

CASE REPORT

Companion or pet animals

Clinical, magnetic resonance imaging and computed tomography imaging findings in a young cat with epidural pyogranulomatous steatitis

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Abstract

A 4-month-old male domestic shorthair cat with a 36-h history of acute and progressive painful paraplegia was referred. The neurological examination was consistent with T3–L3 myelopathy. Magnetic resonance imaging of the thoracolumbar region revealed an extradural mass at the level of T12–T13 that caused moderate spinal cord compression. The cat underwent T12–T13 dorsal laminectomy decompressive surgery, and its neurological signs improved shortly after surgery. A diagnosis of epidural spinal steatitis was reached based on histopathological examination of the compressive mass. To the authors' best knowledge, this is the first description of the magnetic resonance imaging and computed tomographic findings of histopathologically confirmed idiopathic sterile pyogranulomatous inflammation of epidural fat in a cat. Epidural idiopathic sterile pyogranulomatous steatitis should be included as a possible differential diagnosis in young cats with acute and progressive T3–L3 myelopathy signs.

BACKGROUND

Epidural idiopathic sterile pyogranulomatous steatitis (EISPS) is an inflammation of the epidural fat, which is infrequently reported in dogs^{1–5} and is extremely rare in cats.⁶ However, complete magnetic resonance imaging (MRI) and computed tomography (CT) studies were not present in the previously described feline case.

EISPS and sterile panniculitis are often used as synonyms in the literature.^{1,2,4} However, their exact aetiology and pathogenesis remain unknown. Some factors have been suspected, such as an immune-mediated reaction to foreign material, pancreatic disorders, nutritional deficiency, drug reaction, insect bite and vasculopathy. A primary immune-mediated reaction and infectious agents (fungal and bacterial) are also considered potential aetiological factors.⁷ The lack of infectious agents and foreign material in some cases of EISPS and the good response to systemic glucocorticoids suggest the involvement of a histiocytic inflammatory response.^{7–9}

Genetic predisposition to panniculitis in Miniature Dachshund dogs has been postulated.⁷ It has been suggested that this breed may also be predisposed to EISPS.^{1,10,11}

The purpose of this report was to describe the combination of clinical, imaging and pathological findings and long-term outcomes in a young cat with EISPS after surgical decompression.

CASE PRESENTATION

A 4-month-old 1.92-kg sexually intact male domestic shorthair cat was referred for evaluation of paraplegia. Thirty-six hours prior to presentation, the cat showed acute onset of kyphosis and pelvic limb stiffness. No history of trauma was reported. Vaccinations, ectoparasite and endoparasite prevention were up to date, and there had been no travel history.

On presentation, the cat was alert and responsive. No abnormalities were detected on general physical examination.

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Neurological examination revealed paraplegia, tail paralysis and questionably absent nociception in the pelvic limbs and tail. The spinal reflexes were intact in all limbs. The cross extensor reflex was bilaterally elicited in the pelvic limbs. The pelvic limbs had markedly increased extensor tone and coxo-femoral flexion, bilaterally. No abnormalities were detected on cranial nerve examination. Mild discomfort was elicited on palpation of the thoracolumbar vertebral column. The neurologic examination findings were suggestive of T3–L3 myelopathy. Further investigations, including blood-work and diagnostic imaging, were performed.

INVESTIGATIONS

Results of haematology, biochemistry profile and urinalysis were within normal limits. MRI of the thoracolumbar spine was performed using a 1.5 T magnet (Philips Ingenia CX). The cat was anaesthetised and positioned in dorsal recumbency.

Dorsal (T2w with mDIXON), sagittal (T2w, T1w, T1w + C [intravenous injection of 0.1 mmol/kg of gadobutrol (Gadovist, Bayer)]) and transverse (T2w, B-FEE, T2*, T1w, T1w + C, 3D T1w + C) were acquired (Figure 1).

The MRI study revealed strong T2-weighted (T2w) and T1-weighted (T1w) hyperintensity and marked contrast enhancement of the articular processes of T12 and T13 vertebrae and the surrounding epaxial muscles. There was a lesion centred on the T12–T13 articular process joints, which was slightly more severe on the left side. The margins of T12–T13 articular processes were ill defined. The abnormal tissue was bulging within the vertebral canal bilaterally, causing mild to moderate, extradural and dorsolateral compression of the spinal cord. At T12–T13, there was T2w and T1w hyperintense, strongly contrast enhancing, extradural material extending from the cranial third of T12 to T13–L1 disc space, which was more marked dorsally. The spinal cord had a patchy T2w hyperintense intramedullary signal and patchy contrast enhancement at this level. Dorsal to L2 vertebral body, there was also mild syringomyelia. Diffusely increased meningeal contrast enhancement of the entire lumbar spinal cord was also present (Figure 1a–e).

A CT study of the thorax, abdomen and vertebral column was performed immediately after the MRI study to further assess the osseous structures involved and for staging purposes, using an 80-slice CT scanner (Aquilion prime, Canon Medical Systems). No injection of iodinated contrast medium was performed due to recent injection of gadolinium. Lung (thorax), bone (vertebral column) and soft tissue (thorax, vertebral column and abdomen) reconstructions were performed (Figure 2). The CT study revealed bilateral cortical and subchondral osteolysis of the zygapophyseal joints (or facets) of T12–T13, centred on the joint space. The intracanal and paravertebral soft tissue structures did not show any abnormalities. The rest of the axial skeleton was unremarkable. The thoracic and abdominal structures were unremarkable (Figure 2).

Cerebrospinal fluid was obtained from the lumbar cistern. Cytological examination revealed neutrophilic to mixed increased cellularity. No infectious agents were detected. Due to the insufficient sample volume, manual cell count and protein concentration could not be measured. Cytological examination of the epaxial musculature above T12–T13

LEARNING POINTS/TAKE HOME MESSAGES

- Epidural idiopathic sterile pyogranulomatous steatitis (EISPS) should be considered as a differential in young cats with acute onset of progressive and painful T3–L3 myelopathy.
- EISPS should be considered as a differential diagnosis in young cats with magnetic resonance imaging or computed tomography imaging findings compatible with an extradural mass compressing the spinal cord.
- Surgical treatment should be considered to achieve a definitive diagnosis and successful treatment in cats with EISPS, which can lead to successful long-term outcomes.

(obtained via ultrasound-guided fine needle aspirate) revealed moderate, pyogranulomatous to mixed inflammation with mesenchymal reactivity and evidence of acute and chronic haemorrhage. No bacteria were seen on direct smear and no bacterial growth was yielded when cultured. Serology for *Toxoplasma gondii* immunoglobulin G (IgG) and IgM and feline coronavirus was within reference range. Urine culture yielded no growth.

DIFFERENTIAL DIAGNOSIS

Based on the clinical history and signalment of the cat and initial diagnostic findings, sterile pyogranulomatous inflammation of the epidural fat was considered the main differential diagnosis. Other differential diagnoses included inflammatory/infectious diseases (arthritis of T12–T13 articular process joints and focal epidural empyema) or, less likely, neoplasia.

TREATMENT

The cat underwent surgical decompression via T12–T13 dorsal laminectomy, sparing the articular facets on both sides. It was elected to proceed with this approach because the compressive material was located dorsal to the spinal cord. The epidural fat at that level was more prominent, displayed a slightly discoloured appearance and indeed caused dorsal spinal cord compression. This fatty tissue was removed and the spinal cord was visibly bruised (red discoloured). Complete decompression was achieved. Samples from the epaxial musculature, T12–T13 dorsal lamina and epidural fat were obtained. Histopathological examination revealed subacute pyogranulomatous diffuse and severe steatitis and chondritis (Figure 3). Special staining with periodic acid–Schiff, Gram and Ziehl–Neelsen did not reveal presence of bacterial (including *Mycobacterium* sp.) or fungal organisms. No aerobic or anaerobic bacterial growth was obtained after 24 h of culture of these tissues. Postoperative care consisted of multimodal analgesia (opioids for 3 days and meloxicam for 10 days) and antibiotics treatment (amoxicillin-clavulanic acid [13 mg/kg, PO, q 8 h], metronidazole [10 mg/kg, PO, q 12 h] and marbofloxacin [2 mg/kg, PO, q 24 h]).

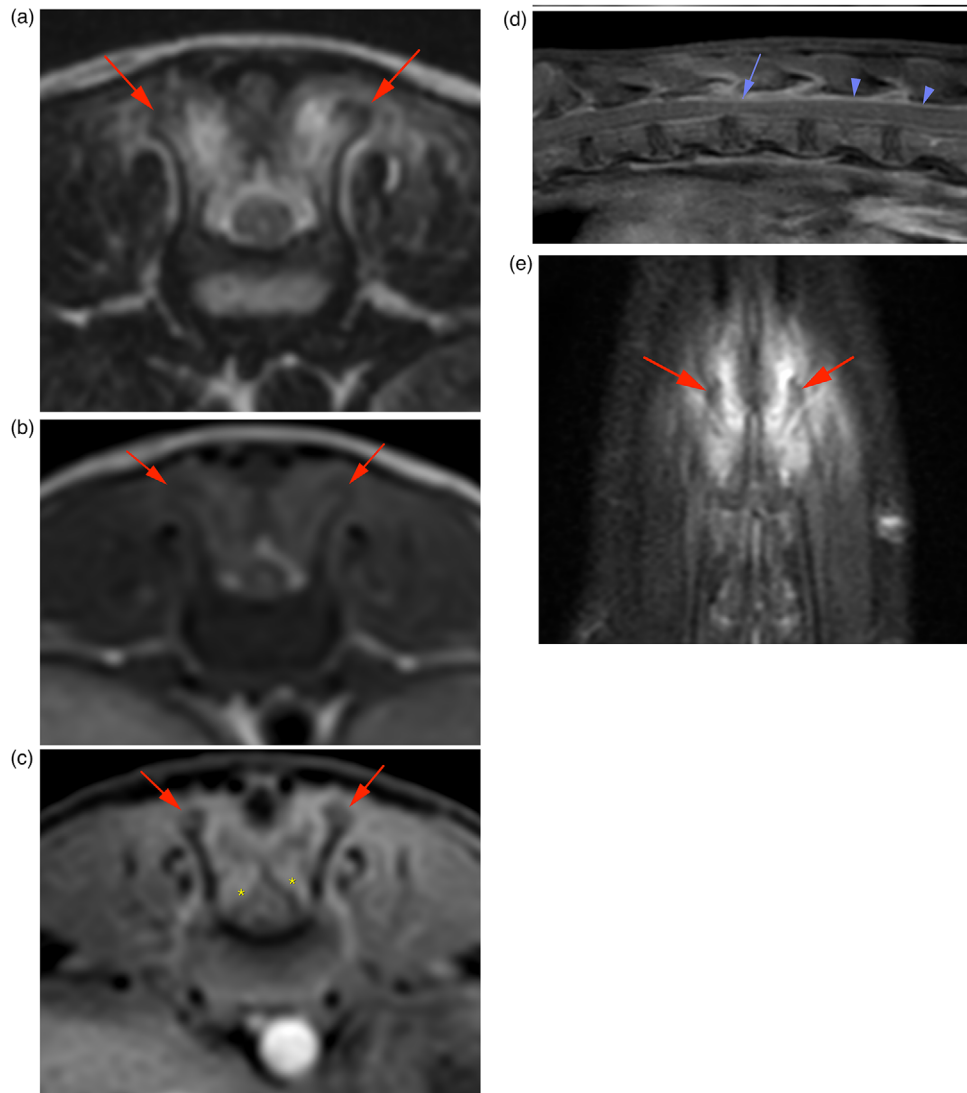


FIGURE 1 T2-weighted (T2w) transverse (a), T1-weighted (T1w) transverse pre-contrast (b), T1w mDIXON transverse post-contrast (c), T1w mDIXON parasagittal post-contrast (d) and T2w mDIXON dorsal (e) centred on T12–T13. The articular processes (red arrows) and peripheral soft tissues are hyperintense on T1w and T2w sequences and show strong contrast enhancement. Abnormal material protrudes bilaterally within the dorsolateral part of the vertebral canal (yellow stars and blue arrow). There was increased meningeal contrast enhancement (blue arrowheads).

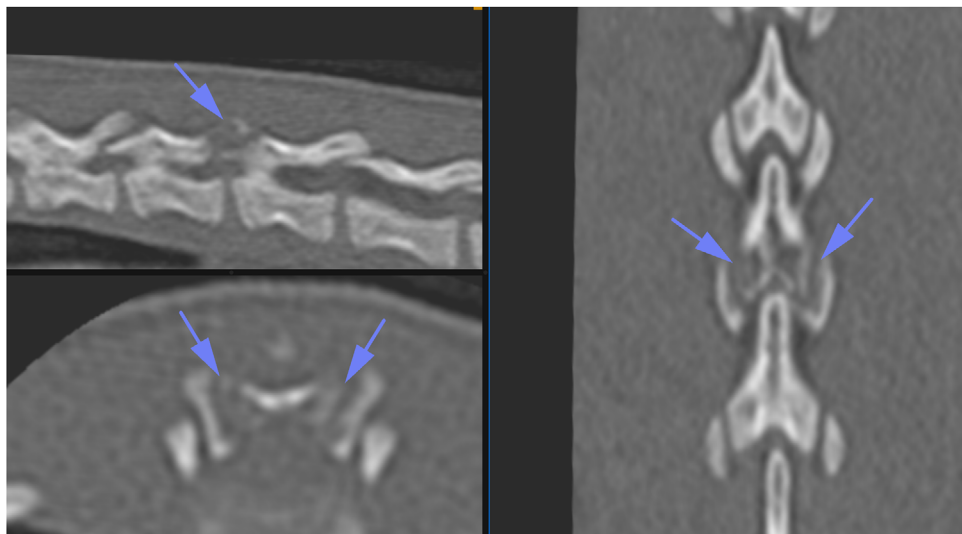


FIGURE 2 Multiplanar reconstruction of computed tomography (CT) images of the vertebral column on bone reconstruction using a bone window (working length: 450, window width: 4500). There is lysis of the articular processes at T12–T13, mostly involving the subchondral bone (blue arrows).

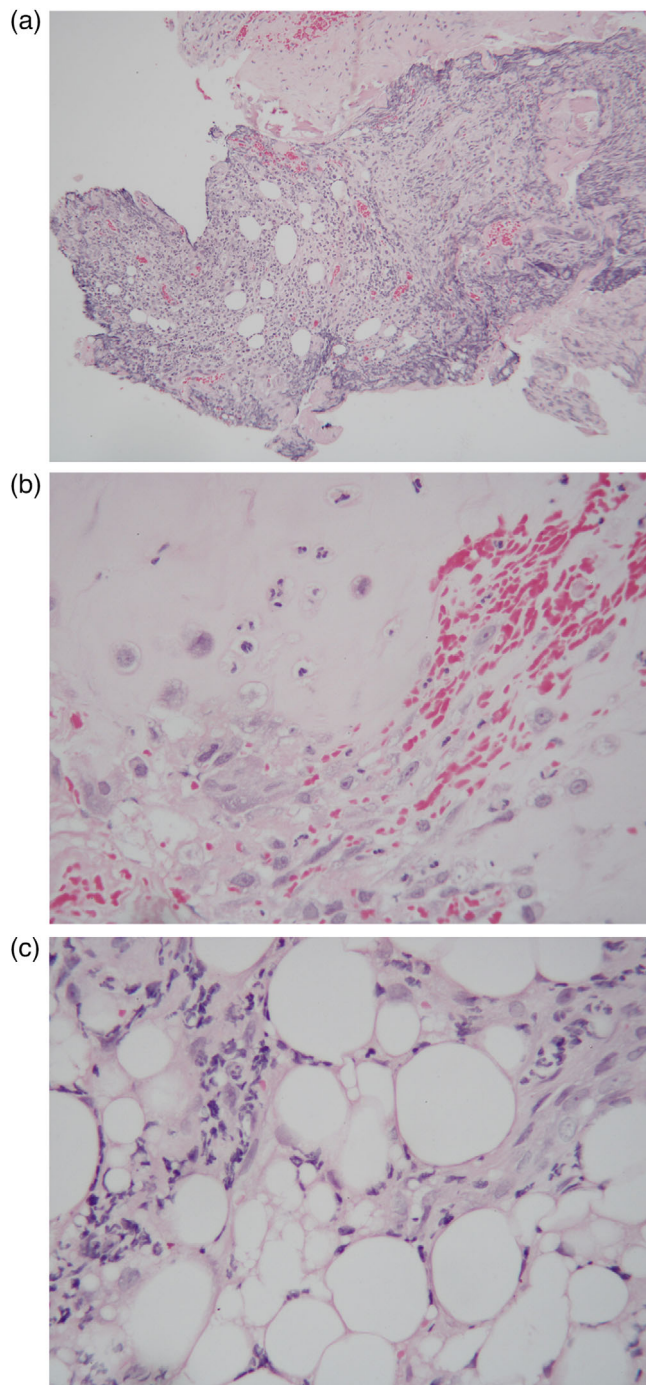


FIGURE 3 Histopathology of the surgically excised epidural material. (a) Low-magnification view of the severe sterile inflammation effacing the epidural fat and dissecting fragments of cartilaginous material and fragments of lamellar bone. (b) Closer view of portions of cartilaginous tissue infiltrated by neutrophils (asterisk). (c) Close-up view of the inflammation infiltrating the epidural adipose tissue. Haematoxylin and eosin staining: 40 \times (a) and 400 \times (b and c) magnifications.

OUTCOME AND FOLLOW-UP

The cat was discharged five days after surgery, being non-ambulatory paraparetic and able to urinate and defecate. Exercise restriction for 4 weeks and physical therapy were prescribed.

At re-evaluation 4 weeks after surgery, the cat was ambulatory, although moderate paraparesis and marked pelvic limb proprioceptive ataxia were noted.

Eight weeks after discharge, the cat was ambulatory with mild pelvic limb proprioceptive ataxia. Metronidazole, marbofloxacin and amoxicillin-clavulanic acid were discontinued 8, 10 and 16 weeks after diagnosis, respectively. After a phone conversation 8 months after the surgery, the owners reported an almost complete resolution of the clinical signs, with only mild ataxia of the pelvic limbs.

DISCUSSION

Although the most common feline spinal disorders are non-lymphoid neoplasia, followed by intervertebral disc disease, fracture/luxation, ischaemic myelopathy and feline infectious peritonitis virus myelitis,¹² the young cat in the present report had subacute pyogranulomatous diffuse and severe steatitis and chondritis. In veterinary medicine, only one case of spinal idiopathic sterile pyogranulomatous inflammation has been documented in cats⁶; however, our study is the first to describe the CT findings with a longer follow-up and screening to rule out underlying causes (inflammatory, infectious and immune-mediated diseases) in this species.

CT or MRI could be useful to provide more detailed information about EISPS. In this report, findings on MRI revealed hyperintensity of the lesion on T2w and T1w images, showing mild to moderate contrast enhancement. Generally, differential diagnoses for hyperintense lesions on both sequences include haemorrhage and fatty lesions such as lipoma. Fat suppression image may be useful to distinguish adipose tissue from other types of tissue.^{3,13} In our case, the signal of the tissue present at the location of the epidural fat was not suppressed on sequence with fat suppression. This is due to the fact that oedema and inflammation caused by EISPS change the composition of the epidural fat, preventing adequate suppression of its signal. Histological examination revealed a pyogranulomatous myositis and steatitis of the epidural fat. Therefore, definitive diagnosis was based on histopathology examination of the tissue.

In the case described herein, the combination of findings on MRI, CT, cytological, histopathological evaluation and culture was paramount in establishing a definitive diagnosis. CT findings of EISPS may be similar to other inflammatory/infectious (vertebral osteomyelitis or discospondylitis) or neoplastic spinal diseases. For the initial diagnosis of vertebral osteomyelitis in humans and discospondylitis in small animals, although MRI is the gold standard, CT can demonstrate many of the same findings as radiographs and even MRI, particularly when intravenous contrast is used to better assess the soft tissues.^{14–16} Some of the most common CT features of these conditions (vertebral bone lysis) are similar to those seen in the cats with EISPS presented herein. In this case, the lysis involved the subchondral bone of the articular processes, whereas in canine and feline discospondylitis and in human vertebral osteomyelitis, the lesions are mostly found affecting the vertebral body.

The outcome of EISPS is considered favourable after complete removal of the compressive tissue with or without systemic corticosteroid therapy.¹ Six studies found that EISPS caused spinal cord compression in 11 dogs and one cat.^{1–3,6,8,9} All of them except one dog⁴ experienced a positive neurologic outcome after decompressive surgery, three of them did

not receive corticosteroid therapy and achieved good recovery after the surgical procedure.^{3,5,6}

Our case was not treated with glucocorticoids because, although the cultures yielded no growth, the samples were obtained after the initiation of intravenous antimicrobial therapy, which may have yielded a false-negative result. For this reason, the antimicrobial treatment was continued.

In conclusion, the clinical and imaging findings in the present report increase the awareness of EISPS as a possible differential diagnosis in young cats with signs of acute onset of thoracolumbar myelopathy and spinal hyperesthesia. Advanced imaging, decompressive surgery and histopathological analysis helped achieve a definitive diagnosis and successful treatment in this case.

AUTHOR CONTRIBUTIONS

Case management: Rita Gonçalves and Emili Alcoverro. *Executing and analysing the MRI and CT:* Jeremy Mortier. *Analysing the histopathological samples:* Emanuele Ricci. *Case revision, bibliographic revision and writing of the paper:* Ana Martinez, Emili Alcoverro and Jeremy Mortier. *Manuscript revision:* Ana Martinez, Jeremy Mortier, Rita Gonçalves, Emanuele Ricci and Emili Alcoverro.

CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

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ETHICS STATEMENT

No ethical approval has been required for this study, as it was not experimental. Written informed consent has been taken beforehand from the owners.

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IMAGE QUIZ

Figure 1d

T1-weighted (T1w) mDIXON parasagittal post-contrast: contrast enhancement of the articular processes (red arrows) and peripheral soft tissue. Abnormal material protrudes within the dorsal part at the vertebral canal. There was increased meningeal contrast enhancement.

MULTIPLE CHOICE QUESTION

What is the likely diagnosis?

POSSIBLE ANSWERS TO MULTIPLE CHOICE QUESTION

- Lymphoma
- Feline infectious peritonitis virus myelitis
- Epidural idiopathic pyogranulomatous steatitis
- Haemorrhage

CORRECT ANSWER

Epidural idiopathic pyogranulomatous steatitis.

The fat signal does not suppress STIR (short tau inversion recovery) sequences. This makes an epidural idiopathic pyogranulomatous steatitis a differential diagnosis. Oedema and inflammation may change the composition of the epidural fat and this may prevent a full suppression of its signal on magnetic resonance imaging sequences such as STIR.