 to L7 in dog 1 **(Figure 1)**, a focal lesion at C7-T1 in dog 2, and diffuse lesions from L5 to S1 in dog 3 **(Figure 2)**, from T1 to T4 in dog 4, and from C4 to C7 in dog 5. In dogs 2 and 3, the lesions were associated with the corresponding sites of diskospondylitis observed on radiographs. Dog 3 had lesions within the adjacent paraspinal musculature. In dog 4, MRI confirmed the finding of a pocket of fluid dorsal to T2. In addition, a second pocket of fluid was found in the dermal and subcutaneous tissues above T1. A tubular tract connected these 2 pockets, reaching the T2-3 interarcuate ligament.

All of the extradural lesions were well-circumscribed and appeared hyperintense on T2-weighted images and mildly hyperintense (dogs 1, 2, and 3) or hypointense (dogs 4 and 5) on T1-weighted images **(Figure 3)**. All lesions were contrast-enhancing on T1-weighted images following IV administration of gadolinium-based contrast medium (gadobutrol in dogs 1, 2, and 3 and gadopentetate dimeglumine in dogs 4 and 5). Contrast enhancement was either diffusely homogeneous (dogs 1 and 2), heterogeneous (dog 3), or ring-like (dogs 4 and 5) and was moderate to strong in all dogs except dog 1, which had only minimal contrast enhancement of lesions. Diagnostic imaging findings were most consistent with spinal epidural empyema, although spinal epidural hematoma and epidural steatitis were also considered as possible differential diagnoses.
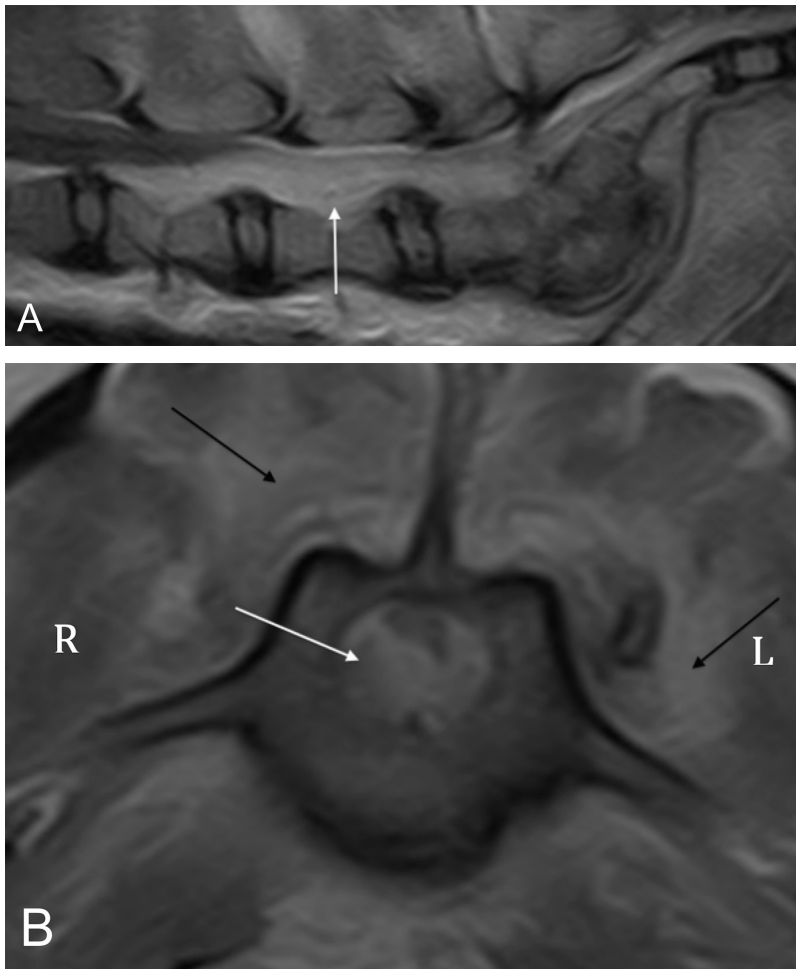
Percentage of spinal cord compression was calculated on transverse T2-weighted MRI images by comparing spinal cord diameter at the level of maximum compression with spinal cord diameter at the nearest level where the spinal cord appeared normal, as described.[1] Spinal cord or cauda equina compression was considered mild (median, 20%; range, 5% to 35%) in all dogs except dog 1, which had moderate (50%) compression.

Additional diagnostic testing was performed in each dog as appropriate, depending on clinical signs. In dog 2, which had a history of dysuria, abdominal ultrasonography revealed several prostatic cavitary lesions consistent with prostatic abscesses. In dog 5, abdominal ultrasonography revealed free abdominal fluid and 2 echogenic foci consistent with migrating foreign bodies in the sublumbar region. Also in this dog, results of MRI were suggestive of pleural effusion, which was confirmed by means of thoracic CT and thoracocentesis. Multifocal soft tissue lesions suggestive of septic embolization were seen at the periphery of the lung fields on CT images.

Figure 1—Sagittal T1-weighted postcontrast MRI image of the lumbar vertebrae (A) of a 10-year-old neutered male Golden Retriever (dog 1) with spinal epidural empyema and a transverse T2-weighted image obtained at the level of L5 (B). In the sagittal image, notice the multiple, minimally contrast-enhancing extradural lesions (arrows). On the transverse image, notice the hyperintense epidural material (arrow).

Figure 2—Sagittal (A) and transverse (B) T1-weighted postcontrast MRI images of a 10-year-old sexually intact male English Bulldog (dog 3) with spinal epidural empyema. On the sagittal image, notice the heterogeneously contrast-enhancing extradural lesion (arrow) extending from L7-S1 (which shows signs of diskospondylitis) to the cranial aspect of L5, causing dorsal displacement of the cauda equina. On the transverse image, which was obtained at the level of L7, notice the severe dorsolateral displacement of the cauda equina by heterogeneously contrast-enhancing epidural material (white arrow). Patchy contrast enhancement of the paraspinal musculature is visible (black arrows).

Because of the presumptive diagnosis of spinal epidural empyema in all dogs, samples were collected and submitted for bacterial culture in an attempt to identify an underlying focus of bacterial infection. In dog 1, urine samples obtained by means of cystocentesis were submitted for urinalysis and bacterial culture. Results of bacterial culture were negative, but cocci were seen during examination of the urine sediment. In dog 2, which had concurrent prostatitis, blood, urine, and prostatic wash samples were submitted for analysis. Urinalysis revealed a high leukocyte count (250 cells/hpf; reference range, 0 to 5 cells/hpf), and cytologic examination of prostatic wash fluid revealed a moderate number of degenerated neutrophils with intracellular rods and activated macrophages with leukophagia. *Escherichia coli* was isolated from urine, blood, and prostatic wash samples. In dogs 3 and 5, urine samples obtained by

means of cystocentesis were submitted for bacterial culture, but results were negative. A *Pasteurella* sp was isolated from fluid aspirated from the paraspinal lesion observed in dog 4, and a *Corynebacterium* sp was isolated from a pleural fluid sample obtained from dog 5. Cytologically, the pleural effusion was characterized by a high number of degenerated neutrophils and intracellular rods.

Cytologic examination of skin scrapings from dog 3, which had chronic pyoderma, revealed *Demodex canis* and diffuse neutrophilic inflammation, but skin samples were not submitted for bacterial culture. Cisternal CSF samples were collected from dogs 1, 4, and 5. All CSF samples had a moderate to high protein concentration (median, 261 mg/dL; range, 58 to 594 mg/dL; reference range, < 25 mg/dL), and samples from dogs 4 and 5 had neutrophilic pleocytosis (52 to 54 cells/μL; reference range, < 5 cells/μL with 86% to 90% neutrophils). In dog 4, cytologic examination of the CSF sample revealed a small number of cocci within neutrophils. For all CSF samples, results of bacterial culture were negative.

In dog 1, results of fecal analysis for *Angiostrongylus vasorum* were negative. In dog 2, results of a coagulation profile were unremarkable.

In all 5 dogs, a concurrent infectious process was identified, including urinary tract infection (dog 1), diskospondylitis with prostatitis (dog 2), diskospondylitis with dermatitis (dog 3), a paraspinal abscess (dog 4), and pyothorax (dog 5).

Medical management of the spinal epidural empyema was chosen on the basis of 1 or more of the following criteria used for human patients[2–12]: minimal to no spinal cord compression (dogs 2 and 4), mild neurologic deficits (dog 3), and severe lesion extension (dogs 1 and 4). In dog 5, medical management was chosen because financial limitations of the owner precluded surgical intervention.

Treatment consisted of IV administration of broad-spectrum antimicrobials. Dog 1 received amoxicillin-clavulanic acid (20 mg/kg [9 mg/lb], IV, q 12 h); dog 2 received a combination of amoxicillin-clavulanic acid (20 mg/kg, IV, q 12 h) and marbofloxacin (2 mg/kg [0.9 mg/lb], IV, q 24 h); dog 3 received marbofloxacin (2 mg/kg, IV, q 24 h); dog 4 received cefuroxime (20 mg/kg, IV, q 12 h), enrofloxacin (2.5 mg/kg [1.14 mg/lb], IV, q 12 h), and metronidazole (10 mg/kg [4.5 mg/lb], IV, q 12 h); and dog 5 received
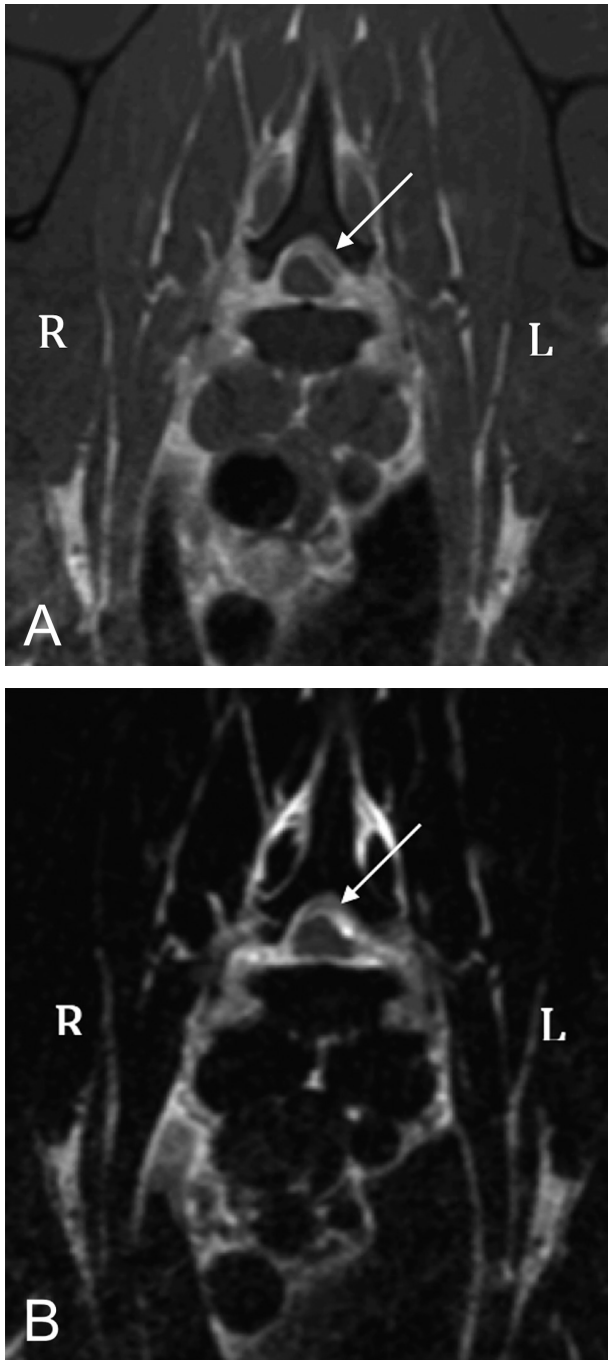
Figure 3—Transverse T1-weighted postcontrast (A) and T2-weighted (B) MRI images obtained at the level of T1 in a 2-year-old sexually intact female Cocker Spaniel (dog 5) with spinal epidural empyema. In the left dorsolateral aspect of the epidural space, epidural material that is hypointense on the T1-weighted image and hyperintense on the T2-weighted image can be seen causing mild compression and distortion of the spinal cord (arrow).

a combination of cefuroxime (20 mg/kg, IV, q 12 h) and metronidazole (10 mg/kg, IV, q 12 h). In dogs 2, 4, and 5, antimicrobials were chosen on the basis of results of antimicrobial susceptibility testing; in dogs

1 and 3, antimicrobials were chosen empirically. All dogs were eventually transitioned to oral antimicrobial administration (with cephalexin replacing cefuroxime in dogs 4 and 5). Antimicrobial administration was continued for a median of 3 months (range, 2 to 24 months; data missing for dog 4) after dogs were discharged from the hospital.

All dogs except dog 3 were treated with methadone (0.2 mg/kg [0.09 mg/lb], IV, q 4 h), buprenorphine (0.02 mg/kg [0.009 mg/lb], IV, q 6 h), or tramadol (2 mg/kg, PO, q 12 h) for pain management; median duration of administration was 5 days (range, 4 to 7 days). In dog 2, methadone and tramadol were insufficient to control signs of pain, and a constant rate infusion of ketamine (0.2 mg/kg/h, IV) and lidocaine (1.2 mg/kg/h [0.55 mg/lb/h], IV) was administered for the first 5 days. In dogs 1 and 3, paracetamol (10 mg/kg, IV, q 12 h) or carprofen (2.2 mg/kg [1 mg/lb], SC, q 12 h) was administered while dogs were hospitalized. In dogs 4 and 5, dexamethasone (0.1 mg/kg [0.045 mg/lb], SC, q 24 h) was administered for 3 days, and in dog 2, gabapentin (20 mg/kg, PO, q 8 h) was administered for 13 days. In dogs 1, 2, and 3, pain medications were administered until dogs were considered to be free from signs of pain during a recheck examination. In dogs 4 and 5, pain medications were administered until dogs were discharged from the hospital.

The dog with prostatitis (dog 2) received monthly injections of delmadinone acetate (1 mg/kg [0.45 mg/lb], SC) and was castrated 4 months after initial examination. The dog with *Demodex* dermatitis (dog 3) was treated with moxidectin.

All dogs improved markedly following initiation of medical management, with rapid resolution of signs of spinal pain, and were ambulatory when discharged from the hospital a median of 7 days (range, 5 to 13 days) after initial examination.

Dog 1 had residual hind limb ataxia 1 week after discharge, but did not have any signs of pain or neurologic deficits when reexamined 2 months after discharge. Dog 2 had mild ataxia 2 months after discharge with no apparent signs of pain. Four months after discharge, no neurologic deficits could be found, and no signs of ongoing infection were seen on follow-up radiographs. Dog 3 had no signs of pain or neurologic deficits when examined 1 week after discharge, and results of a follow-up examination 6 months later were unremarkable. Dog 4 regained deep pain perception after 2 days and was discharged 7 days after initial examination with residual ataxia and ambulatory paresis but no detectable signs of pain. Unfortunately, this dog was lost to follow up after it was discharged. Dog 5 was discharged 5 days after initial examination without signs of pain but with mild residual ataxia. Mild ataxia was still present when the dog was reexamined 4 months later.

Discussion

Spinal epidural empyema and spinal abscess formation refer to bacterial infections within the extra-

dural space.[2] Although an abscess is enclosed by inflammatory tissue, the limits for spinal empyema are the epidural space itself. These rare conditions have the potential to cause severe myelopathy in dogs and cats.[13-17] The term abscess is most commonly used in human medicine, whereas the term empyema has been used more commonly in companion animals and is often used to describe both conditions. Differentiating between an abscess and empyema may be difficult without histologic confirmation. For simplicity, the term empyema was used for the present case series.

Routes of infection previously described for humans and animals with spinal epidural empyema include hematogenous spread of bacteria from a distant site, direct extension from an adjacent infected structure, penetrating injuries, and migrating foreign bodies.[3,4,14] Not surprisingly, diskospondylitis or osteomyelitis is frequently found in association with spinal epidural empyema.[13,18-20,d]

In veterinary medicine, spinal epidural empyema is currently regarded as a surgical emergency, and results of surgical treatment have been reported previously.[14,20-25] However, because a definitive diagnosis is more easily obtained with surgical management, there may be a reporting bias. For human patients, the choice of medical versus surgical management of spinal epidural empyema remains controversial.[2,3,5,6] Rapid surgical drainage was previously considered to be the treatment of choice, but up to 50% of human patients are now treated medically,[2,6,7] and it has been reported that there is a higher mortality rate within the first 4 weeks after initial examination in human patients treated surgically than in patients that receive medical treatment alone. On the other hand, recovery time may be longer in human patients managed medically.[8]

Other than a single conference abstract[d] describing 3 dogs with spinal epidural empyema that were successfully treated medically, all previous reports[13,17,20,21,23,24] of successful management of spinal epidural empyema in dogs involved surgical management. In human medicine, numerous reports[7-10] describe successful management with antimicrobials alone or in conjunction with percutaneous aspiration of septic fluid. Outcome has generally been comparable to that associated with surgery, with success rates ranging from 6% to 100%.[3-5] However, selection bias might have affected the results of many of those retrospective studies, in that patients who underwent surgery may have been more severely affected than patients treated medically. To our knowledge, the present case series is the first to show that medical treatment may be a viable alternative to surgery in certain dogs with spinal epidural empyema.

In the dogs described in the present report, the diagnosis of spinal epidural empyema was suspected on the basis of clinical signs, results of clinicopathologic testing, and MRI findings. Clinical signs and clinicopathologic findings in these dogs were similar to those reported previously for dogs with spinal epidural empyema.[13,14,20-25] Similarly, MRI findings were consistent with those previously reported for dogs with spinal epidural empyema, including hyperintense extradural lesions on T2-weighted images and contrast enhancement in a diffuse or ring-like pattern on T1-weighted images after IV administration of gadolinium-based contrast medium.[5,13,18,19] However, lesions on precontrast T1-weighted images varied from mildly hyperintense (dogs 1, 2, and 3) to hypointense (dogs 4 and 5). In veterinary patients, lesions associated with spinal epidural empyema have previously been reported to be hypointense on T1-weighted images.[13] Because empyema might be masked by adjacent hyperintense epidural fat, fat saturation of T1-weighted and T2-weighted images is recommended in human patients.[11] This might explain the mild hyperintensity of lesions on T1-weighted images for dogs 1, 2, and 3 in the present series. In addition, the use of different types of MRI units (low field vs high field) might have contributed to differing intensity of lesions on T1-weighted images among cases in the present series and cases described previously[13] (mildly hyperintense lesions in low-field images and hypointense lesions in high-field images).

Two important considerations in the treatment of spinal epidural empyema include identification of the infectious agent, to aid in antimicrobial selection, and decompression of neural structures through debridement of the infected area. Although surgery provides an attractive method of achieving both of these goals, there are limitations to consider and potential advantages of less invasive approaches.

For instance, even though surgery provides direct access the lesion, in previous reports[13,14,20,21] of dogs with spinal epidural empyema, results of bacterial culture of samples obtained at the time of surgical debridement were positive for only 12. In human patients, positive results for bacterial culture of blood samples in conjunction with consistent clinical signs and MRI findings are considered diagnostic for spinal epidural empyema,[19] and organisms isolated from blood samples closely match those recovered from empyema fluid.[3,5,10,12] Even without surgical debridement of the affected area, bacterial organisms were identified in 3 of the 5 dogs described in the present report. Although a causative agent was not identified in the other 2 dogs, the clinical signs and MRI findings provided sufficient evidence for a presumptive diagnosis of spinal epidural empyema. In addition, both of these dogs improved following initiation of antimicrobial treatment, supporting the diagnosis.

The importance of immediate surgical decompression in veterinary patients with acute spinal cord compression remains unknown, but a recent study[26] has suggested that the severity of spinal cord injury is not related to the degree of compression alone. Two mechanisms have been proposed to explain the pathophysiology underlying the neurologic signs observed in patients with spinal epidural empyema: direct compression of neural tissue in the spinal ca-

nal, and formation of ischemic injuries secondary to thrombosis or vasculitis.[27,28] Experimental studies[27,29] have suggested that compression plays a larger role than ischemia, because neurologic improvement is observed after surgical decompression, and in dogs, diskospondylitis results in neurologic deficits when granulation tissue extends into the spinal canal, causing compression.[18] However, a study[1] examining spinal cord compression associated with diskospondylitis found that the degree of spinal cord compression did not correlate with the severity of neurologic signs and the outcome was similar between dogs treated with decompressive surgery and those managed medically. In the present case series, one of the dogs with the most severe neurologic deficits had a minimally compressive lesion (dog 2), and another dog (dog 4) with no deep pain sensation in the pelvic limbs nevertheless made a full recovery without surgery.

The additional value of collecting CSF samples in dogs with spinal epidural empyema remains questionable, in that results of bacterial culture of CSF samples are often negative,[20] as was the case for the 3 dogs in the present report from which CSF samples were obtained. For these dogs, CSF samples were retrieved from sites cranial to the lesion to avoid needle placement within the infected lesion, which may have contributed to the negative results.[30] However, a more likely explanation of the negative culture results is that the epidural space is anatomically well separated from the arachnoid space by the dura mater and arachnoid mater. Collection of CSF samples is typically avoided in human patients suspected to have spinal epidural empyema because of the risk of spreading the infection to the subarachnoid space and causing rapidly spreading bacterial meningitis.[3,5] If a definitive diagnosis is pursued (eg, because a primary infectious focus is not identified or there is a poor response to antimicrobial administration), it may be safer to obtain samples by means of percutaneous aspiration of infectious material or surgical exploration of the affected site, rather than attempting to culture CSF.

Findings for dogs in the present report suggested that medical management might constitute a viable alternative in some dogs with suspected spinal epidural empyema. However, there currently are no guidelines to assist veterinarians in deciding whether surgical or medical treatment is most appropriate for a given patient. Criteria used in human medicine to assist in this decision-making process, as well as findings for the 5 dogs reported here, give some insight into how to decide on the most appropriate treatment. Human patients are selected for medical management on the basis of one or more of the following criteria[2-5,7,8,31-33]: absence of substantial neurologic deficits, noncompressive spinal lesions, extensive lesions for which multiple surgical decompressive procedures would create a substantial risk of vertebral instability, poor anesthetic or surgical candidates, and paraplegia for > 48 to 72 hours. All of the dogs

in the present report met one or more of these criteria: dogs 3 and 5 were ambulatory; dog 2 had a focal, minimally compressive lesion; all 5 dogs had < 50% compression of the spinal cord; and dogs 1, 3, 4, and 5 had extensive lesions. There were also financial constraints for the owners of dogs 3 and 5. Given the results for these dogs, we propose that criteria similar to those used in human patients may be useful in helping to decide on the most appropriate treatment for dogs suspected to have spinal epidural empyema.

Acknowledgments

Footnotes

a. Vet-MR Grande, Esaote, Genova, Italy.
b. MR, Paramed, Genova, Italy.
c. Gyroscan Intera NT, Philips, Best, The Netherlands.
d. Escriou C, Duchene LS, Gilbert S, et al. Spinal epidural infection medically treated in 3 dogs: MRI features and follow-up, clinical findings and outcome (abstr), in *Proceedings*. 24th Annu Symp Eur Soc Vet Neurol/Eur Coll Vet Neurol, Trier, Germany, 2012.

References

1. Davis MJ, Dewey CW, Walker MA, et al. Contrast radiographic findings in canine bacterial discospondylitis: a multicenter, retrospective study of 27 cases. *J Am Anim Hosp Assoc* 2000;36:81–85.
2. Reihsaus E, Waldbaur H, Seeling W. Spinal epidural abscess: a meta-analysis of 915 patients. *Neurosurg Rev* 2000;23:175–205.
3. Curry WT Jr, Hoh BL, Amin-Hanjani S, et al. Spinal epidural abscess: clinical presentation, management, and outcome. *Surg Neurol* 2005;63:364–371.
4. Zimmerer SME, Conen A, Muller AA, et al. Spinal epidural abscess: aetiology, predisponent factors and clinical outcomes in a 4-year prospective study. *Eur Spine J* 2011;20:2228–2234.
5. Rigamonti D, Liem L, Sampath P et al. Spinal epidural abscess: contemporary trends in etiology, evaluation, and management. *Surg Neurol* 1999;52:189–197.
6. Siddiq F, Chowfin A, Tight R, et al. Medical vs surgical management of spinal epidural abscess. *Arch Intern Med* 2004;164:2409–2412.
7. Karikari IO, Powers CJ, Reynolds RM, et al. Management of a spontaneous spinal epidural abscess: a single-center 10-year experience. *Neurosurgery* 2009;65:919–924.
8. Grieve JP, Ashwood N, O'Neill KS, et al. A retrospective study of surgical and conservative treatment for spinal extradural abscess. *Eur Spine J* 2009;9:67–71.
9. Tang HJ, Lin HJ, Liu YC, et al. Spinal epidural abscess—experience with 46 patients and evaluation of prognostic factors. *J Infection* 2002;45:76–81.
10. Connor DE, Chittiboina P, Caldito G, et al. Comparison of operative and nonoperative management of spinal epidural abscess: a retrospective review of clinical and laboratory predictors of neurological outcome. *J Neurosurg Spine* 2013;19:119–127.
11. Mink JH, Gordon RE, Deutch AL. The cervical spine: radiologist's perspective. *Phys Med Rehabil Clin N Am* 2003;14:493–548.
12. Nussbaum ES, Rigamonti D, Standiford H, et al. Spinal epidural abscess: a report of 40 cases and review. *Surg Neurol* 1992;38:225–231.

13. De Stefani A, Garosi LS, McConnell FJ, et al. Magnetic resonance imaging features of spinal epidural empyema in five dogs. *Vet Radiol Ultrasound* 2008;49:135-140.

14. Dewey CW, Kortz GD, Bailey CS. Spinal epidural empyema in two dogs. *J Am Anim Hosp Assoc* 1998;34:305-308.

15. Granger N, Hidalgo A, Leperlier D, et al. Successful treatment of cervical spinal epidural empyema secondary to grass awn migration in a cat. *J Feline Med Surg* 2007;9:340-345.

16. Jerram RM, Dewey CW. Suspected spinal epidural empyema and associated vertebral osteomyelitis (physitis) in a dog. *J Vet Emerg Crit Care* 1998;8:216-221.

17. Schmiedt CW, Thomas WB. Spinal epidural abscess in a juvenile dog. *Vet Comp Orthop Traumatol* 2005;18:186-188.

18. Carrera I, Sullivan M, McConnell F, et al. Magnetic resonance imaging features of discospondylitis in dogs. *Ultrasound* 2011;52:125-131.

19. Friedmand DP, Hills JR. Cervical epidural spinal infection: MR imaging characteristics. *AJR Am J Roentgenol* 1994;163:699-704.

20. Lavely JA, Vernau KM, Vernau W, et al. Spinal epidural empyema in seven dogs. *Vet Surg* 2006;35:176-185.

21. Cherrone KL, Eich CS, Bonzynski JJ. Suspected paraspinal abscess and spinal epidural empyema in a dog. *J Am Anim Hosp Assoc* 2002;38:149-151.

22. Holloway A, Dennis R, McConnell F, et al. Magnetic resonance imaging features of paraspinal infection of the dog and cat. *Vet Radiol Ultrasound* 2009;50:285-291.

23. Savage K, Holtom PD, Zalavras CG. Spinal epidural abscess: early clinical outcome in patients treated medically. *Clin Orthop Relat Res* 2005;439:56-60.

24. Sutton A, May C, Coughlan A. Spinal osteomyelitis and epidural empyema in a dog due to migrating conifer material. *Vet Rec* 2010;166:693-694.

25. Plessas IN, Jull P, Volk HA. A case of canine discospondylitis and epidural empyema due to *Salmonella* species. *Can Vet J* 2013;54:595-598.

26. Jeffery ND, Levine JM, Olby NJ et al. Intervertebral disk degeneration in dogs: consequences, diagnosis, treatment, and future directions. *J Vet Intern Med* 2013;27:1318-1333.

27. Feldenzer JA, McKeever PE, Schaberg DR, et al. The pathogenesis of spinal epidural abscess: a pathophysiological model in the rabbit. *Neurosurgery* 1987;20:859-867.

28. Lasker BR, Harter DH. Cervical epidural abscess. *Neurology* 1987;37:1747-1753.

29. Baker AS, Ojemann RG, Swartz MN, et al. Spinal epidural abscess. *N Engl J Med* 1975;293:463-468.

30. Thomson CE, Kornegay JN, Stevens JB. Analysis of cerebrospinal fluid from the cerebellomedullary and lumbar cisterns of dogs with focal neurological disease: 145 cases (1985-1987). *J Am Anim Hosp Assoc* 1990;196:1841-1844.

31. Duarte RM, Vaccaro AR. Spinal infection: state of the art and management algorithm. *Eur Spine J* 2013;22:2787-2799.

32. Tompkins M, Panuncialman I, Lucas P, et al. Spinal epidural abscess. *J Emerg Med* 2010;39:384-390.

33. Pradilla G, Nagahama Y, Spivak A, et al. Spinal epidural abscess: current diagnosis and management. *Curr Infect Dis Rep* 2010;12:484-491.