Lumbosacral Intervertebral Disk Disease in Six Cats

Medical records of six cats diagnosed with lumbosacral intervertebral disk disease were reviewed. Clinical signs included reluctance to jump, low tail carriage, elimination outside the litter box, reluctance to ambulate, pelvic-limb paresis, urinary incontinence, and constipation. All cats had lumbosacral hyperpathia on palpation. Computed tomography in four cats revealed evidence of extradural spinal cord compression at the seventh lumbar (L_7) to first sacral (S_1) vertebral interspace. Compression was confirmed via myelography in three of these four cats, with confirmation in the fourth cat at the time of decompressive laminectomy. Each of the six cats underwent dorsal decompressive laminectomy at the L_7 to S_1 interspace. Postoperative clinical follow-up lasted 3 to 35 months, with most cats having excellent outcomes. **J Am Anim Hosp Assoc 2008;44:109-115.**

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Introduction

Intervertebral disk disease (IVDD) within the lumbosacral spine is a frequently reported and documented clinical disease syndrome in dogs, but little is known about lumbosacral IVDD in cats. Disk degeneration and protrusion occur commonly in cats and are routine incidental findings at necropsy.[1,2] However, IVDD in cats is not usually accompanied by recognized clinical signs.[1,2]

To the authors' knowledge, only one clinical case of feline lumbosacral IVDD has been reported.[12] Therefore, this retrospective study was performed to evaluate clinical signs and surgical outcomes in cats diagnosed with lumbosacral IVDD to further understand this disease process in domestic cats.

Materials and Methods

Medical records of feline cases of lumbosacral IVDD that were referred to California Veterinary Specialists between October 2002 and October 2006 were reviewed. Inclusion criteria consisted of 1) diagnosis of lumbosacral IVDD based on the results of myelography, computed tomography (CT), or confirmation at the time of surgery and 2) subsequent lumbosacral decompression. Age, sex, breed, body weight, clinical history, indoor/outdoor status, presenting clinical signs, cerebrospinal fluid analysis, radiographic findings, surgical findings, and outcome for each cat were recorded if available. Follow-up surveys were administered to the clients of all cases, and residual clinical signs were recorded.

All cases were assessed for degree of "functional recovery," which was based on regaining the ability to walk, fecal and urinary continence, and absence of persistent back pain.[4] Recovery was rated "excellent" if the owner felt that the cat had complete resolution of lumbosacral hyperpathia, with appropriate ambulation and elimination. Recovery was rated "good" if clinical signs were improved but mild residual lumbosacral hyperpathia was seen. Recovery was rated "fair" if significant clinical signs persisted, but quality of life was still acceptable. Recovery was

"poor" if clinical signs did not resolve and quality of life was unacceptable.

For the purpose of the current study, Hansen's type I disk disease was defined as disk extrusion into the spinal canal that causes a focal compressive myelopathy. Hansen's type II disk disease included protrusion of disk material within an intact annulus, resulting in a compressive myelopathy.[5] Spinal hyperpathia was defined as an unpleasant, painful response to a noxious stimulus (especially if repeated). Such a response is characterized by delay, overreaction, and aftersensation.[5]

Results

Out of a total of 11,151 cats seen within the study time frame, six cats were diagnosed with lumbosacral IVDD, suggesting a 0.05% incidence in the authors' hospital population. Descriptive characteristics for the six cases are presented in Table 1. The cases were equally divided between spayed females and castrated males, with domestic shorthair (DSH) being the most common breed. The mean age was 12.6 years (median 12 years), and the mean body weight was 6.5 kg [Table 1]. All six cats were kept strictly indoors.

The most common signs from the clinical histories were reluctance to jump, which was observed in all cats, and low tail carriage, which was seen in three cats [Table 2]. All cats were ambulatory on presentation and had significant pain when direct digital pressure was applied to the lumbosacral region. All five cats with a tail had pain on tail hyperextension (hyperextension was not possible in the Manx). Digital rectal evaluation was not performed in any conscious cat. Significant ancillary findings included severe bilateral stifle osteoarthritis, diabetes mellitus, and chronic renal failure (case no. 1); bilateral medially luxating patellae (case no. 4); and obesity, a ruptured cranial cruciate ligament, and cystic calculi (case no. 6).

Cerebrospinal fluid was collected from the cerebellomedullary cistern in three of the six cats; all cell counts and protein concentrations were within the reference ranges. The CT scans, which were performed in four cats, did not reveal any articular process thickening or sclerosis. Two of the four CT scans suggested a ventral compressive lesion of the cauda equina [Figures 1, 2]. The CT scan of case no. 3 revealed lumbosacral caudal malformation and dural malformation, which were confirmed by myelography.

The CT scan for case no. 4 revealed stenosis of the spinal canal within a lumbosacral transitional vertebra at the seventh lumbar (L_7) to first sacral (S_1) interspace [Figure 3]. Survey spinal radiographs of this cat confirmed that a transitional lumbosacral vertebra was incompletely fused to the remainder of the sacrum [Figure 4]. Myelography confirmed mild ventral compression at the L_7 transitional lumbosacral vertebra [Figure 5].

Dorsal decompressive laminectomies were successfully performed at the L_7 to S_1 interspace in all cats. Case no. 4 also underwent right-sided medial patellar stabilization during the same anesthetic/surgical period. One cat had evidence of extruded disk material within the spinal canal, consistent with Hansen's type I disk disease. Five cats had no obvious disk extrusion, but one of these had a Hansen's type II disk protrusion.

A preoperative dose of methylprednisolone sodium succinate[a] (30 mg/kg) was administered intravenously to each cat at the start of the surgical procedure.[6] No glucocorticosteroids were administered in the postoperative period.

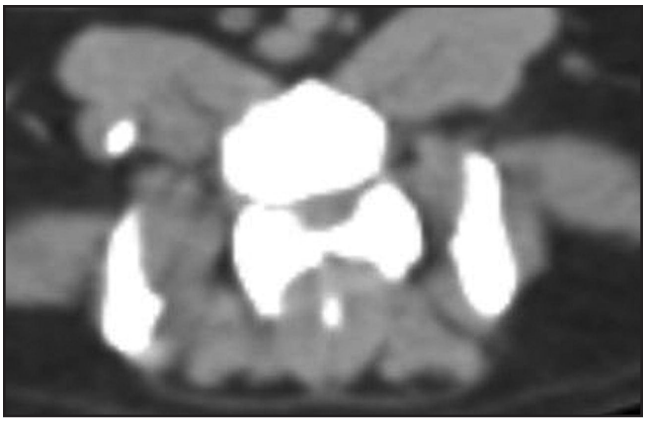

Figure 1—A computed tomographic (CT) image of case no. 5 with mild caudal equina compression at the lumbosacral junction.

Figure 2—A CT image of case no. 6 with mild loss of epidural fat on the right side.

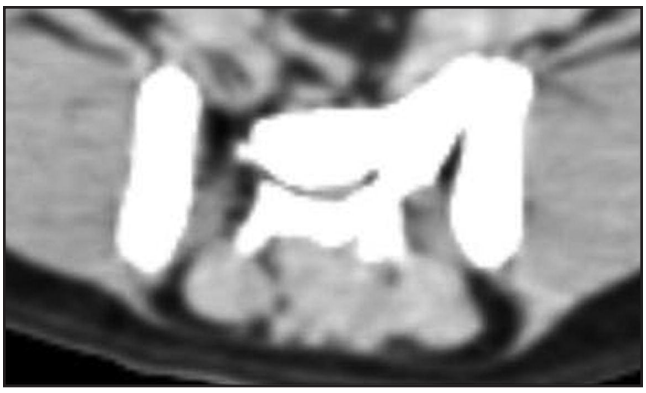

Figure 3—A CT image of case no. 4 with narrowing of the vertebral canal within the body of the transitional lumbosacral vertebra.

Table 1

Clinical Findings in Six Cats With Lumbosacral Intervertebral Disk Disease

Case No.	Age (y)	Sex*	Breed†	Weight (kg)	Clinical Signs	Site‡	Diagnostics§	Management	Follow-up (mo)	Outcome
1	17	CM	Abyssinian	4.5	Reluctance to jump, pelvic-limb paresis	L_7-S_1	R	Surgery	9	Fair
2	12	CM	DSH	6.8	Reluctance to jump, spinal hyperpathia	L_7-S_1	R, M, CSF	Surgery	35	Excellent
3	15	SF	Manx	4.7	Urinary incontinence, constipation	L_7-S_1	R, M, CT, CSF	Surgery, cystostomy tube	23	Fair
4	7	SF	DSH	6.3	Straining to defecate, low tail carriage	L_7-S_1	R, M, CT, CSF	Surgery, patellar stabilization	15	Excellent
5	13	SF	DLH	6.5	Reluctance to jump, low tail carriage	L_7-S_1	CT	Surgery	3.5	Excellent
6	12	CM	DSH	10.4	Reluctance to ambulate, low tail carriage	L_7-S_1	CT	Surgery	3	Excellent

* CM=castrated male; SF=spayed female
† DSH=domestic shorthair; DLH=domestic longhair
‡ L_7=seventh lumbar vertebra; S_1=first sacral vertebra
§ R=survey radiographs; M=myelogram; CSF=cerebrospinal fluid analysis; CT=computed tomography

Table 2

Clinical Histories of Cats With Lumbosacral Intervertebral Disk Disease

Case No.	Clinical Histories	Duration (mo)	Resolution After Surgery	Cause of Death
1	Chronic renal failure	24	None	Congestive heart failure
	Bilateral stifle osteoarthritis	17	None	
	Weak in hind limbs	12	Complete	
	Hind-limb muscle atrophy	12	Improved	
	Unwilling to jump	8	Improved	
	Urine retention	6	Improved	
2	Unwilling to jump	6	Improved	N/A*
	Lethargy	2	Complete	
	Hiding	2	Complete	
	Back pain	2	Complete	
	Hind-limb lameness	1	Complete	
3	Recurrent constipation	1	Improved	N/A
	Urinary incontinence	1	None	
	Unwilling to jump	1	Complete	
4	Bilateral medial patellar luxation	Unknown	None	N/A
	Unwilling to jump	3	Complete	
	Back pain	2	Complete	
	Carrying tail low	2	Complete	
	Straining to defecate	1	Complete	
5	Fell 12 feet 7 years previously			N/A
	Unwilling to jump	6	Complete	
	Decreased tail movement	4	Complete	
	Unwilling to walk	2	Complete	
6	Right CCL rupture†	2	None	N/A
	Difficulty sitting and squatting	2	Complete	
	Unwilling to walk	2	Complete	
	Unwilling to jump	2	Complete	
	Carrying tail low	2	Complete	
	Difficulty getting into litter box	1	Complete	

* N/A=not applicable
† CCL=cranial cruciate ligament

Postoperative follow-up ranged from 3 to 35 months. Clinical signs were markedly improved within 2 weeks in four cats. Case no. 1 showed a slight improvement but had continued urine retention that may have been caused by an unrelated underlying disease process. Case no. 3 continued to have urinary incontinence, so a permanent cystostomy tube was placed during a later surgical procedure. Owner survey responses revealed that the outcomes were "excellent" in four cats and only "fair" in two cats.

Discussion

Little information has been published on IVDD in cats. To the authors' knowledge, only 30 cats with clinical IVDD have been reported in the veterinary literature,[3,7-12] with only a single case report of lumbosacral IVDD.[12] These 30 cats had a total of 32 localized lesions [Table 3],[3,8,11,12] because two cats had evidence of compression at both the 13th thoracic (T_{13}) to first lumbar (L_1) vertebral interspace and the fourth lumbar (L_4) to fifth lumbar (L_5) vertebral

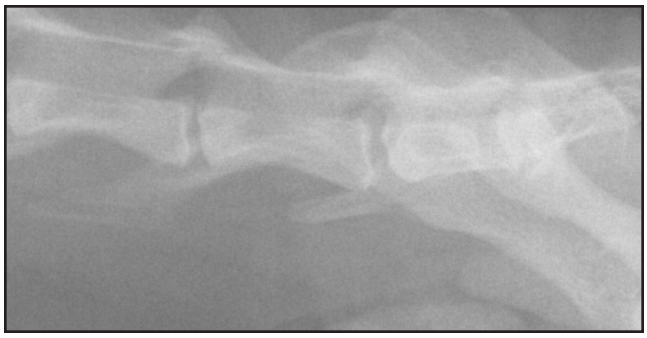

Figure 4—Survey spinal radiograph of case no. 4, showing transitional lumbosacral vertebra.

Figure 5—Myelographic image of case no. 4, showing mild ventral compression at the seventh lumbar (L_7) transitional vertebra.

Table 3

Location of 32 Lesions Among Cases of Feline Intervertebral Disk Disease Reported in the Veterinary Literature

Lesion Location	No. of Cats
C_3-C_4	1
C_5-C_6	2
T_9-T_{10}	1
T_{11}-T_{12}	1
T_{11}-L_1	1
T_{12}-T_{13}	2
T_{13}-L_1	7
L_1-L_2	2
L_2-L_3	1
L_4-L_5	7
L_4-L_6	1
L_5-L_6	3
L_6-L_7	2
L_7-S_1	1

interspace. Most (23) of these cats were affected in the thoracic or lumbar spine and underwent hemilaminectomy, dorsal laminectomy, fenestration, or durotomy.[3] Clinical outcome data were available for 20 of these 23 cats, with 18 showing postoperative improvement.[3]

Clinically significant IVDD has been reported in cats ranging in age from 1.5 to 17 years, with a mean age of 7 years.[3,9] The cats in this study were substantially older (mean 12.6 years), although still within the range reported in the literature. Chondrodystrophic breeds of dogs suffer from disk degeneration at an early age, whereas IVDD in cats appears to be a condition of middle-aged animals.[3]

In one case report, a 5-year-old DSH presented with acute hemiparesis and Horner's syndrome. Magnetic resonance imaging (MRI) revealed left-sided dorsal displacement of the spinal cord at the third cervical (C_3) to fourth cervical (C_4) vertebral interspace. The presumptive diagnosis was focal spinal cord edema with intervertebral disk extrusion. With conservative treatment, this cat gradually improved over 6 months.[11]

A retrospective study evaluated IVDD in six cats.[10] Radiographic studies confirmed narrowed disk spaces, mineralized disks, and one or more extradural compressive lesions in each cat. All intervertebral disk extrusions were in the thoracolumbar spine. Hemilaminectomy was performed in all cats, and the removed extradural material was confirmed as coming from degenerative disks. Neurological recovery was good to excellent in five (83%) of the six cats. It was concluded that IVDD in cats has many similarities to IVDD in dogs, and healthy cats with acute disk extrusion respond well to decompressive surgery.[10]

A retrospective study evaluated IVDD in 10 cats diagnosed over an 11-year period.[9] All disk herniations occurred in the thoracolumbar spine, with an increased incidence at the L_4 to L_5 vertebral interspace. Eight cats had Hansen's type I disk disease, and two cats had Hansen's type II disk disease. Nine cats had radiographic imaging, which consisted of myelography (in eight cats), CT (in three cats), or both (in two cats). The 10th cat was diagnosed at necropsy after euthanasia for clinical signs. Seven cats had been treated with corticosteroids before referral. Hemilaminectomy was performed on seven cats, of which six had extruded disk material in the spinal canal, and one had mild extradural compression over a narrowed disk space. Two cats had minimal compression on myelography and improving clinical signs, so they were conservatively managed with strict confinement for 4 weeks. Follow-up in seven cats revealed that recovery was good in one conservatively managed cat but poor in the other one, which never regained urinary or fecal continence. Recovery was good to excellent in four cats

undergoing surgery, but it was only fair in one ambulatory cat because of fecal and urinary incontinence.[9]

Type I disk disease has been reported to occur more commonly in the thoracolumbar region of cats, which is consistent with findings in dogs.[9] In the authors' study, only one cat had a type I IVDD, while five cats had type II disease. The higher proportion of type II lumbosacral IVDD in the cats of this study is consistent with that of lumbosacral IVDD in dogs.

The single case of lumbosacral IVDD reported in the literature involved an 8-year-old, castrated male DSH.[12] The cat presented with a 2-day history of back pain, flaccid tail, and urinary and fecal incontinence. Myelography, CT, and an epidurogram revealed an L_7 to S_1 extradural lesion. Exploratory surgery and histopathology confirmed a Hansen's type II disk protrusion. The cat regained neurological function by 6 weeks after surgery.

In dogs, the most common clinical presentation of lumbosacral disease is degenerative lumbosacral stenosis (DLSS) with protrusion of a Hansen's type II disk.[13] This occurs most commonly in middle-aged and medium- to large-breed dogs, especially German shepherds.[12-14] Causes of lumbosacral IVDD in dogs include thickening of the dorsal annulus, mechanical instability of the lumbosacral junction, osteophyte formation within the spinal canal, thickening of the articular facet joint capsule, thickening of the interarcuate ligament, and transitional vertebrae.[13] Lumbosacral hyperpathia is the most consistent clinical finding, and signs can be acute or chronic. Diagnosis of DLSS may be difficult, and DLSS can be easily confused with chronic orthopedic conditions. The diagnostic evaluation includes general, orthopedic, and neurological examinations, followed by radiographs and advanced imaging techniques such as myelography, discography, epidurography, CT, and MRI. If a lesion is not demonstrated by diagnostic imaging, exploratory surgery may be indicated.

In a study of 131 dogs with DLSS treated by dorsal laminectomy with fenestration, 56% were German shepherds, with a 2:1 male:female ratio.[14] The most common clinical signs were 1) pain on hyperextension of the tail or hind limbs (98%); 2) reluctance or pain when jumping, rising from a prone position, or climbing stairs (92%); and 3) lumbosacral pain (85%) and unwillingness to perform extensive physical activity because of pain or stiffness (85%). Overall, 79% of dogs returned to normal after surgery, and 93% showed clinical improvement within the follow-up period (26±17 months). The rate of recurrence based on owner observation or clinical examination was 18%.[14]

The above results are similar to those reported in another study of 69 dogs with DLSS that underwent dorsal laminectomy. The male:female ratio was 2.6:1, with German shepherds as the most common breed (27% of cases).[15] Clinical signs were also similar and included lower lumbar pain (77%); difficulty jumping, climbing, rising, or sitting (53%); hind-limb lameness (38%); tail paralysis (16%); and urinary or fecal incontinence (16%). Neurological examination revealed pain on palpation or hyperextension of the lumbosacral junction (91%) and conscious proprioceptive deficits (39%). The outcomes after surgeries were again good, with excellent results reported for 78% of dogs during follow-up (38±22 months).[15]

As with DLSS of dogs, diagnosis of cats with IVDD may be difficult, and IVDD can be easily confused with chronic orthopedic conditions. Clinical histories and physical findings for the cats in the authors' study were similar to those reported for dogs with DLSS. All cats in the authors' study were reluctant to jump, with low tail carriage seen in three cats and reluctance to ambulate in two cats. All cats had pain when the lumbosacral region was palpated, and all (except the Manx) had pain on tail hyperextension. Hansen's type II disease predominated in this study, as it does in dogs, and recoveries after dorsal decompressive laminectomies were good to excellent in most cats. The authors have not to date noted any recurrence of clinical signs in the six cats that were treated for IVDD, which is consistent with the other literature reports of IVDD in cats. Recurrence has been documented in dogs.[16]

In contrast to dogs with DLSS reported in the literature, the cats in the authors' study were older, with a mean age of 12.6 years. No specific breed or sex predilection was noted. One of the cats in this study had evidence of a Hansen's type I disk extrusion at the time of surgery, and one had urinary retention. The persistent, postoperative urinary incontinence in the Manx was probably associated with sacrocaudal dysgenesis, which is inherited in this breed as an autosomal dominant trait with varying degrees of sacral or lumbar deformities.[17]

Sacrocaudal dysgenesis and associated malformations have been recognized in most breeds of cats and are especially common in tailless breeds. Many tailless cats do not have neurological deficits, and sacral/caudal deformities are usually reported as incidental radiographic findings.[17] Clinical findings depend on the degree of cauda equina or spinal cord malformation. Signs include paraparesis, paraplegia, megacolon, atonic bladder, absent anal reflex, urinary and fecal incontinence, and perineal analgesia. Diagnosis is made based on clinical signs, radiography, myelography, and CT scan. Bladder expressions and fecal softeners can be used to manage mildly affected cases, but recurrent urinary tract infections, megacolon, and chronic constipation are common persistent problems. Treatment is ineffective in severely affected cats.[17]

Three cats in the authors' study had preexisting orthopedic disease:

1) The cat with concurrent cranial cruciate ligament rupture was obese and had cystic calculi. The stifle injury was not addressed during follow-up, and this cat had residual postoperative lameness.
2) The cat with medially luxating patellas had stabilization of one patella performed during the same anesthetic/surgical episode as the dorsal decompressive laminectomy. Clinical signs resolved completely, and

the contralateral patella was stabilized 6 weeks after the first surgery.

3) The cat with a history of chronic renal failure and bilateral stifle degenerative joint disease (DJD) showed improvement of lumbosacral clinical signs after surgery, but urinary retention persisted. The DJD was not treated. Nine months after surgery, this cat died from renal disease and congestive heart failure.

It is not known if the orthopedic disease was a factor in the development of lumbosacral IVDD or if IVDD was a sequela of orthopedic conditions in this patient population.

Limitations of this study include its retrospective nature and the lack of consistent diagnostic imaging data for all cases. Computed tomographic scans were performed in four of the cases, but lumbosacral lesions can be difficult to visualize with CT. Magnetic resonance imaging may have improved the authors' diagnostic capabilities, but recent research on DLSS in dogs suggests a high degree of agreement between CT and MRI.[18] Therefore, the authors feel that CT imaging was an acceptable diagnostic modality. Furthermore, the recent canine study showed only a slight to fair agreement between diagnostic imaging and surgical findings.[18]

A further limitation of the study was the small sample size. However, this is consistent with the low numbers of feline IVDD cases that have been reported previously. All of the cats in this study underwent surgical decompression; therefore, no conservatively managed cases were available for comparison.

Conclusion

Until recently, lumbosacral IVDD in cats has been infrequently recognized and reported, with only 30 reported cases of feline IVDD.[3,8,11,12] With one exception, all cases in this series had long-standing clinical histories and clinical signs attributable to lumbosacral disease. Therefore, the authors feel that the clinical signs correlated well with their diagnostic and surgical findings. Furthermore, all cats had clinical resolution or were significantly improved after lumbosacral decompression, regardless of concurrent disease and advanced age.

Subtle signs such as reluctance to jump and low tail carriage may be overlooked as early indicators of IVDD in cats. Therefore, the incidence of lumbosacral IVDD in cats may be higher than previously recognized. Lumbosacral IVDD should be considered as a differential diagnosis in all cats having caudal lumbar pain or pain on tail hyperextension.

Footnotes

[a] Solu-Medrol; Pharmacia, Kalamazoo, MI 49001

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