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CASE REPORT

Zoo animals

Spinal epidural empyema in a colobus monkey

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Abstract

A 10-year-old, male colobus monkey presented with a kyphotic appearance. Lumbar spinal pain was suspected, and the colobus was started on a course of meloxicam. With no improvement noted, it was sedated for examination. Haematology revealed a neutrophilia. An empirical course of clavulanate potentiated amoxicillin was started. With no improvements seen after a 10-day course, clinical examination was repeated. Abdominal ultrasound and abdominal radiographs revealed an abdominal effusion near the lumbar vertebrae, and haematology revealed a neutrophilia with toxic neutrophils. An enrofloxacin course was started. Three days later, the colobus presented with acute paraplegia with neurolocalisation to the T3–L3 spinal cord segments. A magnetic resonance imaging of the vertebral column was performed. Magnetic resonance imaging revealed multifocal osteolytic vertebral body lesions, compression fractures of multiple vertebral bodies and ventral spinal cord compression. Differential diagnosis included infectious osteomyelitis with spinal epidural empyema or less likely, neoplasia. Due to the poor prognosis, euthanasia was elected.

KEYWORDS
neurology, primates, wildlife, zoo animals

BACKGROUND

Spinal epidural empyema (SEE) are bacterial infections that cause purulent material to accumulate in the epidural space of the vertebral canal, but they do not possess a capsule.[1–3] SEEs have been observed in humans, dogs and cats. Clinical signs might include anorexia, lethargy, pyrexia, and most frequently observed is spinal pain. Signs then can progress to neurological dysfunction such as paresis and plegia.[1,3,5]

As far as the authors are aware, this is the first recorded case in the literature of SEE in a colobus monkey (*Colobus guereza*) and this case highlights the clinical presentation, diagnostic imaging and histopathological features of the disease syndrome in this species.

CASE PRESENTATION

A 10-year-old, male, entire colobus monkey (*Colobus guereza*) initially presented to the veterinary team at Safari West in January 2022 with a mildly kyphotic appearance and slower movement. Due to the kyphotic appearance in conjunction with the slow movement, lumbar spinal pain was suspected, and the monkey was started on a meloxicam course (0.2 mg/kg per os [PO orally] every 24 hours) for 1 week. After 1 week, a very mild improvement was seen in its mobility. With no significant improvement noted, the colobus was sedated for a physical exam and initial diagnostics, which included haematology and serum biochemistry, radiographs, urinalysis and an abdominal ultrasound.

INVESTIGATIONS

A full physical exam was performed and revealed no significant abnormalities. Haematology and serum biochemistry showed a mild mature neutrophilia and monocytosis in addition to a mildly elevated gamma-glutamyl transferase (GGT). Due to the mild neutrophilia and monocystosis, the colobus was started on a trial course of clavulanate potentiated amoxicillin (clavamoxx) (11 mg/kg PO every 12 hours) and was continued on the meloxicam course (0.2 mg/kg PO every 24 hours). Radiographs of the vertebral column, thorax and abdomen were taken, and no abnormalities were observed. An abdominal ultrasound was performed, and no abnormalities were seen. A urinalysis via cystocentesis was completed. Other than occasional ammonium phosphate and calcium oxalate crystals, the urinalysis was within normal limits. Mild improvements were seen in the colobus's mobility during the first week of the meloxicam and clavamoxx treatment;

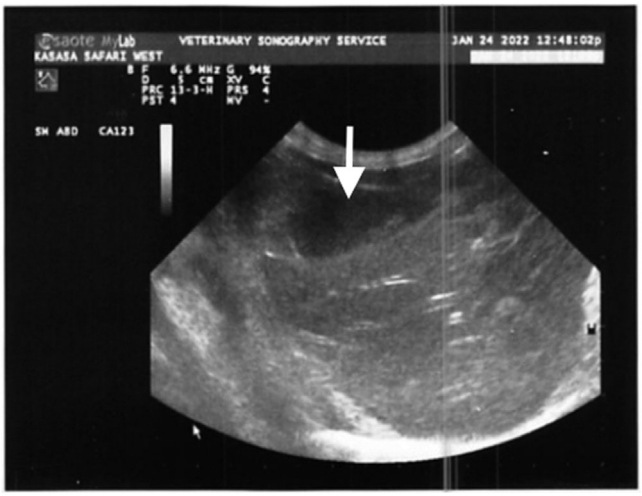

FIGURE 1 An ultrasound image showing the small abdominal effusion (arrow). The effusion was observed in the dorsocranial abdomen, near the liver, and below the lumbar vertebrae.

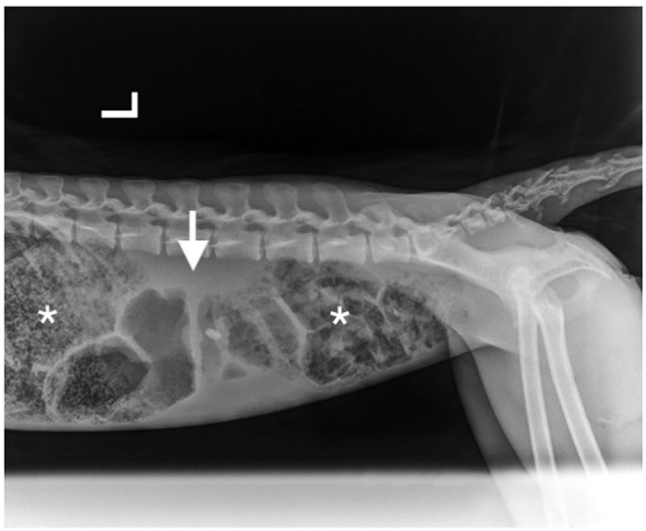

FIGURE 2 An abdominal radiograph of the colobus in left lateral recumbency showing the abdominal effusion ventral to the lumbar vertebral column (arrow). There is also a distended stomach full of food material as well as gaseous distension of the gastrointestinal tract observed (asterisks); however, these are all normal in this species.

however, symptoms worsened during the second week of treatment. Its gait appeared slower than previously and it was not as active in its environment.

With a continued decline, the colobus was sedated for additional diagnostic tests. Repeat haematology and serum biochemistry showed a normal neutrophil count, but with slightly toxic neutrophils and a mild monocytosis. A mildly elevated GGT was also seen, but this was lower overall in comparison to the previous blood sample. A full-body ultrasound was performed. On abdominal ultrasound, a small abdominal effusion was seen near the lumbar vertebrae (Figure 1). Additional abdominal radiographs were taken, which also showed the abdominal effusion (Figure 2). Abdominocentesis was performed. The effusion returned as a protein-rich transudate. Additional diagnostics included a faecal culture toxin panel. No *Salmonella*, *Shigella*, *Plesiomonas*, *Edwardsiella*, *Aeromonas*, *Campylobacter* or *Yersinia* were isolated on the faecal culture. A faecal flotation was performed as well, and the results found few *Trichuris* spp. A deep pharyngeal swab was also taken and cultured. The culture grew few *Enterobacter* and a mild amount of *Escherichia coli*, in addition to normal flora. Due to toxic neutrophils being noted microscopically on the blood samples, the monkey was started on enrofloxacin (5 mg/kg PO once a day) in addition to the meloxicam. The clavamoxx course was discontinued.

Three days later, the colobus presented with acute paraplegia. On examination, it appeared lethargic. Neurological examination revealed paraplegia with absent nociception on both pelvic limbs. The thoracic limbs were normal on examination. The neurolocalisation was to T3–L3 spinal cord segments. At this stage, the most likely differential diagnoses included meningiomyelitis, intervertebral disc herniation and neoplasia.

Two days later, the colobus was taken for magnetic resonance imaging (MRI) at a local specialty and emergency veterinary hospital. MRI of the entire vertebral column included T2-weighted (W), T1W (pre- and post-contrast), short tau inversion recovery sequences in sagittal, transverse and dorsal planes. MRI revealed multifocal osteolytic vertebral body lesions of T9–L4 and L7 vertebral bodies. Pathological compression fractures of T10 and T11 vertebral bodies were observed. There was also ventral spinal cord extradural compression at the level of T10, T11, L2 and L4, with the compression at L2 being the most significant and consistent with epidural empyema (Figure 3).

LEARNING POINTS/TAKE-HOME MESSAGES

- Spinal epidural empyema should be considered a differential diagnosis for any primate that presents with spinal pain and progressive myelopathy.
- Magnetic resonance imaging should be performed as soon as possible to obtain an early and accurate diagnosis. Surgical decompression and drainage as well as appropriate antibiotic therapy should be considered as treatment options based on clinical signs and magnetic resonance images.
- Quick action for diagnosis and treatment should be made a priority to ensure the likelihood of successful treatment.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis based on the signalment, history and MRI findings included infectious osteomyelitis with SEE with pathological vertebral fractures, or less likely metastatic neoplasia.

TREATMENT

Due to the severe neurological status (paraplegia with absent nociception) and the MRI features, the prognosis was considered very poor. Therefore, euthanasia was recommended. The colobus was euthanased after the MRI.

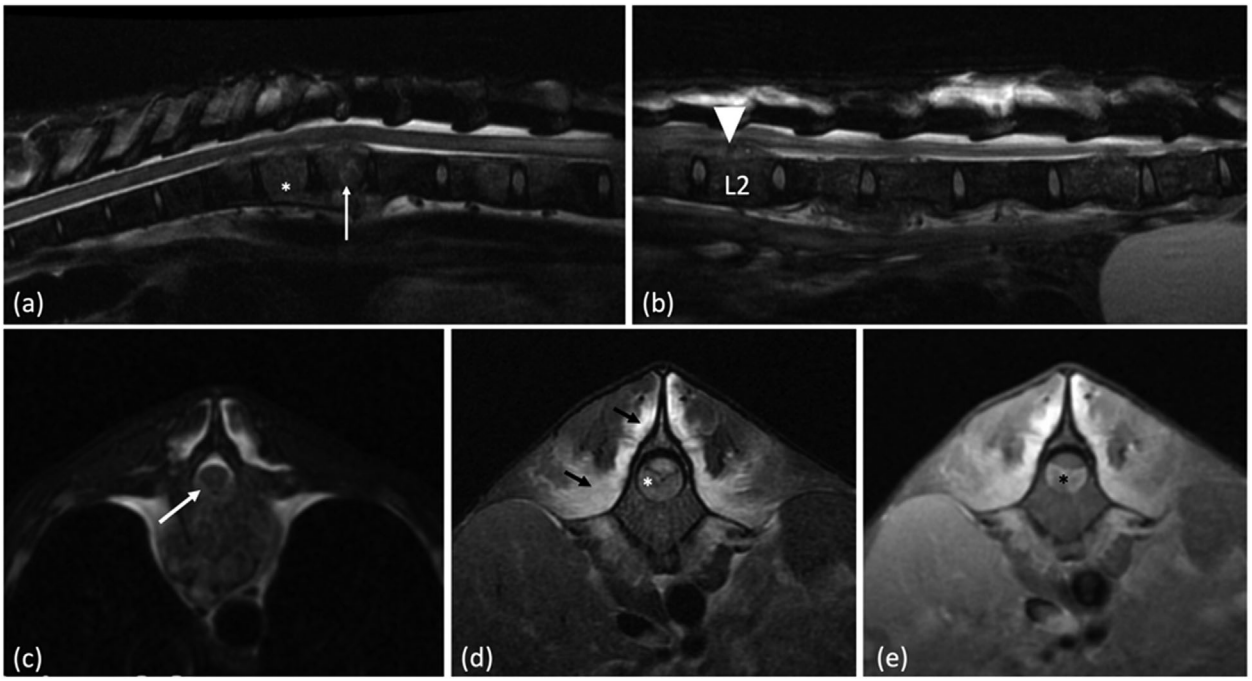

FIGURE 3 A magnetic resonance image of the thoracolumbar vertebral column of the colobus monkey. (a and b) Lateral fat-suppressed T2-weighted (T2W) images of the thoracolumbar vertebral column showing the compressive pathological fractures at T10 (white asterisks) and T11 (white arrow) vertebrae. There is extradural material hyperintense to normal spinal cord being more prominent at the level of L2 vertebra (arrowhead). (c) Transverse fat-suppressed T2W image at the level of T11 vertebra showing the extradural material (white arrow). (d) Transverse fat-suppressed T2W image and (e) transverse T1W post-contrast image at the level of L2. There is severe spinal cord compression caused by the epidural material (hyperintense on T2W image [white arrow] with marked contrast enhancement [black arrow]). The epaxial muscles show marked hyperintensity on fat-suppressed T2W image (black arrows) with also marked contrast enhancement.

OUTCOME AND FOLLOW-UP

The colobus was submitted for pathological examination. On gross pathologic exam, the lesions affecting the vertebrae and spinal cord were identified as vertebral body abscesses and epidural empyema. Necrosuppurative osteomyelitis affecting the vertebrae was observed (Figure 4). In addition, severe compressive myelopathy of the lumbar spinal cord secondary to epidural empyema was observed at the level of L1 and L2 vertebrae. Further findings included multifocal fibrinosuppurative necrosis of the spleen, necrosuppurative hepatitis, multifocal plasmacytic lymphocytic interstitial and perivascular myositis, widespread microvascular fibrin thrombi and vascular necrosis and vasculitis, and disuse atrophy of the pelvic limbs. A culture of the necrosuppurative tissue in the vertebral canal was performed. This culture yielded no bacteria. A culture of the splenic necrosis was also performed. This culture yielded *Enterobacter* species as well as *Yersinia pseudotuberculosis*. Histopathology on all other organs was performed, which yielded no bacteria.

DISCUSSION

SEE are bacterial infections that cause accumulation of purulent material in the epidural space of the vertebral canal without the presence of a capsule.[1–3] In dogs, these infections can occur via haematogenous spread, direct extension, postoperative wound infections, epidural catheters, traumatic penetrating wounds or lumbar puncture sites.[1,3,4] In dogs, clinical signs include apparent spinal pain, pyrexia, lethargy and anorexia. Signs then progress to neurological dysfunction such as paresis, plegia and sometimes incontinence.[1,3,5] Cultures of the SEE seen in dogs have revealed *Enterobacter cloacae*, *Staphylococcus*, *Pasteurella multocida* and *E. coli*.[4]

In humans with SEE, the bacteria gain access to the epidural space via haematogenous spread or direct access to the vertebral canal.[4,6,7] Risk factors include immunocompromised states such as diabetes mellitus or chronic kidney failure, bacteremia, intra-venous drug use, or through procedures involving the vertebral canal such as epidurals or central nervous system surgery.[6,7] In humans, symptoms start with fever and back pain and can progress to neurologic deficits, paraplegia, sepsis and death. The majority of human SEEs are caused by *S. aureus*, but can also be seen with gram-negative bacteria such as *E. coli* and *Enterobacter* species.[7]

In humans and dogs, conservative and surgical management are considered for treatment.[1–3,6,7] Conservative management includes epidural pus drainage, if possible, and appropriate antibiotic therapy.[3] In the cases where the epidural pus cannot be drained, a trial course with wide-spectrum antibiotics is initiated. Surgical management includes emergency surgical decompression and drainage of the epidural space, as well as prolonged course of antibiotics based on culture and sensitivity of the organism is performed.[3,6,7] In dogs, both surgical and conservative management were found to be effective treatments, regardless of clinical severity at presentation.[3] In humans, surgical management appears to be the preferred choice of treatment when the patient has neurological deficits.[6,7] In humans, it was found that if there is a delay in the diagnosis of SEE, permanent neurologic deficits, including irreversible paraplegia, can occur.[6–8]

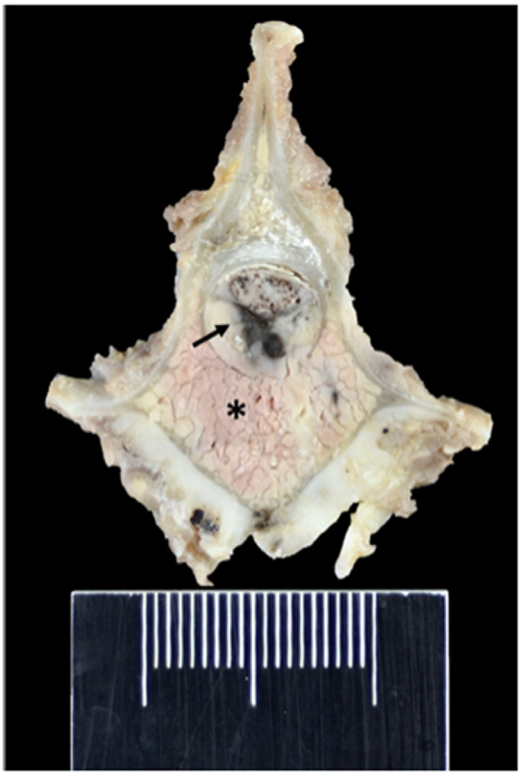

FIGURE 4 A cross-section of the L2 vertebrae on postmortem examination of the colobus. The vertebral body is highly irregular and has a moderate amount of light pink (asterisk), opaque, purulent material within it. There is extradural material (arrow) severely compressing the spinal cord, consistent with spinal extradural empyema.

This colobus presented in a very similar manner to both human and canine cases. The symptoms started with lumbar spinal pain; however, no pyrexia was noted. The symptoms then progressed to paraplegia with absent nociception. However, as the lesions in the colobus monkey did not yield any bacteria on culture, it was difficult to ascertain which bacteria caused the epidural empyema and how it arose. It is possible that the culture was negative due to the fact that the colobus had been on several weeks of antimicrobials before postmortem examination. To our knowledge, this monkey did not have any current medical issues and was not immunocompromised. The patient did not have any recent procedures that involved intravenous or vertebral canal access. Pharyngeal swab cultures did find both *E. coli* and *Enterobacter*; however, these bacteria are common commensals of the gastrointestinal tract in primates.[9,10] These commensals could have spread haematogenously to the vertebrae and led to the osteomyelitis and epidural empyema. As *Enterobacter* was found on faecal culture as well as in the spleen, it is suspected that the *Enterobacter* was the primary pathogen involved in the epidural empyema as well as the suspected acute septic event that followed (based on the findings of widespread acute thrombi and necrosis).

AUTHOR CONTRIBUTION

Emily Cehrs was the clinician responsible for this case and wrote the paper. Elsa Beltran advised and edited the paper.

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CONFLICT OF INTEREST STATEMENT

The authors declare they have no conflicts of interest.

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The authors received no specific funding for this work.

ETHICS STATEMENT

As this is a clinical case, this report does not require any ethical approval.

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REFERENCES

1. Monteiro SR, Gallucci A, Rousset N, Freeman PM, Ives EJ, Gandini G, et al. Medical management of spinal epidural empyema in five dogs. J Am Vet Med Assoc. 2016;249:1180–6.
2. Lavely JA, Vernau KM, Vernau W, Herrgesell EJ, Lecouteur RA. Spinal epidural empyema in seven dogs. Vet Surg. 2006;35:176–85.
3. Laws EJ, Sanchez L, Beltran E, Dominguez E, Ekiri AB, Brocal J, et al. Multicenter study of clinical presentation, treatment, and outcome in 41 dogs with spinal epidural empyema. Front Vet Sci. 2022;9:813316. https://doi.org/10.3389/fvets.2022.813316
4. De Stefani A, Garosi LS, McConnel FJ, Llabres Diaz FJ, Dennis R, Platt SR. Magnetic resonance imaging features of spinal epidural empyema in five dogs. Vet Radiol Ultrasound. 2008;49:135–40.
5. Cherrone KL, Eich CS, Bonzynski JJ. Suspected paraspinal abscess and spinal epidural empyema in a dog. J Am Anim Hosp Assoc. 2002;38:149–51.
6. Ameer MA, Knorr TL, Mesfin FB. Spinal epidural abscess. In: StatPearls [internet]. Treasure Island (FL): StatPearls Publishing; 2022.
7. Van Baarsel ED, Kesbeh Y, Kahf HA, Patel V, Weng B, Sutjita M. Spinal epidural abscess secondary to gram-negative bacteria: case report and literature review. J Community Hosp Intern Med Perspect. 2020;10:60–4.
8. Babu JM, Patel SA, Palumbo MA, Daniels AH. Spinal emergencies in primary care practice. Am J Med. 2019;132(3):300–6.
9. Zaniolo MM, Carvalho dos Santos I, Barbosa LN, Pachaly EMV, Caetano ICDS, Lopes KFC, et al. Antimicrobial resistance and extended-spectrum beta-lactamase production in Enterobacteria isolated from free-living primates. Vector Borne Zoonotic Dis. 2020;20(7):513–6.
10. Zaniolo MM, De Oliveira AF, Dos Santon Tramontin R. Identification of enterobacteria in free-living nonhuman primates in an urban park in the northern region of the state of Parana, Brazil. Semina Ciencias Agrarias. 2018;39(3):1115. https://doi.org/10.5433/1679-0359.2018v39n3p1115

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