Idiopathic sterile inflammation of the epidural fat and epaxial muscles causing paraplegia in a mixed-breed dog

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A 4-year-old sexually intact male mixed-breed dog (weight, 26 kg [57.2 lb]) was referred to the Department of Medicine and Clinical Biology of Small Animals, Ghent University, for the investigation of rapidly progressive general malaise and paraplegia. The day before the initial examination, the dog started to display signs of lethargy and anorexia. Results of a general physical examination, performed by the referring veterinarian, demonstrated an increased body temperature of 39.9°C (104°F). A CBC, biochemical panel, and urinalysis detected pyrexia, tachycardia, and tachypnea. Neurologic examination demonstrated severe spinal hyperesthesia and paraplegia with decreased nociception. Magnetic resonance imaging revealed extradural spinal cord compression at T13-L1 and hyperintense lesions on T1- and T2-weighted images in the epaxial musculature and epidural space.

General physical examination revealed a body temperature of 39°C (102°F), a pulse rate of 140 beats/min, and a respiratory rate of 44 breaths/min. The dog was reluctant to move.

Neurologic examination revealed paraplegia with absent proprioception, urinary incontinence, bilaterally absent cutaneous trunk reflexes, increased patellar reflexes, and decreased nociception in both pelvic limbs. Palpation of the thoracolumbar region elicited signs of extreme pain. A CBC revealed a mild leukocytosis (14.4 × 10^9/L; reference range, 3 to 10 × 10^9/L) with a neutrophilia (14.4 × 10^9/L; reference range, 6 to 12 × 10^9/L) and absolute lymphocytosis (17.1 × 10^9/L; reference range). The dog was premedicated for low-field MRI with acepromazine maleate (0.01 mg/kg [0.0045 mg/lb], IV) and methadone (0.02 mg/kg [0.009 mg/lb], IV). Anesthesia was induced with alfaxalone (2 mg/kg [0.91 mg/lb]) and maintained with isoflurane in oxygen. Magnetic resonance imaging of the thoracolumbar region demonstrated a marked hyperintense, heterogeneous, ill-defined lesion bilaterally in the longissimus thoracis, semispinosus, and multifidus thoracic muscles from the caudal aspect of T10 through the cranial aspect of L2 on the sagittal (Figure 1), dorsal, and transverse (Figure 2) T2-weighted and short tau inversion recovery sequences. This lesion appeared isointense to mildly hyperintense on T1-weighted images and demonstrated prominent enhancement with gadolinium contrast (0.3 mg/kg [0.14 mg/lb], IV). No obvious involvement of the surrounding...
vertebral structures was seen. This lesion extended into
the vertebral canal and was determined to be hyperintense
on T1- and T2-weighted images, causing marked dorsal
to dorsolateral extradural spinal cord compression at the
level of T12-13 and showing mild contrast enhancement.
Immediately following the MRI procedure, CSF was col-
lected by a lumbar puncture and was within reference
limits.
Spinal epidural empyema with inflammation of the epaxial muscles was considered the most likely differential diagnosis. Urine and blood samples were obtained under sterile conditions for further bacteriologic culture. A urinary catheter was placed. Because of the rapidly progressive neurologic deterioration and the presence of extradural spinal cord compression, the dog was immediately admitted for decompressive surgery. Intraoperative analgesia was provided with a continuous rate infusion of fentanyl (7 µg/kg/h [3.2 µg/lb/h], IV) and lidocaine (30 µg/kg/min [13.64 µg/lb/min], IV). The patient was positioned in sternal recumbency and prepped and draped for aseptic surgery in standard fashion. During a standard dorsal approach to the thoracolumbar vertebral column, abnormalities of the epaxial musculature were noticed. The muscles had a swollen, pale, and friable appearance. A standard dorsal laminectomy with preservation of the articular facet joints was performed at the level of T12-13. During inspection of the vertebral canal, the spinal cord was swollen, and the epidural fat had a granular and dark red appearance. Several biopsy samples were collected from the epaxial musculature, epidural fat, and spinous processes. Thereafter, broad-spectrum antimicrobials were administered (amoxicillin-clavulanic acid, 20 mg/kg [9.09 mg/lb], IV, q 2 h). The laminectomy defect was extended cranially and caudally until normal-appearing epidural fat was encountered. This resulted in a continuous dorsal laminectomy from T10 through L2. Finally, the abnormal, granular epidural fat was removed as much as possible by means of curettage, and the vertebral canal was copiously lavaged with sterile physiologic saline (0.9% NaCl) solution. A synthetic cellulose patch was placed in the laminectomy defect, and the surgery site was closed via a routine 3-layer closure by means of polydioxanone and polyglecaprone. The biopsy samples were submitted for aerobic bacteriologic culture and histologic examination.

The dog was hospitalized after surgery with an indwelling urinary catheter and was administered IV fluids, a broad-spectrum antimicrobial (amoxicillin-clavulanic acid, 20 mg/kg, IV, q 8 h), enrofloxacin, (5 mg/kg [2.27 mg/lb], IV, q 24 h), a gastroprotective agent (ranitidine, 2 mg/kg [0.907 mg/lb], IV, q 12 h), and an NSAID (carprofen, 2 mg/kg, IV, q 24 h). The postoperative analgesia consisted of a constant rate infusion of lidocaine (30 µg/kg/min, IV), morphine (0.4 mg/kg [0.18 mg/lb], IV, q 4 h) for the first 24 hours, and paracetamol (10 mg/kg [4.55 mg/lb], IV, q 12 h) for 4 consecutive days. On postoperative day 4 a fentanyl patch (100 µg/h) was applied, and was removed one day before discharge from the hospital, when the dog commenced oral pain medication.

During hospitalization, a complete neurologic examination was performed daily. Two days after surgery, nociception had returned to normal. After 5 days of inappetence, a nasoesophageal tube was placed and enteral feeding was initiated. On the seventh day after surgery, the dog was able to support its weight and started eating on its own. At this time, the urinary catheter and the nasoesophageal tube were removed, and the dog started urinating. The dog was discharged 8 days after surgery. Pending the results of bacteriologic cultures and histologic examinations, the owners were advised to administer enrofloxacin (5 mg/kg, PO, q 24 h), amoxicillin-clavulanic acid (12.5 mg/kg [5.67 mg/lb], PO, q 12 h), carprofen (4 mg/kg [1.81 mg/lb], PO, q 24 h), and ranitidine (2 mg/kg, PO, q 12 h). No physical therapy was initiated at this time.

Cultures of blood and urine samples as well as aerobic bacterial cultures of muscle and epidural fat specimens were negative. Histologic examination of the muscle biopsy specimen revealed diffuse intercellular edema with infiltration of neutrophils between the mostly swollen myocytes, which showed a hyaline eosinophilic or fragmented sarcoplasm. The epidural fat specimen revealed well-differentiated adipose tissue with infiltrated neutrophils and macrophages and scattered bleedings. Histologic examination of the spinous process revealed a normal bony structure. These findings confirmed a diagnosis of suppurative myositis and suppurative epidural steatitis. In light of the negative bacteriologic cultures, antimicrobial treatment was discontinued.

Twenty-nine days, 30 days, 62 days, and 2 years after surgery, the dog was re-evaluated at Gent University. Twenty-nine days after surgery, the dog was able to ambulate with support, although still severely ataxic. At this time, proprioceptive deficits were still present. The owners were advised to initiate hydrotherapy, which they started the same day. The next day, the dog had a clinical relapse, consisting of recurrence of spinal hyperesthesia. On admission, physical and neurologic examinations were unchanged, and a CBC and biochemical panel were unremarkable. A tapering oral prednisolone treatment was initiated for 3 weeks (1 mg/kg [0.45 mg/lb], PO, q 24 h for 1 week; 0.5 mg/kg [0.23 mg/lb], PO, q 24 h for 1 week; and 0.5 mg/kg, PO, q 48 h for 1 week). Sixty-two days after surgery, the dog was able to ambulate independently. Neurologic examination revealed only moderate pelvic limb ataxia. No more clinical relapses were noticed by the owners. Two years after surgery, only mild pelvic limb ataxia was present, and further neurologic examination was unremarkable. Three years after surgery, the owners confirmed an unchanged neurologic status.

**Discussion**

Although idiopathic sterile inflammation of adipose tissue, referred to as panniculitis, more commonly affects subcutaneous tissue, its presence in the vertebral canal is rare. Specific MRI findings for the patient described in this report may help in reaching a presumptive diagnosis of this neurologic disorder. This is notable because the clinical signs alone may have suggested differential diagnoses such as neoplasia or infection, for which the prognosis might be expected in many instances to be poor. For patients with idiopathic sterile inflammation of epidural fat, surgical decompression may allow a definitive diagnosis and successful long-term outcome in affected patients.

The histopathologic abnormalities described in the patient of the present report resemble those of panniculitis, a multifactorial inflammatory condition of the subcutaneous fat. Panniculitis is rather uncommon in dogs and cats and is idiopathic in the majority of cases. Most affected animals have only skin lesions, but they may have generalized signs such as inappetence, depression, lethargy, and pyrexia.2–5

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One report describes 5 Miniature Dachshunds with thoracolumbar idiopathic sterile pyogranulomatous inflammation of epidural fat causing spinal cord compression and neurologic signs. All dogs were treated surgically by a hemilaminectomy. Epidural fat specimens submitted for histopathologic examination revealed pyogranulomatous inflammation of the epidural fat, histologically similar to subcutaneous idiopathic sterile pyogranulomatous inflammation or sterile panniculitis. Therefore, the condition in these Dachshunds was considered to have a similar pathogenesis to panniculitis. In contrast to the dog of the present report, none of the previously reported Miniature Dachshunds showed systemic signs, and MRI of the vertebral column was not performed in any of the dogs.

Spinal epidural empyema was considered the most likely differential diagnosis in the dog of the present report. This is characterized by an accumulation of purulent material within the vertebral canal. Dogs with spinal epidural empyema are typically pyrexic with a rapidly progressive myelopathy. Lesions are generally hyperintense on T2-weighted images, iso- to hypointense on T1-weighted MRI images, and associated with mild to moderate peripheral or diffuse contrast enhancement. In contrast, the lesion in the dog of the present report was hyperintense on T1-weighted images, probably caused by the adipose nature of the affected tissue, which typically causes a T1 hyperintensity. Therefore, T1 hyperintensity might be a key feature to distinguish spinal epidural empyema from an inflammatory lesion of the epidural fat.

A continuous dorsal laminectomy over 6 vertebral bodies, as performed in our patient, can be considered a rather invasive procedure that can potentially cause vertebral instability. However, since the primary differential during surgery was infectious in nature, it was not considered opportune to use spinal implants to stabilize the vertebral column. Special care was taken to leave the articular facet joints completely intact during surgery.

Histologic examination revealed a suppurative myositis and suppurative steatitis of the epidural fat. Samples of the skin and subcutaneous tissue were not obtained because it was not clinically relevant at that time. If we consider a similar pathogenesis of idiopathic sterile inflammation of epidural fat to cutaneous idiopathic sterile panniculitis, the histopathologic lesion can be granulomatous, pyogranulomatous, suppurative, cosinophilic, necrotizing, or fibrosing. In the report of the Miniature Dachshunds, all lesions were pyogranulomatous in nature, whereas the lesions in the patient of the present report were suppurative. The presence of a suppurative inflammation could suggest an underlying infectious cause for this dog’s clinical signs, but there was no growth on bacteriologic culture of the tissues, and no bacteria were demonstrated on histologic examination of epidural fat and paraspinal muscles. In the veterinary literature, 15 cases of spinal epidural empyema are described, and only 2 included positive bacterial cultures.

Identified bacteria included *Enterobacter cloacae*, coagulase-positive *Staphylococcus*, *Pasteurella multocida*, (hemolytic) *Escherichia coli*, β-hemolytic *Streptococcus canis*, *Staphylococcus intermedius*, *Bacteroides* spp, *Pevotella* spp, and *Enterococcus fecalis*. In 2 cases, *Clostridium perfringens* was identified on anaerobic cultures of epidural specimens obtained during decompressive spinal surgery. In dogs, the most successful sites for positive cultures are the surgical site and blood, which were both negative in the patient described in the present report. In human medicine, culture of the surgical site is 90% sensitive, which can be increased up to 97% when no preoperative antimicrobials are administered. In the dog of the present report, antimicrobials were administered only after biopsy and culture samples were obtained, which makes false-negative results for bacteriologic culture unlikely. However, anaerobic bacteriologic cultures were not performed and therefore cannot be excluded, although no bacterial organisms were identified on histologic examination of the epidural fat.

Retrospective studies have demonstrated successful treatment of a large number of dogs with panniculitis with immunosuppressive drugs. Almost 30% of the dogs went into remission, whereas approximately 63% needed prolonged, alternate-day immunosuppressive treatment. Almost all dogs were treated orally with prednisone (1 to 3.5 mg/kg [0.45 to 1.59 mg/lb], PO, q 12 h), sometimes in combination with cyclosporine-A, azathioprine, dapsone, or vitamin E, for 3 to 8 weeks. Since the dog of the present report and all Miniature Dachshunds of the previous report were treated by decompressive surgery, it is difficult to establish an indication or efficacy for glucocorticoid administration in dogs with an epidural location of affected adipose tissue. Two out of 5 Miniature Dachshunds neurologically improved without adjunctive corticoid therapy. Because of the suspicion of an underlying infectious cause, the patient described in the present report was not started immediately on immunosuppressive therapy. When the laboratory results returned all negative, the dog was already improving clinically and not started on an adjunctive therapy at that time. At the time of the suspected clinical relapse, the dog was successfully treated with a tapering prednisolone regimen, although differentiating between a relapse and fatigue due to hydrotherapy is difficult in this case.

We can conclude that idiopathic sterile epidural steatitis can be a cause of thoracolumbar myelopathy and severe spinal pain, possibly combined with systemic signs such as pyrexia, anorexia, and general malaise. Magnetic resonance imaging demonstrated nonspecific inflammatory features in the paraspinal muscles, although the lesion in the vertebral canal can be differentiated from empyema through the hyperintense signal on both T1- and T2-weighted images. However, a definitive diagnosis can be achieved only by bacterial and histologic examinations. Decompressive surgery may result in improvement of neurologic signs and is indicated to reach a definitive diagnosis. The role of additional or initial immunosuppressive therapy is uncertain and warrants further investigation.

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a. Airis Mate, Hitachi Ltd, Tokyo, Japan.
c. Dotarem, Guerbet, Brussels, Belgium.
d. Gelfoam, Pfizer Manufacturing, Puurs, Belgium.
e. Perfluosalgan, Bristol-Meyers Squibb, Braine-l’Alleud, Belgium.
References


From this month’s AJVR

Pharmacokinetics of intravenously and orally administered meloxicam in sheep

Matthew L. Stock et al

**Objective**—To determine the pharmacokinetics of meloxicam after IV and PO administration to 6 healthy sheep.

**Animals**—6 healthy adult Dorset cross sheep (5 males and 1 female).

**Procedures**—Meloxicam (0.5 mg/kg, IV, or 1.0 mg/kg, PO) was administered in a randomized crossover design with a 10-day washout period. Blood samples were collected at predetermined times over 96 hours. Serum drug concentrations were determined by high-pressure liquid chromatography with mass spectrometry. Computer software was used to estimate values of pharmacokinetic parameters through noncompartmental methods.

**Results**—Following IV administration (n = 5), the geometric mean (range) elimination half-life was 14.0 hours (10.5 to 17.0 hours), volume of distribution was 0.204 L/kg (0.171 to 0.272 L/kg), and clearance was 0.17 mL/min/kg (0.12 to 0.27 mL/min/kg). Following oral administration (n = 6), maximum serum concentration was 1.72 µg/mL (1.45 to 1.93 µg/mL), time to maximum serum concentration was 19.0 hours (12.0 to 24.0 hours), clearance per bioavailability was 0.22 mL/min/kg (0.16 to 0.30 mL/min/kg), and terminal half-life was 15.4 hours (13.2 to 17.7 hours). Bioavailability of orally administered meloxicam was calculated as 72% (40% to 125%; n = 5). No adverse effects were evident following meloxicam administration via either route.

**Conclusions and Clinical Relevance**—Meloxicam administered PO at 1.0 mg/kg has good bioavailability with slow elimination kinetics in sheep. These data suggested that meloxicam may be clinically useful, provided the safety and analgesic efficacy of meloxicam as well as feed-related influences on its pharmacokinetics are established in ruminants. (*Am J Vet Res* 2013;74:779–783)