Feline Spinal Cord Diseases

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KEYWORDS

• Spinal cord • Cat • Feline infectious peritonitis
• Lymphosarcoma • Tumor • Intervertebral disc

Our knowledge on feline spinal cord diseases has increased in recent years thanks to studies on their prevalence, and histologic and magnetic resonance imaging (MRI) characteristics[1–4]; however, the diagnosis and treatment of some spinal cord diseases, such as feline infectious peritonitis (FIP) and spinal lymphosarcoma, are still a challenge. The objective of this article is to review the recent literature that reports on the most common diseases affecting the spinal cord of cats and to draw some general conclusions that will be useful to formulate the diagnosis and prognosis for feline spinal patients. In particular, the results of a postmortem study[1] from the University of Pennsylvania that consisted of 205 cats with spinal cord diseases are compared with other retrospective studies of spinal cord disease that considered different populations of cats and different criteria for disease evaluation. In a population of 205 cats with histologic confirmation of spinal cord disease, inflammatory/infectious diseases were the most common (affecting 32% of the cats), followed by neoplastic diseases (27%), trauma (14%), congenital or inherited diseases (11%), vascular diseases (9%), degenerative diseases (6%), and metabolic/nutritional diseases (1%).[1] Each of these categories of disease is discussed herein.

INFLAMMATORY/INFECTIOUS DISEASES

Inflammatory/infectious diseases were the most common type of spinal cord disease in cats in 2 recent retrospective studies.[1,3] FIP was the most common inflammatory/infectious disease (51%) in 205 North American cats with histologic confirmation of spinal cord disease.[1] This finding was consistent with a population of 286 English cats with central nervous system (CNS) disease confirmed on postmortem in which more than 50% of cats with inflammatory disease had FIP.[3] The other inflammatory/infectious diseases reported in decreasing order of frequency were bacterial myelitis (16%), cryptococcosis (9%), unknown infectious/inflammatory diseases (8%), toxoplasmosis (6%), eosinophilic meningomyelitis (5%), and idiopathic poliomyelitis (5%).[1]

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Feline Infectious Peritonitis

FIP is a fatal disease caused by the FIP virus, a highly virulent feline coronavirus strain that induces an immune-mediated progressive polyserositis (wet form) and pyogranulomatosis (dry form).[5] Neurologic signs are usually associated with the dry or pyogranulomatous form of FIP, with ocular and/or CNS involvement reported in less than 9% of cats with the wet form and in 60% of cats with the dry form.[6] The neurologic signs are often multifocal, and common clinical signs are ataxia, depressed mental status, pathologic nystagmus, tetraparesis/tetraplegia, head tilt, and seizures.[6–8] Most deaths from FIP occur in cats between the ages of 3 and 16 months.[9] Other spinal cord diseases that cause death in cats younger than 2 years include storage diseases, bacterial myelitis, and trauma; however, these occur less frequently than FIP in young cats.[1]

On histopathology, CNS lesions are centered in the meninges, choroid plexus, and ependyma, with submeningeal and parenchymal extension.[10] Lesions are common in the posterior and ventral parts of the brain.[9] Within the spinal cord the cervical region is most commonly affected, and lesions associated with FIP were present in the cervical segments in 93% of cats with spinal FIP (**Fig. 1**).[1]

Obtaining an antemortem diagnosis of neurologic FIP is difficult. In presence of effusion, positive immunofluorescent staining of macrophages is conclusive; however, for the dry form of FIP the definitive diagnosis (based on immunohistochemistry) is not practical if the clinical signs are limited to the CNS.[10,11] Cats with FIP may present some nonspecific hematological abnormalities such as leukocytosis, lymphopenia, nonregenerative anemia, and increased total serum protein caused by hyperglobulinemia (present in about 70% of the cats with the dry form).[11] An albumin/globulin ratio of less than 0.6 is diagnostic for an inflammatory process,[12] and the most common inflammatory processes are FIP. MRI and computed tomography (CT) of the brain of cats with the neurologic form of FIP often show hydrocephalus, and on MRI, periventricular enhancement and cervical syringohydromyelia have also been reported in cases of suspected CNS FIP (see **Fig. 1**).[13–15] Spinal fluid analysis often shows high protein content and pleocytosis with a predominance of neutrophils or lymphocytes, but these findings are not pathognomonic for neurologic FIP, and in some cases cerebrospinal fluid (CSF) analysis may be normal.[6,7] Serum antibody detection is of limited

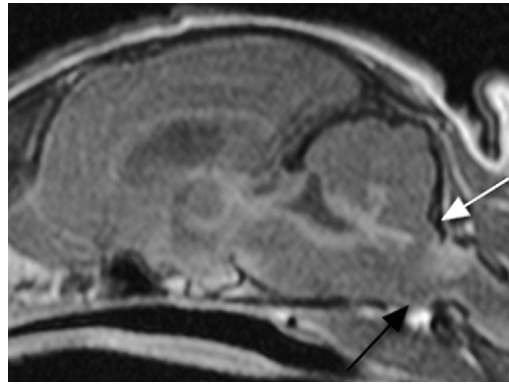

Fig. 1. Sagittal T1-weighted postcontrast image of an 11-month-old Sphynx cat with presumptive diagnosis of FIP. There is ventricular dilation and contrast enhancement associated ventricular lining, consistent with ependymitis, a focal intramedullary contrast-enhancing area at the junction between medulla and first cervical spinal cord segment (*black arrow*), and subtentorial brain herniation (*white arrow).*

help because a high percentage of healthy cats have antibodies against feline coronavirus and will never develop FIP; however, very high titers ($\geq$1:1600) are suggestive of FIP.[10] Detection of feline coronavirus in blood by polymerase chain reaction (PCR) has a high sensitivity, but low specificity, for clinical disease.[10] PCR has accurately differentiated FIP effusions from other types of effusions and can be performed on spinal fluid to diagnose the neurologic form of FIP[10]; however, the author is not aware of any study reporting the sensitivity and specificity of detection of feline coronavirus in spinal fluid by PCR.

There is no treatment with documented efficacy for FIP. Treatment with corticosteroid, cyclophosphamide, ozagrel hydrochloride, ribavirin, melphalan, tylosin, promodulin, human interferon α, feline interferon β or ω, *Propionibacterium acnes*, pentoxifylline, and polyprenyl immunostimulant, as monotherapy or in combination, have been reported, with some cats achieving a remission of the clinical signs.[16,17] In some cases, spontaneous remissions or misdiagnoses may have accounted for responses to treatment; results of some of these studies must be interpreted with caution.[16]

NEOPLASTIC DISEASES

Neoplasms are a common cause of spinal cord disease and were documented in approximately 25% of cases.[1,4] Lymphosarcoma is the most common tumor affecting the spinal cord of cats, with reported prevalence between 28% and 40%.[1,2,4] The second most common tumor was osteosarcoma, representing 27% (14/52) of nonlymphoid tumors; in the same study, glial tumors (9%) and meningioma (7%) were the third and fourth most common tumors affecting the feline spinal cord (**Fig. 2**).[2] By contrast, in 2 retrospective studies with a total of 37 cats with nonlymphoid vertebral or spinal cord tumors treated with surgical cytoreduction, meningioma was the

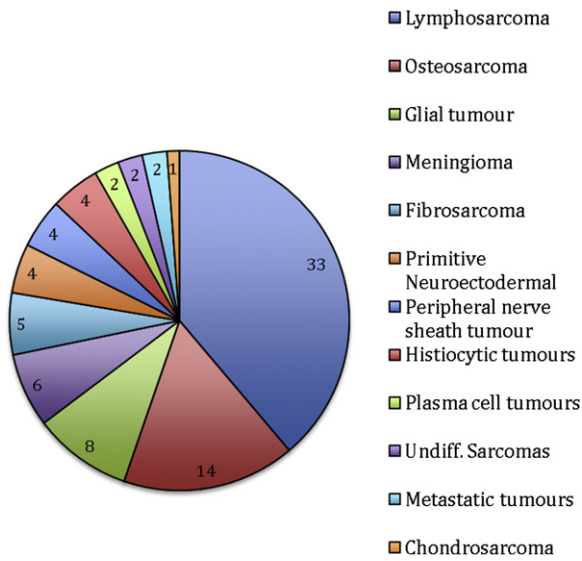

Fig. 2. Prevalence of tumors in a population of 85 cats with histologically confirmed primary or metastatic tumors of the spinal cord or causing spinal cord diseases by local extension from adjacent tissues.

most common tumor representing approximately half (45%–61%) of all the cases.[18,19] In the study by Rossmeisl and colleagues,[19] meningioma was the most common benign tumor (16/18), while osteosarcoma was the most common malignant tumor (3/8). Both the Levy and Rossmeisl studies did not include tumors that were not amenable for surgical cytoreduction, therefore they did not account for intramedullary tumors such as gliomas and primitive neuroectodermal tumors (PNET), and highly invasive and destructive tumors such as advanced osteosarcomas and fibrosarcomas.[18,19]

Lymphosarcoma

Lymphosarcoma was the most common tumor in a retrospective study of 85 cats with definitive diagnosis of spinal cord neoplasms.[2] In this study, cats diagnosed with spinal lymphosarcoma were significantly younger (mean and median age of 6 and 4 years) than cats with other spinal cord tumors (mean and median age of 9 and 10 years).[2] Cats with tumors other than lymphosarcoma had a normal age distribution, with 80% of the cats between the ages of 5 and 14 years; whereas cats with lymphosarcoma presented a bimodal age distribution, with 50% of them younger than 4 years and 25% older than 11 years.[2] The most common clinical signs of spinal lymphosarcoma in cats are progressive asymmetric posterior paresis or paralysis and spinal hyperesthesia,[2,20] as reported for other feline spinal cord tumors; however, cats with lymphosarcoma had a higher prevalence of nonspecific clinical signs such as anorexia, lethargy, weight loss, signs of respiratory tract infection, and abnormal behavior.[2] Presence of nonspecific clinical signs may be explained by immunosuppression associated with positive feline leukemia virus (FeLV) status in 56% of the cats with lymphosarcoma and the postmortem findings of lymphosarcoma involvement of extraneural organs in 85% of the cats.[2] In previous studies the percentage of cats positive for FeLV has been as high as 94%[20] and the percentage of cats with involvement of extraneural sites has been reported as between 43% and 100%.[20,21] A study found that the duration of clinical signs before diagnosis was significantly shorter in cats with lymphosarcoma than in cats with other spinal cord tumors; 93% of the cats with spinal lymphosarcoma were ill for less than 2 months before diagnosis.[2]

Spinal lymphosarcoma represents a challenge for in vivo diagnosis. A definitive diagnosis can be achieved with positive cytology on blood smears in 5% to 13% of the cases,[2,20] bone marrow aspirates in 14% to 67%,[2,22] and spinal fluid analysis in 9% to 35%.[2,20] The variability of positive results in the 3 studies considered is difficult to explain; all 3 studies are retrospective postmortem studies with a similar number of cases,[23–33] and it is possible that the low number of cases is responsible for the different percentages of in vivo tests with a positive result. Combination of multiple diagnostic tests, such as spinal fluid analysis and bone marrow aspirate, may provide a higher diagnostic yield. However, the diagnostic path most likely to provide a definitive in vivo diagnosis of spinal lymphosarcoma appears to be identification of intra- or extraneural masses by physical examination and diagnostic imaging (thoracic radiographs, abdominal ultrasound, spinal radiographs, and MRI or myelography), and fine-needle aspiration (FNA) or biopsy of the mass when possible.[2]

Kidney and bone marrow are the most common extraneural location of spinal lymphosarcoma, based on data from 5 publications, and their investigation may lead to a positive in vivo diagnosis of lymphosarcoma.[2,3,20–22] In cats with spinal lymphosarcoma, lymphosarcoma has been found to affect kidneys in 41% to 100% of cats,[2,20] and bone marrow in 45% to 54% of the cats.[2,22] In the study by Marioni-Henry and colleagues,[2] lymphosarcoma was also found in liver (36%),

skeletal muscle (32%), spleen or lymph nodes (27%), and vertebrae or heart (18%). Within the CNS, lymphosarcoma tends to affect multiple regions of the spinal cord, especially thoracic and lumbosacral, and brain; based on 2 postmortem studies, 31% to 43% of cats with spinal lymphosarcoma had also brain involvement.[2,3] Spinal lymphosarcoma may have an exclusively extradural location, which has been reported in 85% to 96% of the cases in publications from the late 1970s and early 1990s[20–22]; however, most recent publications estimate 34% to 38% of feline spinal lymphosarcoma having an exclusive extradural location, with the majority of the cases (61% to 88%) presenting both extradural and intradural components.[2,3]

Prognosis for spinal lymphosarcoma is poor. Spodnick and colleagues[22] reported a complete or partial remission in a series of 6 cats with spinal lymphoma treated with vincristine, cyclophosphamide, and prednisone; the complete remission rate was 50% and the median duration was 14 weeks. Another cat treated with decompressive surgery and chemotherapy had a remission of 62 weeks. Lane and colleagues[20] reported on a series of 4 cats with spinal lymphosarcoma. Three cats were treated with L-asparaginase, vincristine, and prednisone following local spinal radiation, and 1 cat had surgical cytoreduction; 3 of the cats improved, and 1 was alive 13 months following presentation, but the 3 other cats were euthanized or died within 20 weeks of treatment because of systemic relapse. In the Marioni-Henry and colleagues[2] study, one cat with spinal lymphosarcoma was euthanized after recurrence of clinical signs 38 days following a single dose of local radiation therapy and treatment with prednisone, cyclophosphamide, and vincristine; another cat survived 60 days following surgical cytoreduction and treatment with prednisone, asparaginase, and cytarabine.

Osteosarcoma

Feline osteosarcomas affect more often the appendicular than the axial skeleton. In a retrospective study on feline neoplasms, 58% of the 19 cases of osteosarcoma affected the appendicular skeleton and only 2 affected vertebrae.[23] In another retrospective study of 22 feline osteosarcomas, only 32% affected the axial skeleton and none the vertebrae.[24] Nevertheless, vertebral osteosarcoma was the second most common tumor in a postmortem retrospective study on 85 cats with tumors affecting the spinal cord, affecting 14 cats,[2] and information on another 9 cats with histologically confirmed vertebral osteosarcomas has been reported.[18,19,25–28] Taken together, the 14 cats from the study by Marioni-Henry and colleagues[2] and the 9 other cats that all had vertebral osteosarcomas had a mean age of 8.3 years (median age 8 years, range 3–13 years), and included 12 males and 11 females, of which 19 were domestic short-haired (DSH) cats, 1 a Persian, and 1 an Angora cat.[18,19,25–28] The tumors affected the lumbar vertebrae in 9 cases, the thoracic vertebrae in 7 cases, the cervical region in 4 cases, and the sacrum and coccyx in 1 case each.[2,18,19,25–28] Radiographs showed a lytic lesion in 10 of 11 cases, and a pathologic vertebral fracture in 2 cases; CT revealed a pathologic fracture not visible on radiographs in 1 case. Myelography, performed in 5 cases, showed a compressive lesion with deviation or interruption of the flow of contrast in all cases.[2,26–28] MRI revealed presence of a vertebral mass in 2 cases (**Fig. 3**).[2] An FNA was performed in 3 cases; on cytology, the lesion was diagnosed as a neoplasm and an osteosarcoma in 1 case each, and in the last case the neoplasm was misdiagnosed as a lymphosarcoma.[2]

Cytoreductive surgery can prolong survival in cats with vertebral osteosarcoma, but results tend to be highly variable. Following cytoreductive surgery, 5 cats with vertebral osteosarcoma had a mean and median survival time of 145 and 88 days (range 2–518 days)[2,18,19,25–28]; 3 of these cats were part of a retrospective study of

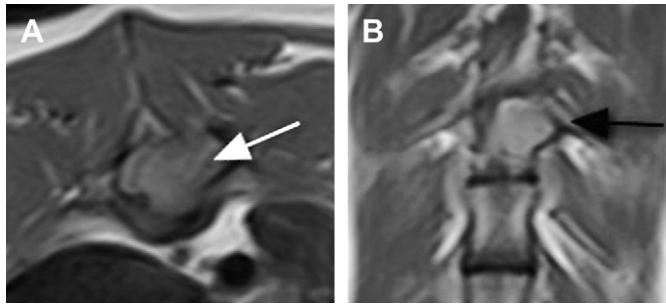

Fig. 3. Vertebral osteosarcoma in a DSH cat. Transverse T1-weighted (*A*) and dorsal T1-weighted postcontrast (*B*) images demonstrate a homogenously contrast-enhancing mass associated with the left pedicle and body of the 10th thoracic vertebra (*arrow*). (*Courtesy of* Sergio Rodenas and Sonia Anor, Neurology Service, Veterinary College, University of Barcelona.)

26 cats with nonlymphoid vertebral or spinal cord neoplasms treated surgically.[19] This study found that cytoreduction is a good palliative treatment, but the prognosis was based on the phenotype of the tumor and the surgeon's impression of a partial or complete excision; in fact, cats with malignant tumors (including the 3 osteosarcomas) were found to have a median survival time of 110.5 days versus 518 days for cats with benign tumors.[19]

Meningioma

Meningioma is the most common feline intracranial tumor, representing 58% of the cases in a recent study[29]; however, spinal meningioma represented only 7% of the cases in a study on histologically confirmed tumors affecting the spinal cord of cats[2] and 8% of the tumors of the feline spinal cord identified by MRI.[4] Based on information on 32 feline spinal meningiomas reported in the literature, the mean and median age of the affected cats is 9.7 and 9 years (range 5–14 years), the majority of cats are DSH (20/27), there is an equal gender distribution with 59% male cats, and the tumors affect more commonly the thoracic spinal cord (19 or 59% of the cases) than the cervical (7 cases) or lumbar (6 cases) spinal cord.[2,18,19,30–34] In all cases in which results of radiographic examination were reported, survey spinal radiographs were normal; myelography revealed an interruption of the normal flow of contrast at the level of the tumor, and in 2 cases where MRI was performed the tumor revealed a strong and homogeneous contrast enhancement (**Fig. 4**).[2,18,19,30–34] Two studies reported the survival time of cats treated with cytoreductive surgery. Levy and colleagues[18] reported a median survival time of 180 days (range 30–600 days) in 4 cats, with a fifth cat alive 1400 days following surgery. Rossmeisl and colleagues[19] reported a median survival time of 426 days (range 211–842 days) in 16 cats; 1 cat in the first study and 5 cats in the second were euthanized due to other conditions, and the reason for euthanasia was unknown in many cases.

TRAUMATIC

Intervertebral Disc Disease

Intervertebral disc disease (IVDD) is uncommon in cats. The incidence of IVDD in cats has been estimated as between 0.02% and 0.12%, whereas in dogs it is estimated at 2%.[35] A postmortem study of IVDD in cats performed in 1958 revealed the presence of

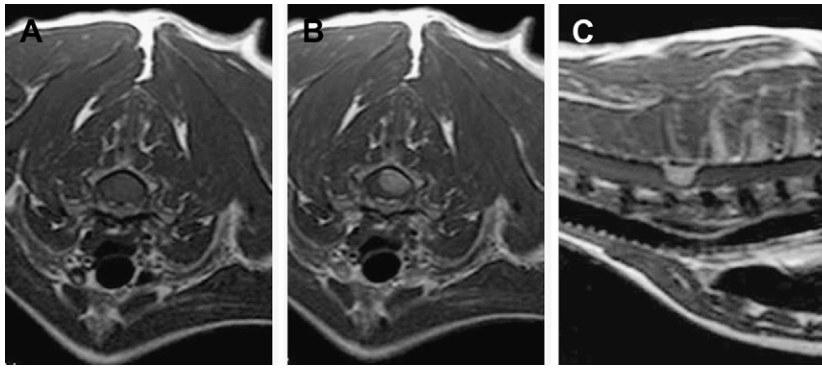

Fig. 4. Meningioma in a 12-year-old male Norwegian Forest cat. Transverse pre- (*A*) and postcontrast (*B*), and sagittal postcontrast (*C*) T1-weighted images at the level of the first thoracic vertebra demonstrate a smoothly marginated, homogenously contrast-enhancing mass displacing the spinal cord to the right. (*Courtesy of* Dr Rodolfo Cappello, North Downs Specialists Referrals, Bletchingley, Surrey, England.)

both Hansen type I (chondroid disc degeneration with annulus fibrosus completely perforated) and type II (fibroid degeneration with bulging annulus fibrosus) disc protrusions.[36] King and colleagues[36] found dorsal disc protrusions in 1 of every 4 cats obtained at random from local general practices, and severe trauma to the spine was the only exclusion criteria. In the same study, Hansen type I protrusions accounted for only 18% (16/91) of all protrusions; the cervical discs had the most protrusions of both types with a peak at C6-7; another peak of incidence was seen at the L4-5 intervertebral disc space.[36] King completed a second postmortem study in 1960, separating type II disc protrusion into small and large. In this study protrusions were more common in the cervical region than in the T10-S1 region; however, if only type I disc protrusions were considered, the cervical and T10-S1 regions had similar incidence (**Fig 5**A).[37] If the small type II protrusions were included, the highest incidence of protrusions was found in the C2-3 disc, and in the T10-S1 region, the incidence peak was not at the thoracolumbar junction as in the dog, but at L4-5.[37] Disc protrusions were found more frequently in older cats, in particular those older than 15 years.[38]

Recently, various investigators have published single case reports or case series of cats with IVDD, and there are 17 reports describing a total of 44 clinically affected cats with 50 intervertebral disc protrusions published between 1981 and November 2009.[1,35,39–53] One case report from 1971 was not considered, because the author later included the same case in a study on spinal lymphosarcoma.[54] Based on the information provided in these publications and including data from 8 cats from Marioni-Henry and colleagues,[1] the median and mean age of cats with clinical signs of IVDD was 8 years (range 1.5–17 years; 28 male cats and 16 female). Twenty-two cats were DSH, 10 domestic long-haired, 2 domestic medium-haired, and 10 (22%) were pure breed cats. The onset of clinical signs was acute in 13 of 43 cats and insidious with a progressive course in 30 of 43 (70%). Spinal hyperesthesia was reported in 22 of 24 (92%) cats. There were Hansen type I disc protrusions in 30 of 45 (67%) and type II in 15 of 45 cats. The most commonly affected intervertebral discs were L4-5 (9 cats), L7-S1, (7 cats), and T13-L1 (6 cats) (**Fig. 5**B). Two case series reports focused on lumbar and lumbosacral intervertebral disc disease in cats, and may have skewed the distribution of IVDD toward those locations, however, also in the study by King and Smith[37] the L4-5 disc space represented the peak of incidence for IVDD of both type I

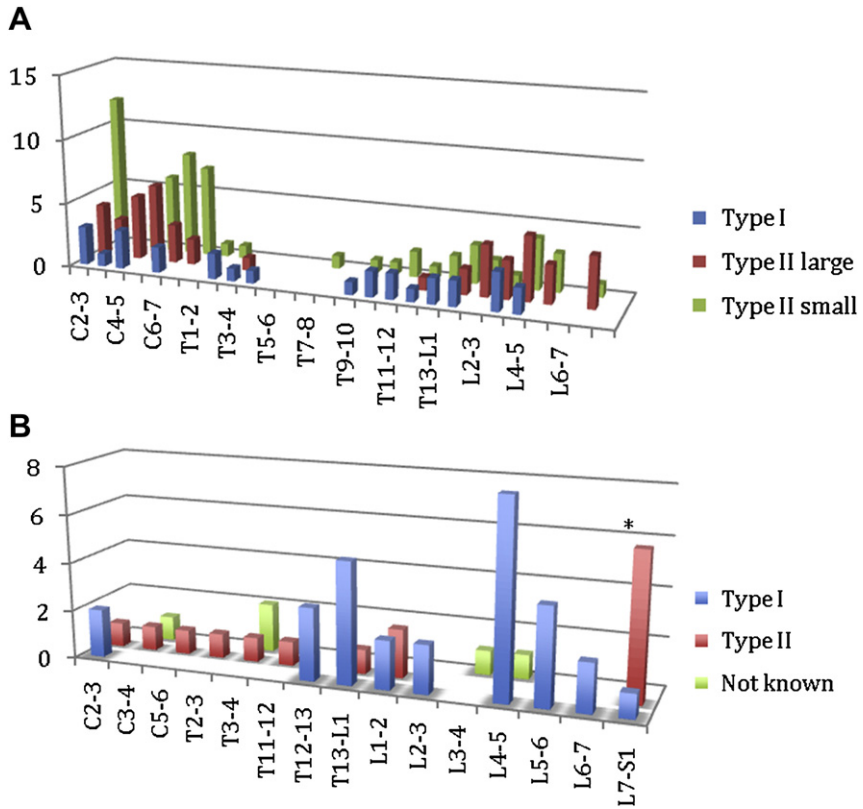

Fig. 5. (*A*) Prevalence of intervertebral disc protrusions in a population of 100 clinically unaffected cats (135 protrusions). (*Modified from* King AS, Smith RN. Disc protrusion in the cat: distribution of dorsal protrusion along the vertebral column. Vet Rec 1960;72:335–7; with permission.) (*B*) Prevalence of intervertebral disc protrusions in a population of 44 clinically affected cats (50 protrusions) published in veterinary literature between 1981 and 2009.[1,35,39–53] *Includes data from a study that only considered L7-S1 intervertebral disc disease in 6 cats.

and type II. The postmortem studies showed a high incidence of cervical disc protrusions, which were less common in clinically affected cats. It has been suggested that the cervical disc protrusions do not cause clinical signs because of the relatively larger size of the cervical vertebral canal.[38]

It is likely that the increased incidence of IVDD at certain locations of the spine is associated with the stance configuration and range of motion of the spine in the cat. A radiographic study of the cat vertebral column showed that during stance, the cat spine exhibits a mild dorsiflexion in the lower lumbar segments, a marked ventroflexion in the lower thoracic and upper lumbar segments, and a severe dorsiflexion in the cranial thoracic (above T9) and cervical segments.[55] The same study looked at the mean stance angles relative to the range of motion and found that during stance the lower lumbar joints (L4-5 to L6-7) are nearly maximally dorsiflexed and the joints from T10 to T13 are maximally ventroflexed, while the other joints are held midrange; this may explain the increased prevalence of IVDD at T12-L1 and L4-6 in clinically affected cats (see **Fig. 5**B).[55] In studies of both clinically affected and unaffected cats, IVDD is also found at unusual locations like T2-3 and T3-4 (see **Fig. 5**). These

locations are unusual because discs between T1 and T10 in the dog and T1-T11 in the cats are covered by the intercapital or conjugal ligament, which extends from the head of one rib to the head of the opposite rib over the intervertebral disc and across the floor of the vertebral canal. This ligament is thought to provide additional support to the disc and protection from intervertebral disc protrusions in dogs.[37,56] However, cats owe their flexibility to their vertebral column being able to achieve a total torsion of almost 180° and that most of the torsion is seen within a small range of the lower thoracic vertebrae from about T4 to T11.[56] Another reason for their flexibility is that the scapulae do not articulate with the axial skeleton, rather, the trunk is supported by muscles (levator scapulae, serratus ventralis, and major and minor rhomboids) that originate on the medial surface of the scapula near the dorsal border and fan out to insert on the trunk in a sling-like arrangement.[56] These muscles suspend the trunk from the scapulae much like the wires on a suspension bridge and help to stabilize the trunk against rolling movements.[56] The net suspensory force vector between scapula and trunk intersects the vertebral column near the T2-3 joint.[56] Therefore, it is possible that the proximity of a very flexible area (T4-11) to a much more stable area (T2-3) of the cat spine may lead to the development of IVDD at those locations, especially during traumatic events; in fact, in 2 cases with T2-3 and T3-4 IVDD an external trauma was documented.[1]

Treatment of cats with IVDD is often successful, especially with surgical decompression of the spinal cord. Thirty cats had surgery, and 16 of them had an excellent outcome with the cat returning to normal; 5 cats had a good outcome with some residual neurologic deficits, 5 had a fair outcome, 3 were lost to follow-up, and 1 died.[1,35,39–53] Ten cats were treated conservatively, 3 cats had a good outcome, and 1 an excellent outcome after treatment with corticosteroids, acupuncture, and physical therapy. Among the cats with IVDD treated conservatively, 1 cat had a poor outcome, 1 died, and 4 were euthanized; the remaining 5 cats were euthanized immediately after the diagnosis.[1,35,39–53]

CONGENITAL DISEASES

Congenital diseases can affect the spinal cord of cats; these are listed here and discussed further in the article on congenital spinal cord diseases by Westworth and Sturges elsewhere in this issue. Sacrocaudal dysgenesis is a constellation of congenital abnormalities commonly reported in Manx and Manx-crossed cats; it may affect the lumbar, sacral, and coccygeal spine, and it is often associated with malformations of the spinal cord such as myelodysplasia, hydromyelia and/or syringomyelia, meningocele or meningomyelocele, and tethered spinal cord.[57–59] Other congenital diseases affecting the spinal cord of cats are cyst-like lesions, such as spinal arachnoid cyst,[60–65] spinal intradural epithelial cyst,[66] spinal dermoid cyst and sinus,[67,68] or vertebral malformations causing compression of the spinal cord, such as hypoplasia of the odontoid process with secondary atlantoaxial luxation, and multiple cartilaginous exostosis.[57]

VASCULAR DISEASES

Vascular diseases affecting the spinal cord represented 9% in a postmortem retrospective study of 205 cats with spinal cord disease, and ischemic myelopathy was suspected in 6.5% of the cases in a study on MRI findings in 92 cats with clinical signs of spinal cord disease.[1,4] In the first study, 15 of 19 older cats (median age 9 years) presented vascular lesions affecting multiple segments of the spinal cord (11/15) and brain (5/15). The most common lesion was spinal cord malacia, in some cases vasculopathy

(5/15); hemorrhage (5/15) or thrombosis (4/15) were also found.[1] In 4 cases, history and histopathologic findings of severe necrosis involving the ventrolateral gray and white matter of thoracolumbar spinal segments were suggestive of ischemic poliomyelomalacia associated with severe abdominal compression and prolonged vasospams of lumbar arteries.[69] Focal malacia was described in 4 cases of vascular myelopathy in the author's study; the cause of the lesion could not be determined though trauma, intervertebral disc disease, or fibrocartilaginous embolism were suspected.[1]

Fibrocartilaginous Embolism

Fibrocartilaginous embolism (FCE) is not commonly reported in cats; however, publications on suspected or confirmed cases of FCE are appearing with more frequency, probably because the advent of MRI increases one's index of suspicion for the surviving cats and provides a better lesion localization for the histopathologic examination. FCE is caused by a small fragment of degenerated disc that occludes the blood supply to the spinal cord, leading to ischemic necrotizing myelopathy.[70] The exact pathophysiology of FCE is not known. It has been suggested that herniated nucleus pulposus reaches the spinal cord entering first the vertebral body vasculature, or that the disc extrudes directly within the spinal vasculature or into persistent embryonal arteries of the annulus fibrosus, or anomalous vasculature, or that the disc enters vessels that reach the nucleus pulposus following chronic inflammation.[71]

Based on information from the veterinary literature, FCE tends to affect older cats, the progression of clinical signs lasts usually less than 24 hours, and the clinical signs are often lateralized and with cervicothoracic localization (**Fig. 6**).[70–77] In 14 cats with FCE (confirmed in 9 cases and suspected in 5) that had a mean age of 9 years (median 10 years, range 4–12 years), there was no breed or gender predisposition. The mean and median progression of clinical signs was 19 and 16 hours; however, progression of clinical signs lasted for more than 24 hours in 2 cases and in 1 case there was a recurrence of clinical signs 24 hours after the onset and initial recovery. The most common clinical signs were tetra- or hemiparesis or hemiplegia (9/14), followed by posterior paresis/paraplegia and Horner syndrome, reported in 5 cases each. Spinal tap was performed in 9 cases; in 5 cases spinal fluid analysis revealed neutrophilic pleocytosis, in 2 cases it was normal, in 1 case there was albuminocytological dissociation, and in 1 case blood contamination. Myelography was performed in 3 cases and showed spinal cord swelling in all of these cases. MRI was performed in 9 cases, characterized by intramedullary lesions hypo- or isointense to normal gray matter on T1-weighted images, and hyperintense on T2-weighted images, with mild or absent uptake of contrast. MacKay and colleagues[76] reported that timing of infarct enhancement is variable; MRI performed within hours from the onset of an infarct may not enhance, whereas 5 to 6 days later the gadolinium enhancement should be more evident. The cervicothoracic intumescence was the spinal cord region with the highest prevalence of suspected or confirmed FCE (50%), followed by the lumbosacral intumescence affected in 21% of the cases; in the dog, a study suggested that the lumbosacral intumescence has the highest incidence of confirmed FCE (47%) followed by the cervicothoracic intumescence with 31%.[78] Nine cats were euthanized, and FCE was confirmed by histologic examination of the spinal cord and findings of extensive malacia and intravascular fibrocartilaginous emboli (see **Fig. 6**B). Three cats made a complete recovery within 2 to 6 weeks and 2 cats showed a significant improvement with only mild conscious proprioceptive deficits within 3 to 7 weeks.[71,74,77]

Even with only 14 cases of FCE, it is possible to observe that the MRI findings of FCE were consistent, that FCE in cats seemed to predominate in the cervicothoracic

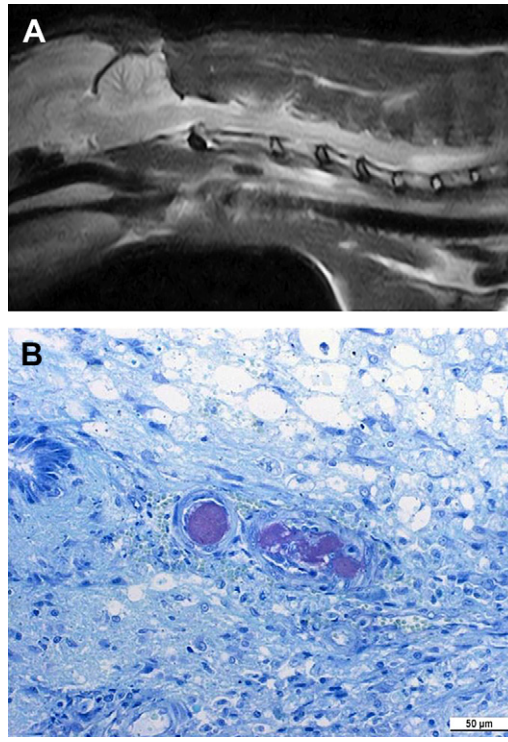

Fig. 6. Fibrocartilaginous embolism in a 9-year-old DSH cat. (*A*) Sagittal T2-weighted image of the caudal cervical spinal cord. There is a hyperintense intramedullary lesion extending from the sixth cervical to the first thoracic vertebra, and 2 degenerated intervertebral discs between the third and fifth cervical vertebrae. (*B*) Histopathological section of the caudal cervical spinal cord of the same cat in **Fig. 6**A. The toluidine blue–stained image reveals fibrocartilaginous emboli (stained purple) filling a vessel lumen within a necrotic section of the spinal cord (original magnification ×50 μm). (*Courtesy of* Sergio Rodenas, Sonia Anor, and Marti Pumarola, Neurology and Pathology Service, Veterinary College, University of Barcelona.)

intumescence, and clinical signs are markedly lateralized, especially when the cervical region is affected. The high incidence of FCE at the cervicothoracic intumescence is probably explained by the high prevalence of degenerated protruding disc in this region in the cat.[36,37] Also, the cervicothoracic junction is essential for head and neck movements in quadrupedal animals, especially for lowering the head.[79] One possible explanation for the marked lateralization of clinical signs may be associated with the arterial supply to the spinal cord of cats and dogs. Approximately half of the central arteries in the feline spinal cord alternate between branching to the right and left side, while the other central arteries have a common stem and supply both sides; the cervical region has a slightly higher frequency of unilateral central arteries (55%) compared with the thoracic (49%) and lumbar spinal cord (47%).[80]

Intraosseus Vascular Malformations

Intraosseus vascular malformations (IVM) causing spinal cord compression have been described in 3 young cats between the ages of 15 and 20 months with a history of 3 to 12 months of chronic progressive posterior paresis partially responsive to

corticosteroid therapy.[81] In some cases, the presentation of IVM can be similar to a neoplastic disease; plain radiographs showed focal decreased bone density or lysis of thoracic vertebrae, T2, T4, and T10-11, respectively; and myelography showed obstruction of the contrast column or extradural compression in all cases.[81] The lesion was described as vessels of varying size with endothelial cells, prominent pericytes, and variable amount of smooth muscles among a loose connective stroma separated by lamellar bony trabeculae, with osteoblasts and infrequent osteoclasts.[81] The bony and vascular proliferations caused severe spinal cord compression. Despite the aggressiveness of vascular proliferations, Wells and Weisbrode[81] considered these lesions as developmental anomalies based on the young age of the cats and the well-differentiated appearance of the vessels including endothelial, perithelial, smooth muscle, and fibrous cells.

Myelopathy Secondary to Aortocaval Fistula

A recent report described myelopathy secondary to aortocaval fistula in a 15-month-old male DSH with history of progressive paraparesis of 3 months' duration.[82] A large aneurysmal dilation of the caudal vena cava and an anomalous vessel arising from the vena cava were identified on abdominal ultrasound. Myelography, nonselective angiography, and contrast-enhanced CT confirmed an aortocaval fistula and vena caval aneurysm, with an engorged vertebral venous plexus causing a bilateral ventrolateral spinal compression from T12 to L4. An attempt to surgically occlude the anomalous vessel was unsuccessful; on necropsy, the ventral and lateral portions of the spinal cord from T7 to L4 showed Wallerian degeneration, which was more severe from T11 to L1 spinal cord segments. Vascular malformations of the CNS such as the one reported are often developmental anomalies, but they can also be acquired secondary to trauma, rupture of an arterial aneurysm, or surgical ligation of blood vessels.[82]

DEGENERATIVE/INHERITED DISEASES

Storage diseases, spinal muscular atrophy, and neuroaxonal or neuronal dystrophy are degenerative and inherited diseases of the spinal cord of cats.[83] Other degenerative myelopathies may have an infectious or nutritional etiology, such as the FeLV or cobalamin deficiency associated myelopathies described below.[84,85] The storage diseases affecting the feline spinal cord are: gangliosidosis GM1/GM2 reported in Siamese, Korat, and domestic cats; glycogenosis type IV reported in Norwegian Forest and domestic cats; sphingomyelinosis (Niemann-Pick disease) in Siamese, Balinese, and domestic cats; and mucopolysaccharidosis type VI in Siamese and domestic cats.[83,86]

Neuroaxonal Dystrophy

Neuroaxonal dystrophy (ND) is a degenerative condition characterized by swelling of the distal segment of axons (spheroids) within the CNS.[87] ND is thought to be hereditary and transmitted by an autosomal recessive gene.[88] The clinical signs are hind limb ataxia progressing to paresis and paralysis, head tremors, and hypermetria.[87] ND was initially described in 6 litters of tricolored DSH cats; the most prominent histopathologic findings were presence of spheroids in the brainstem, atrophy of the cerebellar vermis, and depletion of neuron within the spiral ganglia.[88] Affected kittens developed the first clinical signs at 5 to 6 weeks of age and had a diluted coat color.[88] More recently, ND was reported in 3 DSH cats with onset of clinical signs between 7 and 9 months of age and in 2 Siamese cats with first clinical signs at 2 weeks of age; the 2 Siamese cats and 2 of the DSH had a normal coat color, and no signs of inner ear involvement was found in the 3 DSH.[87,89]

FeLV-Associated Degenerative Myelopathy

Chronically FeLV-infected cats may present with various neurologic signs including lethargy, abnormal behavior, vocalization, hyperesthesia, urinary incontinence, and posterior paresis progressing to paralysis.[84] In a retrospective study of 16 cats with chronic FeLV infection, the most common clinical signs were posterior paresis progressing to paralysis within 1 year.[84] The average age for cats with known birth dates was 9 years. All cats were FeLV seropositive for 2 to 4 years, only 4 of 12 cats had the typical hematological abnormalities associated with FeLV infection, such as anemia, macrocytosis, neutropenia, macrothrombocytes, and thrombocytopenia; 10 cats had lymphopenia, and CSF was analyzed in 7 cats and was unremarkable. On histopathology there were widespread lesions in the spinal cord and brainstem characterized by white matter degeneration with dilation of myelin sheaths; some of the dilated myelin sheaths were devoid of axons and others had intact or swollen axons. Positive FeLV p27 immunostaining was present in glial cells, neurons, and endothelial cells of the sections of all spinal cords examined. FeLV proviral DNA was extracted from spinal cord alone in 5 cats and from spinal cord, brain, spleen, and intestine in another 5 cats; CSF was positive only in 1 cat.[84]

METABOLIC/NUTRITIONAL DISEASES

Hypervitaminosis A

Hypervitaminosis A causes a metabolic osteopathy in cats fed a liver-based diet for months to year.[90] This metabolic osteodystrophy is characterized by bony osteophytes and exostosis around joints, and tendon, ligament, and joint capsule attachments, with the occipital bone and cervical and thoracic vertebrae the most commonly affected sites.[91] Initially, the osseous hyperplasia involves the cranial cervical vertebrae, then with progression of the disease the joints of the cervical and cranial thoracic vertebrae may coalesce and cause complete bony ankylosis. The pathophysiology of vitamin A toxicity is not well understood. It has been hypothesized that vitamin A increases lability of cytomembranes and renders them prone to mechanical injury, leading to the formation of exostosis.[92] Vitamin A toxicity causes also an inhibition of the collagen synthesis and breakdown of musculotendinous insertions in the periosteum during muscular activity, therefore in cats the excessive muscular activity during grooming could explain the predisposition of the cervicothoracic spine to be more commonly affected.[91] Pain, reduced mobility of the neck, and forelimb lameness due to bony ankylosis and nerve root compression are the early clinical signs of vitamin A toxicity in cats. In some cases the lesions may progress to induce paralysis. Radiographic evidence of vitamin A toxicity may be detected after 15 weeks in kittens on an induction diet.[92] It is usual for chronically and severely affected cats to have a poor prognosis for functional recovery, and correction of the diet merely stops progression of the clinical signs; however, in some cases some neurologic deficits seemed to be reversed by correction of the diet.[91]

Nutritional Secondary Hyperparathyroidism

Nutritional secondary hyperparathyroidism (NSH) due to chronic dietary calcium deficiency, which leads to increased serum levels of parathormone and accelerated bone resorption, has become rare since the advent of balanced commercial pet food. In the past this disorder was commonly reported in puppies and kittens fed an all-meat diet.[93] A diet low in calcium will especially affect young growing animals that have an increased demand of calcium for bone growth and minimal calcium reserves. In response to a decrease in serum calcium concentration there is an increase

in secretion and synthesis of parathyroid hormone, which leads to increased bone resorption, renal calcium reabsorption/phosphorus excretion, and renal synthesis of active vitamin D. The clinical signs will reflect the effects of severe osteopenia and hypocalcemia. In a report of 6 cats with NSH, 2 cats presenting with spinal fractures associated with severe osteopenia were euthanized due to severe neurologic deficits; the other 4 cats improved after a change in diet.[93]

Myelopathy Associated with Cobalamin Deficiency

A 9-year-old cat with history of chronic pancreatitis associated with deficiency of serum cobalamin and folate concentrations presented with a progressive history of ataxia affecting all 4 limbs and tetraparesis.[85] Spinal reflexes were within normal limits. At the postmortem examination, the spinal cord presented bilateral and symmetric degeneration of the white matter. The most severe lesions affected the center of the dorsal columns of the caudal cervical and cervicothoracic segments with severe loss of fibers, marked astrocytosis, fibrosis, and proliferation of blood vessels. The cat had low serum concentration of cobalamin and folates, and clinical signs consistent with exocrine pancreatic insufficiency (EPI), confirmed by the histologic examination of the pancreas. Serum cobalamin concentrations are markedly decreased in most cats with exocrine pancreatic disease, because the intrinsic factor, which allows cobalamin absorption in the liver, is produced only by the pancreas in the cat. It is likely that chronic EPI and cobalamin deficiency caused the severe myelopathy in this cat as has been described in humans.[85]

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