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PARESIS SECONDARY TO AN EXTRADURAL HEMATOMA IN A SUMATRAN TIGER (*PANTHERA TIGRIS SUMATRAE*)

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Abstract: A 15-yr-old female Sumatran tiger (*Panthera tigris sumatrae*) was presented to the Boren Veterinary Medical Teaching Hospital at Oklahoma State University with a 3-wk history of progressive hind limb weakness. Neurologic evaluation was limited to review of videotape that demonstrated weakness and ataxia with conscious proprioceptive deficits of the tiger's pelvic limbs. Spinal radiography demonstrated disc space narrowing, and myelography demonstrated a large extradural compressive lesion at the level of L_{2-3}. Computed tomography did not reveal bone involvement. Surgery was performed to decompress the spinal cord and obtain a definitive diagnosis. A right hemilaminectomy was performed after a dorsal approach to the lumbar spine. Histologic examination of the mass revealed a consolidated extradural spinal hematoma, presumed to be secondary to intervertebral disc herniation. Despite incomplete resection of the mass and plastic deformation of the spinal cord, the tiger returned to normal ambulation within 3 wk of surgical decompression.

Key words: Sumatran tiger, *Panthera tigris sumatrae*, paresis, disc herniation, myelography, computed tomography, hemilaminectomy.

CASE REPORT

A 15-yr-old, 86.8-kg Sumatran tiger (*Panthera tigris sumatrae*) at the Oklahoma City Zoological Park showed a sudden onset of weakness, reluctance to rise, and anorexia and was immobilized with medetomidine hydrochloride (Domitor, Pfizer, Exton, Pennsylvania 19341, USA; 0.04 mg/kg, i.m.) combined with ketamine hydrochloride (KetaFlo, Abbott Laboratories, North Chicago, Illinois 60064, USA; 2.5 mg/kg, i.m.) by blow dart (Dan-Inject®, CO2/Air Pressure Injection Pistol and Dart Syringe, distributed by Wildlife Pharmaceuticals, Inc., Fort Collins, Colorado 80524, USA). Physical examination was unremarkable. Serum biochemistry profile and complete blood count values were within normal ranges.[13] Serologic tests for feline leukemia virus (FeLV) p27 antigen, feline immunodeficiency virus (FIV), feline infectious peritonitis (FIP) virus, and *Toxoplasma gondii* antibodies were negative. Abdominal and pelvic radiographs were unremarkable except for incidental intervertebral disc mineralization at L_{2-3} and L_{6-7}.[19] Although initial evaluation failed to determine etiology, infectious, metabolic, abdominal, and coxofemoral joint diseases appeared unlikely. While still immobilized, the tiger was administered 2 L of 0.9% saline (sodium chloride injection 0.9% USP, Abbott Laboratories) with 10 ml vitamin B complex (vitamin B complex fortified, Aspen Veterinary Resources, Ltd., Kansas City, Missouri 64116, USA) divided i.v. and s.c. The effects of medetomidine were reversed with atipamezole (Antisedan, Pfizer; 0.2 mg/kg, i.m.). During the next several days, the tiger's appetite and strength steadily improved, so further diagnostics were not pursued.

Three weeks later, the tiger re-presented with weakness, reluctance to move, hind limb ataxia with crossing over of the feet, and toe dragging consistent with conscious proprioceptive deficits and was videotaped. A review of the videotape at the Boren Veterinary Medical Teaching Hospital at Oklahoma State University indicated that the neurologic signs were consistent with a thoracolumbar spinal cord lesion causing pelvic limb conscious proprioceptive deficits and weakness. The tiger was transported to the University, where general anesthesia was induced as before with medetomidine and ketamine, intubated, and anesthesia maintained using isoflurane (IsoFlo, Abbott Laboratories; 1–3%) in 100% oxygen.

Hematology and serum biochemistry values were again normal, as was an electrocardiogram. A pelvic limb demonstrated bilaterally decreased number and amplitude of motor unit potentials with some denervation (fibrillation) potentials on the right side.

Lateral and ventrodorsal spinal radiographs dem-

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onstrated mineralized intervertebral discs at L_{2-3}, L_{6-7}, and L_7-S_1. The L_{2-3} intervertebral disc space was suspected as the source of intervertebral disc disease (IVDD) due to the radiographic signs of intervertebral disc mineralization within the intervertebral space, intervertebral foramen narrowing, and collapse of the space between the articular processes. A myelogram was performed by L_{5-6} intradural puncture and administration of 0.3 mg/kg of water-soluble contrast medium (Iohexol, Omnipaque 240, Nycomed Inc., Princeton, New Jersey 08540, USA). Examination of cerebrospinal fluid obtained immediately before myelography did not indicate inflammatory or infectious processes within the central nervous system (CNS). The myelogram revealed a large, well-defined, right-sided extradural compressive lesion at L_2 (Fig. 1A, B). Computed tomography (CT) confirmed the presence of a soft tissue or fat density mass estimated to be 6 cm long and localized at L_2 and L_3 that occupied the entire right half of the vertebral canal without evidence of bone involvement (Fig. 2).

At the conclusion of the CT, apnea developed, possibly due to accumulation of contrast agent within the cranial CNS, ventilation was assisted, and surgery to decompress the lesion was postponed. Diazepam (Abbott Laboratories; 0.1 mg/kg, i.v.) was administered to reduce the likelihood of postmyelogram seizures, and atipamezole (0.2 mg/kg, i.m.) was given to reverse the medetomidine. The tiger recovered from anesthesia without further incident and was returned to the Oklahoma City Zoological Park.

The tiger's clinical signs remained stable for 3 wk, and it was then anesthetized as before. Surgery used a right dorsolateral approach to the thoracolumbar spine and hemilaminectomy (dorsolateral laminectomy) of the L_2 and L_3 vertebrae, as would be performed in a large dog,[31] to expose the vertebral canal and a large extradural mass (Fig. 3). The friable, apparently encapsulated soft mass adhered to the dura. Approximately 85–90% of the mass was dissected from the paraspinal tissues, but resection was abandoned when severe venous sinus bleeding was encountered. A partial durectomy was performed, and the mass did not appear to invade the spinal cord, which returned to a normal position within the vertebral canal after mass removal. The plastic deformation of the compressed cord segment failed to return to a normal shape. All collected paraspinal tissues were placed in 10% formalin and submitted for histologic examination. The tiger recovered from anesthesia uneventfully.

To minimize possible injury, the tiger was kept in its indoor cage without shelves or logs to climb on for 4 wk after surgery. The tiger began ambulating normally within 3 wk of surgical decompression and started jumping on shelves after 4 wk. At 7 mo after surgery, it remained clinically normal and was placed on display again.

Histologic examination of the extradural mass revealed sweeping bands of fibrous connective tissue containing disseminated infiltrates of neutrophils, hemorrhage, and numerous hemosiderophages. Scattered islands of chondroid tissue were embedded in the regions of hemorrhage and fibrous connective tissue (Fig. 4A, B). Special stains confirmed extensive mineral deposits. An extradural hematoma and inflammation containing extruded degenerate intervertebral disc material was diagnosed.

DISCUSSION

Paraparesis and ataxia are not common in domestic cats. Primary differentials for cats with neurologic signs localized to the thoracolumbar spine include trauma, vascular causes, infectious and inflammatory diseases, and neoplasia.[9,20,35] Trauma is a common cause of acute pelvic limb paresis or plegia in domestic cats and often may be associated with vertebral luxation or subluxation; fractures of the vertebral column, limbs or pelvis; or peripheral nerve damage.[23] Normal radiographs after trauma do not rule out myelopathy due to dynamic vertebral subluxation. Vascular causes of paraplegia in domestic felines include aortic thromboembolism, commonly associated with cardiomyopathy, and fibrocartilagenous embolism.[24,29,36,38] The most commonly diagnosed inflammatory or infectious causes of spinal myelopathy in domestic cats are FeLV, toxoplasmosis, and cryptococcosis,[5,14,20,24] but FIP has been reported as the cause of caudal paresis in two cats.[11] Lymphosarcoma (LSA) is the most common cause of spinal cord disease in domestic cats and has been reported in 5.3% of domestic feline cases.[1,7,8,20,22,23,34,35]

Several potential etiologies of neurologic disease have been reported in large captive felids, but clinical myelopathy has been rarely reported.[6,10,25,27] FeLV p27 antigens and anti-FIV and anti-FIP antibodies have been detected in captive African lions (*Panthera leo kruegeri*).[25]

Thoracolumbar disc disease (TLDD) is one of the most common causes of pelvic limb paresis in domestic dogs (*Canis familiaris*) but rarely presents as a clinical disease in domestic cats (*Felis cattus domesticus*).[24] Signs include paraspinal pain, pelvic limb paresis, and paraplegia. Disc degeneration in dogs is classified as either type I or type II based on metaplastic changes of the nucleus pulposus and annulus fibrosus, which lead to characteristic path-

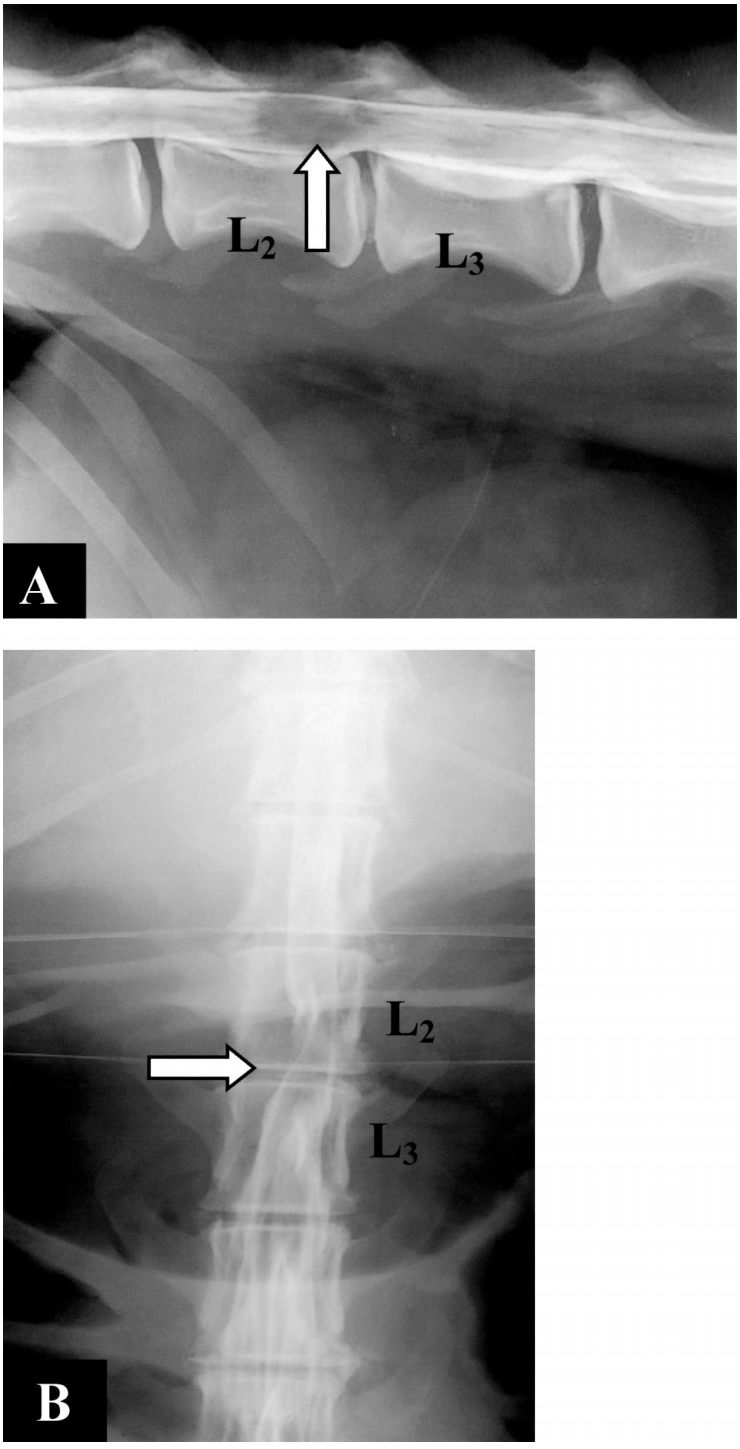

Figure 1. Right lateral (**A**) and dorsoventral (**B**) projections of a myelogram on a Sumatran tiger (*Panthera tigris sumatrae*) showing a large, right-sided extradural lesion (arrows) at L_{2-3}.

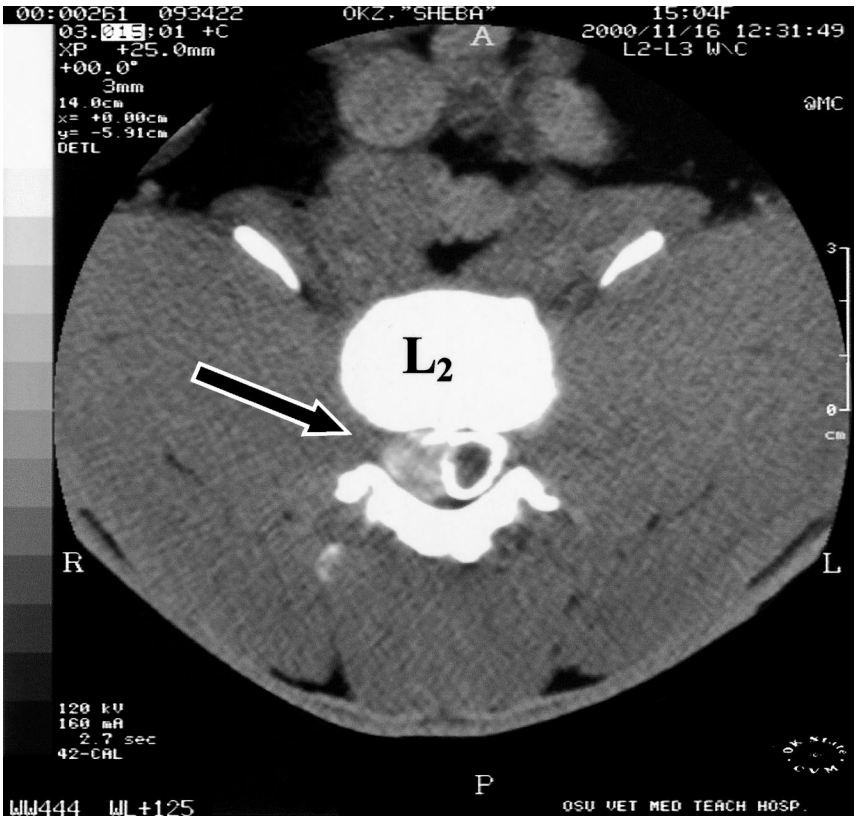

Figure 2. Intravenous contrast-enhanced cross-sectional CT at L2 of a Sumatran tiger (*Panthera tigris sumatrae*) showing a right-sided extradural lesion compressing the spinal cord (arrow) but with no bone involvement.

ophysiologic changes within the intervertebral disc.[3,24] Type I TLDD results from metaplasia of the nucleus pulposus with weakening of the dorsal annulus fibrosus, rupture, and extrusion (herniation) of nucleus pulposus into the vertebral canal. Type I TLDD occurs most frequently in young to middle aged, small, chondrodystrophic breeds such as the Dachshund and Pekingese.[24] Type II TLDD results from fibrinoid metaplasia of the nucleus pulposus with dorsal migration of the nucleus within the annulus and protrusion (bulging) of annulus fibrosus into the vertebral canal. It occurs most frequently in middle to older aged, large breeds such as the German Shepherd, Labrador Retriever, and Doberman Pinscher.[12,24] The severity of clinical neuropathy depends on neuroanatomic location, the volume and contact area of the disc protrusion or extruded nucleus, the velocity of the disc protrusion or extrusion, and the extent and duration of spinal cord compression.[12]

Although there are few reports of intervertebral disc disease (IVDD) in domestic cats, spinal degeneration with associated IVDD is commonly observed at necropsy. Type II lesions appear to be more common than type I lesions in domestic cats.[4,15,16] Spinal degeneration in cats with IVDD appears to be analogous to that seen in dogs. Disc protrusion can cause clinically significant neurologic disease in domestic cats.[2,12,14,17,28,30] The most common cause of spinal cord disease in domestic cats is LSA.[9,23,34,35]

As in domestic cats, spinal degeneration appears to be a common disease condition in large captive felids. An investigation of spinal disease in 37 large captive felids (13 lions, 16 tigers, four leopards [*Panthera pardus*], one snow leopard [*Panthera uncia*], and three jaguars [*Panthera onca*]) revealed eight cases of spinal degeneration with IVDD or spondylosis.[19] The clinical signs observed included decreased activity, rear limb muscle atrophy, and chronic or intermittent rear limb paresis. Multifocal lesions including disc mineralization, disc protrusion, and marked spondylosis were seen radiographically or on necropsy. The median age of these felids was 18 yr. The clinical and radiographic findings and the histopathologic changes appeared

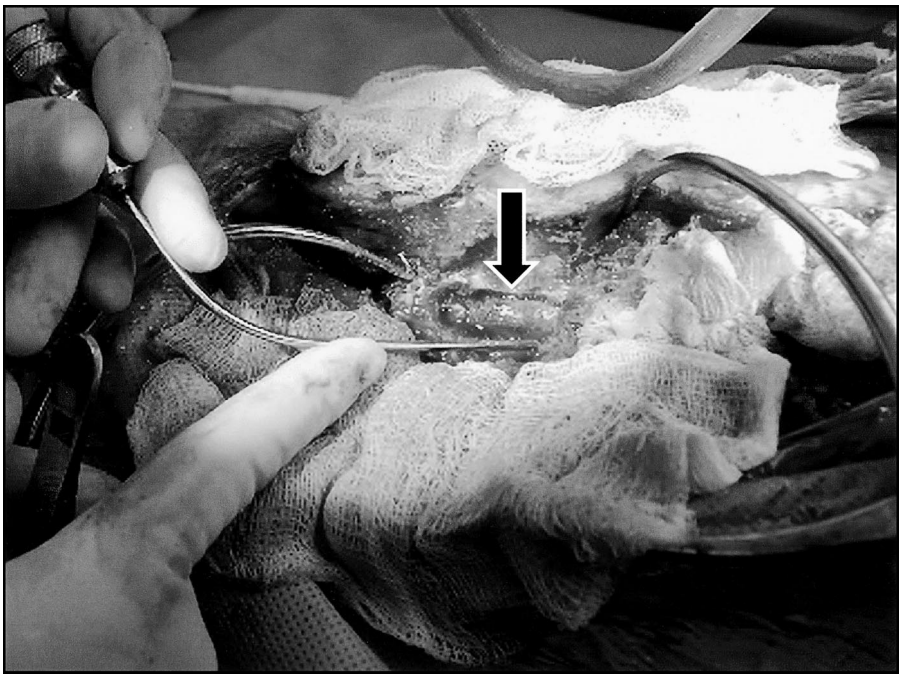

Figure 3. Intraoperative photograph showing the large extradural hematoma (arrow) exposed by dorsal hemilaminectomy of L_{2-3} in a Sumatran tiger (*Panthera tigris sumatrae*) with intervertebral disc disease.

consistent with those reported in IVDD of dogs. Medical management was attempted in two of these large felids, and long-term improvement was reported in one. One case of caudal cervical disc protrusion has been reported in a captive Bengal tiger (*Panthera tigris tigris*) that resolved favorably 6 wk after surgical intervention.[21] This animal demonstrated clinical IVDD at C_{6-7} and had a nonclinical ventral disc protrusion at L_{5-6}.

Despite spinal degeneration appearing to be a common although generally subclinical disease in domestic and large captive felids,[19] clinical TLDD has not been reported previously in large captive felids. The incidence of intervertebral disc protrusion in domestic cats has been estimated to be similar to that of nonchondrodystrophic dogs. In cats, type II IVDD from dorsal annular disc protrusion is more common than type I disc extrusion.[15] However, IVDD in domestic cats is most often nonclinical, and the intervertebral disc degeneration begins in the inner annulus as opposed to the nucleus pulposus in dogs.[12] According to one necropsy study of domestic cats, animals with spinal degeneration ranged between 10 wk and 18 yr of age.[4] The frequency of multiple lesions appears to increase with age in domestic cats.[17]

Fourteen cases of IVDD causing clinically significant myelopathy in domestic cats including both cervical and thoracolumbar myelopathy secondary to IVDD have been reported, with median age of 8 yr at presentation.[2,12,14–17,28,30,33] Diagnostic and surgical techniques, similar to those described for canine IVDD, have been used for the diagnosis and treatment of feline IVDD. The radiographic signs commonly observed in dogs with IVDD are disc mineralization, disc space narrowing, intervertebral foramina narrowing, abnormal articular process spacing, opacified mass within the vertebral canal, dorsal vertebral body osteophytoses, and spondylosis (exostoses). With the exception of intervertebral disc mineralization, theses signs are generally not seen on spinal radiographs of aged domestic cats.[12] Thus, radiographs are mostly beneficial in cases of trauma and bony neoplasia,[16,17] whereas myelography is generally indicated to localize a compressive lesion.[26,32,37] Increased detail provided by CT or magnetic resonance imaging may improve diagnostic and therapeutic decision making.[18] Conservative medical treatment of IVDD in these 14 domestic cats was largely unsuccessful (one good outcome in seven cases), whereas surgical intervention was uniformly successful (seven of seven had good outcomes). Therefore, decompressive surgery is recommended as the therapy of choice for feline TLDD.

A degenerative or traumatic type I intervertebral

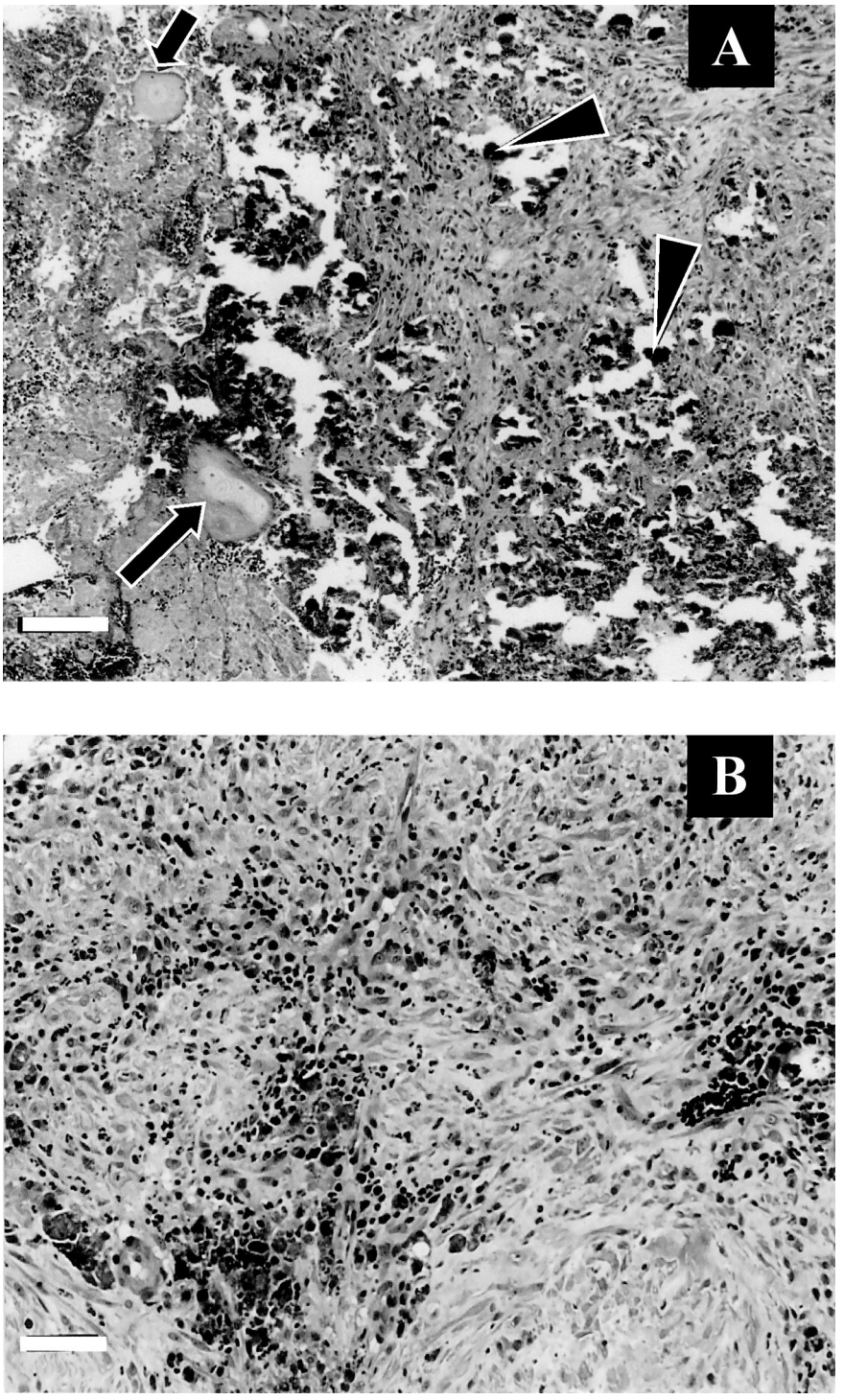

Figure 4. **A.** Tissue removed from the L_{2-3} epidural space of a Sumatran tiger (*Panthera tigris sumatrae*) consists of an admixture of fibrous connective tissue, hemorrhage, and inflammation with islands of chondroid (arrows) and foci of mineralization (arrowheads) embedded in this milieu. H&E stain, scale bar = 75 μm. **B.** A higher magnification reveals dense fibrous connective tissue infiltrated by neutrophils, macrophages, hemorrhage, and numerous hemosiderophages. H&E stain, scale bar = 40 μm.

disc extrusion (or both) may have caused the tiger's initial, mild clinical neuropathy, which appeared to resolve. A subsequent, more severe disc extrusion may have caused a vertebral venous sinus hemorrhage resulting in extradural hematoma formation, spinal cord compression, and inflammation of paraspinal tissues with reoccurrence of more severe neurologic deficits.

CONCLUSIONS

Degenerative spinal disease, including IVDD, should be considered in older, large captive felids with decreased activity, inappetence, or hind limb weakness or ataxia,[19,21] and appropriate imaging diagnostics should be pursued when the results of initial diagnostic tests have ruled out any other causes of neuropathy. Surgical decompression offers the best prognosis for recovery from clinically evident IVDD in large felids.

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