Fracture of L7 vertebral articular facets and pedicles following dorsal laminectomy in a dog

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- Degenerative lumbosacral stenosis is a common disease of middle-aged dogs and is particularly prevalent in male German Shepherd Dogs.
- Decompression of the cauda equina is usually achieved by performing a dorsal laminectomy and partial dorsal annulectomy of the offending disk.
- Strict exercise restriction must be enforced after performing multi-level dorsal laminectomy and diskectomy because of the risk of fracture of the vertebral articular facets or pedicles after surgery.

A 6.5-year-old working (perimeter guard dog) sexually intact male German Shepherd Dog was referred to the Atlantic Veterinary College for evaluation and treatment of progressive hind limb weakness and ataxia. Clinical signs were initially observed following a training session 3 months prior to admission, and the dog's condition had only partially responded to rest. A decrease in conscious proprioception and ataxia was observed in both hind limbs. Proprioceptive positioning was delayed in both hind limbs, and abnormal wear was observed on the dorsal aspect of the toenails. Hyperesthesia was observed when the tail was elevated and upon direct pressure over the lumbosacral region. In addition, signs of pain could be elicited by digital pressure on the sacrum during rectal palpation. Although the aggressive nature of the dog limited the neurologic examination, mentation and forelimb function were considered clinically normal.

With the exception of a slight increase in serum glucose concentration (6.5 mmol/L; reference range, 3.3 to 5.6 mmol/L) and moderate lymphopenia (0.535×10^9 cells/L; reference range, 0.8 to 5.1×10^9 cells/L) attributed to stress, results of CBC determination and serum biochemical analysis were within reference limits.

Findings on a vertebral radiograph included ventral spondylosis at the T10-11, T13-L1, L1-2, and L2-3 intervertebral spaces. A severe spondylotic reaction was also observed at the lumbosacral junction with widening and wedging of the disk space at that location (**Fig 1**).

Results of CSF analysis were unremarkable with the exception of a minimal increase in protein concentration (0.29 g/L; reference range, 0.17 to 0.25 g/L), consistent with mild blood contamination. The nucleated cell count was within reference limits.

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Figure 1—Lateral radiographic view of the lumbosacral junction in a German Shepherd Dog. Notice the severe spondylosis and wedging of the L7-S1 intervertebral space.

A cisternal myelogram was performed with iohexol (0.3 ml/kg [0.135 ml/lb]). The dural sac extended to the proximal aspect of the sacrum. Myelography revealed dorsal deviation of the dural sac and slight extradural compression of the cauda equina at the L7-S1 intervertebral disk space. Extension of the vertebral column slightly worsened the compression. An epidurogram was deemed unnecessary.

A dorsal laminectomy with preservation of the vertebral pedicles and the vertebral articular facets (Funkquist type B procedure) was performed over the L7-S1 disk space. Because of apparent compression of the cauda equina cranial to the lumbosacral junction, the dorsal laminectomy was extended cranially over the L6-L7 disk space, and the final opening extended from the caudal half of L6 to S2. The pedicles and articular facets were left intact. Disk fenestration by partial dorsal annulectomy was performed at the L6-L7 and L7-S1 disk spaces.

The dog had an uncomplicated recovery and was discharged the following day. The owner was instructed to strictly confine the dog for the following 8 weeks, including limited leash walking and cage confinement when off leash.

One week following discharge, the dog developed a seroma over the surgical site. The dog received cephalexin (25 mg/kg [11.25 mg/lb], PO, q 8 h) from the referring veterinarian. One week later, during a short walk, the dog yelped and collapsed. The dog was again admitted to the veterinary teaching hospital for evaluation. The dog was ambulatory but had signs of severe back pain, marked ataxia, and intermittent signs of radiculitis that were worse on the right hind limb. A large ($15 \times 10 \times 5$ cm) seroma was present over the surgery site. Rectal temperature was 39.7 C (103.5 F) and the dog was highly apprehensive. A fine-needle aspirate of the seroma was performed. Serosanguineous fluid was retrieved and submitted for cytologic analysis and aerobic and anaerobic bacterial culture. In the meantime, the dog was given enrofloxacin (10 mg/kg [4.5 mg/lb], PO, q 24 h) in addition to the cephalexin. Results of CBC and serum biochemical analyses were unremarkable. Cytologic evaluation of the aspirated fluid revealed large numbers of erythrocytes and moderate numbers of nondegenerative neutrophils. Macrophages containing erythrocytes or cellular debris were also identified often. These findings were consistent with a diagnosis of hematoma.

Vertebral radiography (**Fig 2**) revealed a small radiolucent line on the articular facet of L7, visible on the lateral view. The radiolucent line could not be confirmed on the ventrodorsal view.

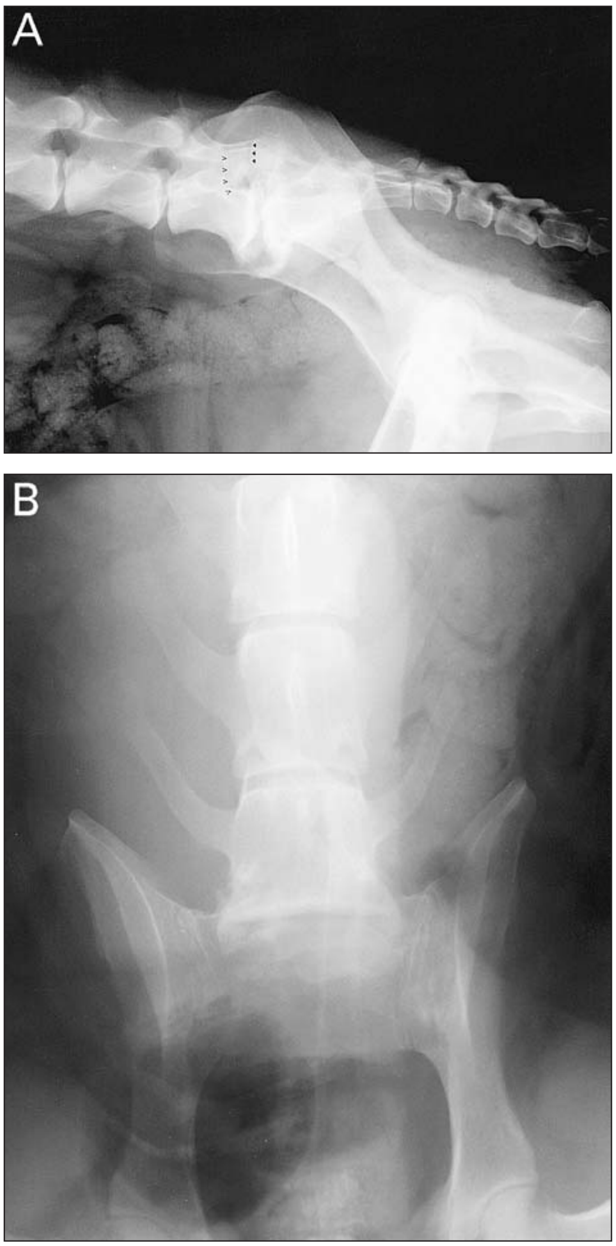

Figure 2—Lateral (A) and ventrodorsal (B) radiographic views of the lumbosacral junction of the same dog as in Figure 1. In the lateral radiographic view, notice the fracture of the left articular facet (closed arrowheads) and fracture of the right articular facet and pedicle (open arrowheads) of L7.

Exploratory surgery was performed. In addition to a large subcutaneous hematoma, the left caudal articular facet and the right pedicle and caudal articular facet of L7 were fractured. The right pedicle was fractured immediately caudal to the cranial articular facets, and the fractures extended caudally at the level of the vertebral body. The fracture fragments were removed. The L7 nerve roots appeared grossly intact. The vertebral column was stabilized with intramedullary pins and methylmethacrylate cement. Two end-threaded 1/8-in-diameter, positive-profile pins were inserted through the articular facets of S1 and directed ventrally and laterally so as to engage the wings of the sacrum and the ilium. Two other similar pins were inserted through the body of L6 at approximately 50° from the sagittal plane. Two 3/32-in-diameter, smooth Steinmann pins were inserted through the articular facets of L6 and L7, and 2 similar pins were inserted through the remainder of the pedicles of L7 and into the body of L7. A large fat graft was placed over the laminectomy site. The pins were cut, leaving approximately 3 cm protruding from the vertebral bodies dorsolaterally, and the exposed pins were notched with a pin cutter. Two 20-g packages of methylmethacrylate, mixed with 1 g of cefazolin powder, were used to connect and secure the pins (**Fig 3**). Abundant lavage with cool saline (0.9% NaCl) solution and the thick fat graft preserved the cauda equina from thermal damage.

Although ataxia was still observed following surgery, signs of radiculitis had resolved. Neurologic signs steadily improved following surgery. The results of the bacterial cultures were negative. Antimicrobials were discontinued 7 days after surgery, and the dog was discharged from the hospital. Recommendations for the dog included cage rest for 1 month followed by a progressive return to exercise. Free exercise, running, and jumping were not allowed for a minimum of 3 months. One month following discharge, the owner reported that the dog had regained strength and coor-

Figure 3—Lateral radiographic view of the lumbosacral junction of the same dog as in Figure 1. Notice the vertebral stabilization by use of intramedullary pins and methylmethacrylate.

dination and appeared pain free but still occasionally scuffed its toenails. The dog has been retired from its duties and was kept as a pet. Telephone conversation with the owner 1 year following the surgery revealed that the dog was free of any apparent back pain and was ambulating normally.

Degenerative lumbosacral stenosis is a disease of middle-aged dogs and is well recognized in working dogs.[1] Male dogs and German Shepherd Dogs are particularly at risk for the disease.[2] Lumbosacral stenosis is characterized by a progressive compression of the nerves forming the cauda equina. Signs of lower back pain that may be associated with exercise are often the initial abnormality.[3] As the disease progresses, weakness and proprioception deficits of the hind limbs become evident. Severe neurologic deficits are usually seen late in the course of the disease and warrant a guarded prognosis.[4]

Although signalment and clinical signs are often highly suggestive of the disease, diagnostic imaging is usually used to confirm the diagnosis and rule out other disease processes. Computed tomography and magnetic resonance imaging play a major role in the diagnosis of intervertebral disk disease in humans, and their benefits to veterinary patients have also been recently confirmed.[5] However, as a result of financial or logistic reasons, myelography and epidurography are still commonly used for the diagnosis of cauda equina syndrome. Because the dural sac does not extend past the lumbosacral space in some dogs, myelography is not always rewarding. For this reason, epidurography is usually the preferred method for diagnosing cauda equina compression.[4,6] In the dog of our report, the dural sac extended past the first sacral vertebra, and the compression of the cauda equina could be diagnosed on the myelogram. An epidurogram therefore was not performed.

Although a fixation-fusion technique has been described for the treatment of lumbosacral stenosis,[7] the most widely accepted method of treatment is decompression of the cauda equina by performing a dorsal laminectomy with partial dorsal annulectomy.[4] The effect of these procedures on vertebral stability has been incompletely investigated in dogs.

The stability of the vertebral column is provided by 3 complementary systems: a passive system composed of the ligaments connecting the vertebrae, an active system composed of the muscles and tendons surrounding the vertebral column, and a neural control system that receives and processes information from various receptors to control the active system.[8] The passive system is often divided into dorsal and ventral compartments. The vertebral body, the intervertebral disk, and the ventral and dorsal longitudinal ligaments constitute the ventral compartment, whereas the dorsal compartment consists of the vertebral lamina, pedicles, articular facets, joint capsules, ligamentum flavum, and the spinous processes and their associated ligaments.[9] Alteration or dysfunction of any of these systems has the potential to cause spinal injury if the compensatory mechanisms provided by the 2 other systems are exceeded.[8]

Viateau et al[10] studied the biomechanical properties of lumbosacral vertebrae in clinically normal dogs and in dogs with vertebral spondylosis. The authors of that study found altered biomechanical behavior of spondylotic lumbosacral vertebral segments, compared with unaffected segments. Furthermore, findings of the study[10] support the importance of the dorsal ligamentous structures and the articular facets for vertebral column stability, particularly in the presence of disk degeneration and spondylosis.

Smith and Walter[11] have shown that surgical procedures or trauma affecting the dorsal compartment of the lumbar vertebrae significantly decrease the stability and strength of the vertebral column. Removal of the supraspinous and interspinous ligaments resulted in a 36.2% decrease in vertebral column rigidity in extreme flexion and a 62.4% decrease in ultimate bending strength of the L3-L4 vertebral segment. Dorsal laminectomy with bilateral facetectomy markedly decreased vertebral column stiffness, increased the range of motion, and decreased the ultimate breaking strength by 75%.[11] Because of the significant effect of articular facet removal on vertebral column stability, preservation of the facets and pedicles has been recommended when performing dorsal decompression of the vertebral column.[2,11]

Disk fenestration also negatively affects vertebral column stability. The importance of the integrity of the intervertebral disk has been well documented in the literature. Ventral cervical fenestration caused instability of the cervical vertebrae in large-breed dogs.[12] A small lateral fenestration did not significantly affect lateral bending stiffness of the vertebral column in dogs,[13] whereas larger fenestration induced significant instability and significantly decreased bending strength.[14] The influence of the size of the annulus tear on vertebral column stability has also been observed in other species.[15]

The active role of the epaxial musculature on the stabilization of the vertebral column has recently been investigated. Stretching of the interspinous ligaments in cats caused electromyographic activity in the epaxial musculature adjacent to the vertebral segment.[16,17] These changes were evident on either side of the site of stimulation and spanned a minimum of 3 vertebral motion units. This mechanism, by causing contraction of the epaxial musculature, is believed to protect the vertebral column against instability if excessive stretching of the ligaments occurs.[16] Although the exact location of the receptors responsible for these electromyographic changes is unknown, the removal of the spinous processes and interspinous ligaments during dorsal laminectomy may deprive the vertebral column from some of the protective effect afforded by the epaxial musculature. Furthermore, the removal of the spinous processes deprives the muscles of their attachment to the vertebrae and therefore reduces their ability to counteract the instability should contraction occur.

Previously reported[1,18] complications of dorsal laminectomy include seroma formation, iatrogenic trauma to the nerve roots, unresolved clinical signs, muscle fibrosis, and contracture over the spinal cord. Even though instability caused by dorsal laminectomy and fenestration appears likely, fractures associated with this instability, even when multiple sites are exposed, have not been reported in veterinary medicine.[18] Fracture of 1 or both articular facets has been infrequently described for humans following laminectomy. These fractures are suspected to be a source of recurrent pain following back surgery.[19]

It is likely that a combination of factors was responsible for the fracture of the pedicles and articular facets in the dog of our report. The extent of the dorsal laminectomy associated with dorsal annulectomy at both intervertebral spaces may have weakened the vertebral segments to the point that fracture occurred. Although a traumatic incident cannot be ruled out, the owner repeatedly affirmed that the postoperative instructions were strictly followed. The signs of pain and the intermittent signs of radiculitis observed in the dog of our report were likely the result of the inflammation and possibly the intermittent compression of the L7 nerve roots by the fractured articular facets. Both fractures extended to the intervertebral foramen and were therefore in close proximity to the nerve root, particularly on the right side. The removal of the loose fragments and stabilization of the vertebral segment rapidly improved the clinical signs. Strict exercise restriction must be enforced following extended laminectomies associated with dorsal annulectomies of the lumbar vertebrae in dogs, as their cumulative effect could destabilize the vertebral column to the point of fracture. The fractures were difficult to diagnose on the basis of radiographic findings. Computed tomography or magnetic resonance imaging would have certainly provided a more definitive diagnosis and should be considered in dogs whose clinical signs are suggestive of articular facet fracture following dorsal laminectomy.

References

1. Janssens LAA, Moens Y, Coppens P, et al. Lumbosacral degenerative stenosis in the dog: the results of dorsal decompression with dorsal anulectomy and nuclectomy. *Vet Comp Orthop Traumatol* 2000;13:97–103.
2. Lenehan TM. Canine cauda equina syndrome. *Compend Contin Educ Vet Pract* 1983;5:941–950.
3. Palmer RH, Chambers JN. Canine lumbosacral diseases. Part I. Anatomy, pathophysiology, and clinical presentation. *Compend Contin Educ Pract Vet* 1991;13:61–64.
4. Palmer RH, Chambers JN. Canine lumbosacral diseases. Part II. Definitive diagnosis, treatment, and prognosis. *Compend Contin Educ Pract Vet* 1991;13:213–222.
5. Ramirez O, Thrall DE. A review of imaging techniques for canine cauda equina syndrome. *Vet Radiol Ultrasound* 1998;39: 283–296.
6. Janssens LAA. Thoracolumbar disc herniation in the dog. *Vlaams Diergen Tijds* 1990;59:128–136.
7. Slocum B, Devine T. L7-S1 fixation-fusion for treatment of cauda equina compression in the dog. *J Am Vet Med Assoc* 1986;188: 31–35.
8. Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. *J Spinal Disord* 1992;5:383–389.
9. Bruecker KA. Principles of vertebral fracture management. *Semin Vet Med Surg (Small Anim)* 1996;11:259–272.
10. Viateau V, Preault H, Moissonnier P, et al. Caracterísation du comportement biomecanique du rachis lombo-sacre chez le chien. Particularites associées à la spondylose et à la dégénéréscence discale. *Chirurgie* 1994;120:94–99.
11. Smith GK, Walter MC. Spinal decompressive procedures and dorsal compartment injuries: comparative biomechanical study in canine cadavers. *Am J Vet Res* 1988;49:266–273.
12. Macy NB, Les CM, Stover SM, et al. Effect of disk fenestration on sagittal kinematics of the canine C5-C6 intervertebral space *Vet Surg* 1999;28:171–179.
13. Schulz KS, Waldron DR, Grant JW, et al. Biomechanics of the thoracolumbar vertebral column of dogs during lateral bending. *Am J Vet Res* 1996;57:1228–1232.
14. Hill TP, Lubbe AM, Guthrie AJ. Lumbar spine stability following hemilaminectomy, pediculectomy, and fenestration. *Vet Comp Orthop Traumatol* 2000;4:165–171.
15. Ethier DB, Cain JE, Yaszemski MJ, et al. The influence of anulotomy selection on disc competence. A radiographic, biomechanical, and histologic analysis. *Spine* 1994;19:2071–2076.
16. Solomonow M, Zhou BH, Harris M, et al. The ligamento-muscular stabilizing system of the spine. *Spine* 1998;23:2552–2562.
17. Stubbs M, Harris M, Solomonow M, et al. Ligamento-muscular protective reflex in the lumbar spine of the feline. *J Electromyogr Kines* 1998;8:197–204.
18. Fossum TW. Surgery of the lumbosacral spine. In: Fossum TW, ed. *Small animal surgery*. St Louis: Mosby, 1997; 1131–1149.
19. Rosen C, Rothman S, Zigler J, et al. Lumbar facet fracture as a possible source of pain after lumbar laminectomy. *Spine* 1991; 16(suppl 6):234–238.