

Surgical management of a Schmorl's node in an Airedale Terrier and review of the literature

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Keywords

Schmorl's node, lumbosacral spine, dog, spinal fusion, vertebral body defect

Summary

Objective: To describe a clinical case of Schmorl's node affecting the lumbosacral disc in an Airedale Terrier including surgical management, short-term outcome, and review of the literature.

Methods: A five-year-old male Airedale Terrier with signs of chronic spinal pain and right hindlimb muscle fasciculation was diagnosed with a Schmorl's node with computed tomography. Repeat imaging performed two months later identified enlargement of the defect in the seventh lumbar vertebra (L7) and herniation of the lumbosacral disc into the spinal canal.

Results: Dorsal laminectomy and discectomy were performed and the defect was treated with curettage and stabilization of the L7 and first sacral vertebra disc space with pins and bone cement. Immediately postoperatively, the patient had proprioception deficits in the hindlimbs and decreased right patellar reflex. Over the next four months the dog's neurological condition improved and no neurological or gait deficits were present six months postoperatively.

Clinical significance: Schmorl's node may be a cause of signs of chronic pain in dogs. Successful management may be achieved surgically, although in the case reported here, recovery was prolonged. To the authors' knowledge, this is the first report of progressive enlargement of a Schmorl's node in a dog.

plate with disc space collapse and radiolucency within the vertebral body (6, 10).

This report describes the clinical presentation and computed tomographic (CT) progression of a Schmorl's node in an Airedale Terrier. Surgical management and short-term outcome are also reported. To the authors' knowledge, this is the first report of a Schmorl's node in the Airedale Terrier breed and documentation of progression of the intravertebral disc herniation and its treatment with spinal fusion.

Case report

A five-year-old male neutered Airedale Terrier was presented for the complaint of diffuse, generalized pain of two months duration. Upon physical examination, the dog showed signs of pain upon hip extension and had muscle fasciculation in the right hindlimb proximally in the caudal thigh and quadriceps muscle groups. Neurological, orthopaedic, abdominal ultrasonic, and thoracic radiographic examinations did not identify any other abnormalities. The results of a complete blood count, chemistry panel including serum creatinine kinase and pancreatic lipase immunoreactivity, and urinalysis were all within normal reference ranges for the reporting laboratory. Serology for *Anaplasma*, *Ehrlichia canis*, *Borrelia burgdorferi*, *Rickettsia rickettsii*, *Babesia canis*, *Bartonella henselae*, *Bartonella vinsonii*, and *Dirofilaria immitis* antigens were negative. Cytology of the left carpus and left stifle synovial fluid revealed mild chronic suppurative inflammation with approximately 22% of nucleated cells consisting of non-degenerative neutrophils and 78% mononuclear cells. The right stifle cytology was within normal parameters. Computed tomographic images of the spine revealed a large defect in the caudal

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Introduction

Potential causes of lumbosacral spinal pain in dogs include discospondylitis, vertebral osteomyelitis, vertebral physisitis, disc herniation, extradural synovial spinal juxtafacet cysts, polyradiculoneuritis, idiopathic stenosis, osteochondrosis, neoplasia and inflammatory disease (1–5). Rarely, Schmorl's nodes have been reported as a potential cause of spinal pain in the dog and in humans (6, 7). These nodes are

postulated to be due to intravertebral disc herniation through a vertebral body endplate into the body of a vertebra, and may occur in cervical, lumbar and sacral vertebrae in the dog (6, 8, 9). Pain associated with Schmorl's nodes is thought to arise from disc herniation dorsally and nerve root compression as well as inflammation in the cancellous bone marrow of the vertebral body with exposure to disc material (7). Radiographically, Schmorl's nodes may appear as indentations of the vertebral end-



Fig. 1 A) Dorsal and B) sagittal computed tomographic images of the lumbar spine in a bone window of a five-year-old male Airedale Terrier with a history of chronic pain and right hindlimb muscle fasciculation. There is an approximately 7 mm by 8 mm defect in the caudal endplate of the seventh lumbar (L7) vertebra and an 8 mm by 4 mm gas opacity in the intervertebral space between the L7 and first sacral vertebra. In A) the left side of the image is the dog's right side, and in B) the left side of the image is rostral.

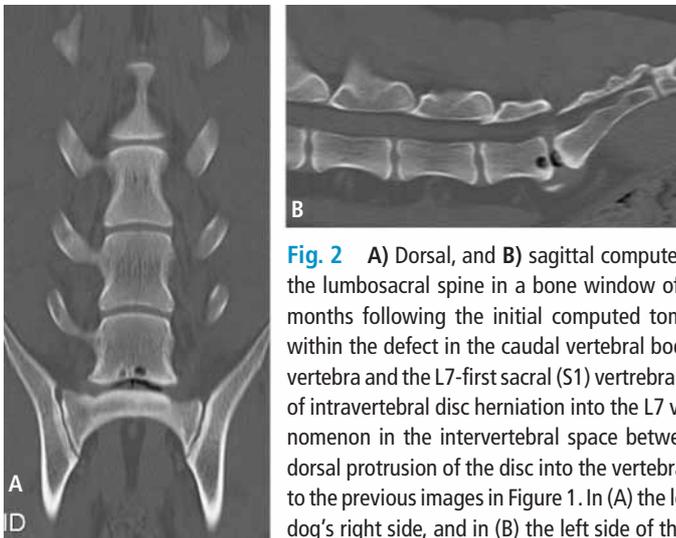


Fig. 2 A) Dorsal, and B) sagittal computed tomographic images of the lumbar spine in a bone window of the dog in Figure 1 two months following the initial computed tomography. Gas is present within the defect in the caudal vertebral body of seventh lumbar (L7) vertebra and the L7-first sacral (S1) vertebra disc, most likely the result of intravertebral disc herniation into the L7 vertebra and vacuum phenomenon in the intervertebral space between L7/S1. Progression of dorsal protrusion of the disc into the vertebral canal is seen compared to the previous images in Figure 1. In A) the left side of the image is the dog's right side, and in B) the left side of the image is rostral.

endplate of the seventh lumbar vertebra (L7) that was approximately 7 by 8 mm and also an 8 by 4 mm gas opacity in the intervertebral space between the L7 and the first sacral vertebra (S1) (► Fig. 1A & B). A fluoroscopy-guided fine needle aspirate of the intervertebral space between L7 and S1 was performed and submitted for culture, however, no aerobic or anaerobic bacteria were recovered. Based upon the computed tomographic and clinical examinations, discospondylitis was suspected. The dog was treated with enrofloxacin^a (136 mg orally once daily for 1 month) along with carprofen^b (75 mg orally every 12 hours), gabapentin^c (100 mg orally every 12 hours), and tramadol^d (100 mg orally every 8 hours). Two months following the initial therapy

the dog exhibited continued muscle fasciculation in the right hindlimb and episodes of vocalization as if in pain. Although no neurological deficits were present, paraspinal palpation of the dorsal lumbar area was resented. Repeat CT without and with intravenous contrast administration showed an enlargement of the gas opacity at the L7-S1 disc space in addition to a new gas opacity within the vertebral body endplate, and cranial progression of the defect further into the body of L7 (► Fig. 2A & B).

^a Baytril: Bayer Health Care LLC, Shawnee, KS, USA

^b Rimadyl: Pfizer Inc, New York, NY, USA

^c Gabapentin: Greenstone LLC, Peapack, NJ, USA

^d Tramadol: Amneal Pharmaceuticals, Glasgow, KY, USA

Additionally, moderate to severe protrusion of the intervertebral disc of L7-S1 into the spinal canal was present.

Dorsal laminectomy with discectomy was performed as previously described (4, 11). The defect in the vertebral endplate of the L7 vertebra was biopsied and surgically curetted. Histopathological analysis of the removed defect indicated the lesion was composed of haematopoietic stem cells, bone fragments, osteoclasts and nucleus pulposus consistent with a diagnosis of a Schmorl's node. Aerobic and anaerobic culture of the removed defect did not yield any bacterial growth. Autologous bone graft harvested from the wing of the left ilium was placed in the vertebral body defect and disc space. Positive profile, end-threaded 4 mm stainless steel pins^e and polymethyl methacrylate bone cement^f was used to stabilize the L7 and S1 vertebra prior to routine closure of the dorsal incision (► Fig. 3) (4, 12). Complications following recovery in the immediate postoperative period included melaena and haematemesis due to presumptive gastric ulceration. This complication was successfully managed by symptomatic treatment including transfusion with two units of packed red blood cells and four units of fresh frozen plasma. Following surgery, there was delayed proprioception in both hindlimbs along with a decreased patellar reflex in the right hindlimb while the sciatic reflexes remained intact bilaterally. The owner reported intermittent lameness in the right hindlimb for four months following discharge from the hospital. At six-months postoperatively, the owner reported that there were no longer any signs of lameness or muscle fasciculation in the right hindlimb and a neurological examination performed by the authors failed to identify any neurological deficits.

Discussion

This report describes the clinical course and surgical management of a Schmorl's node in an Airedale Terrier. The cause of the muscle fasciculation affecting the

^e IMEX Veterinary, Inc., Longview, TX, USA

^f Simplex™ P Bone Cement: Stryker Inc., Kalamazoo, MI, USA

proximal right hindlimb is unknown. It may have been due to compression of nerve roots at the L7-S1 foramen, pain, or anxiety. Following dorsal laminectomy, discectomy and curettage of the defect, the space left was determined to be quite large and resulting instability was felt to be a possible complication postoperatively, therefore, spinal fusion were performed which led to resolution of the clinical signs in the short-term.

A Schmorl's node is defined as a lesion resulting from herniation of disc material into the vertebral body through the cartilaginous end plate of the affected vertebra (10). While these lesions are relatively common in humans, frequently there are no clinical signs present. In rare cases, the signs typically involve lower back pain (7, 13, 14). Two different types of this intravertebral disc herniation exist: central or 'classic Schmorl's node' and retromarginal or 'limbus vertebrae' (10). In humans, the most common vertebrae involved are the thoracolumbar and in dogs it has been reported in the cervical, lumbar and sacral vertebra with the seventh lumbar and first sacral vertebra most commonly affected (6, 8, 9, 15, 16). The thoracic vertebra was affected in the one case that was reported in a sheep (17). Predominantly, males are affected in humans, and of the reports in dogs, where sex of the dog was known, four out of seven were male (6, 8, 15, 16, 18).

Reported affected dog breeds include the German Shepherd (n = 4), Great Dane (n = 1), Cocker Spaniel (n = 1), mixed breed (n = 1), and Standard Poodle (n = 1); in one other case the breed was not reported (6, 8, 9, 15, 16). Of the nine reported cases in dogs, three were incidental findings and the other six were in dogs with clinical signs. The age of the dogs ranged from four months to nine years. The most commonly reported clinical signs in dogs were spinal pain and lameness (6, 8, 15, 16). Only two of the seven reported dogs with clinical signs were treated rather than euthanized, and the treatment involved surgical management with a laminectomy and curettage of the herniated disc and the defect in the vertebral bone. Follow-up was performed for only one dog after surgery, and in that case, the dog had resolution of clinical signs for at least one year following surgery (6).

The aetiology of Schmorl's nodes is unknown but several factors may play a role including breed, mechanical stress, osteochondrosis, and disc degeneration (19). In humans, osteonecrosis of the end plate bone with signs of infarction are commonly present and may be the cause of vertebral body end plate collapse with subsequent herniation of adjacent disc material into the defect (18). Other factors in humans that may play a role include juvenile kyphosis (Scheuermann's disease), trauma (especially axial loading trauma causing

vertebral endplate fractures), age related endplate changes, metabolic and neoplastic diseases, genetic primary or secondary bone dysplasias, and osteoarthritis (7, 10, 20, 21). Reports in dogs include both central and retromarginal lesions and may be associated with osteochondrosis or disc degeneration in some cases (6). Multiple reasons for weakening of the endplates have been described in humans including a developmental weakness where indentations of the endplate subsequent to regression of the chordis doralis leaves a weakened area in the vertebral body endplate (10). Schmorl reported an 'ossification gap' corresponding to a perforation of the endplate present in children that normally disappears in adults (6). The presence of a gas opacity within the disc and associated vertebral body defect, as seen in the dog reported here, is theoretically due to accumulation of nitrogen following crack or fissure of the intervertebral disc and vertebral body, and is termed vacuum phenomenon (22).

Schmorl's node in the dog appears to be a rare cause of clinical signs. The incidence in humans is reportedly higher than in dogs and in many individuals the nodes are quiescent (23). The lower reported occurrence in dogs may be due to the differences in anatomy and biomechanics between the species. Humans have a cartilaginous vertebral endplate while dogs have a solid bone

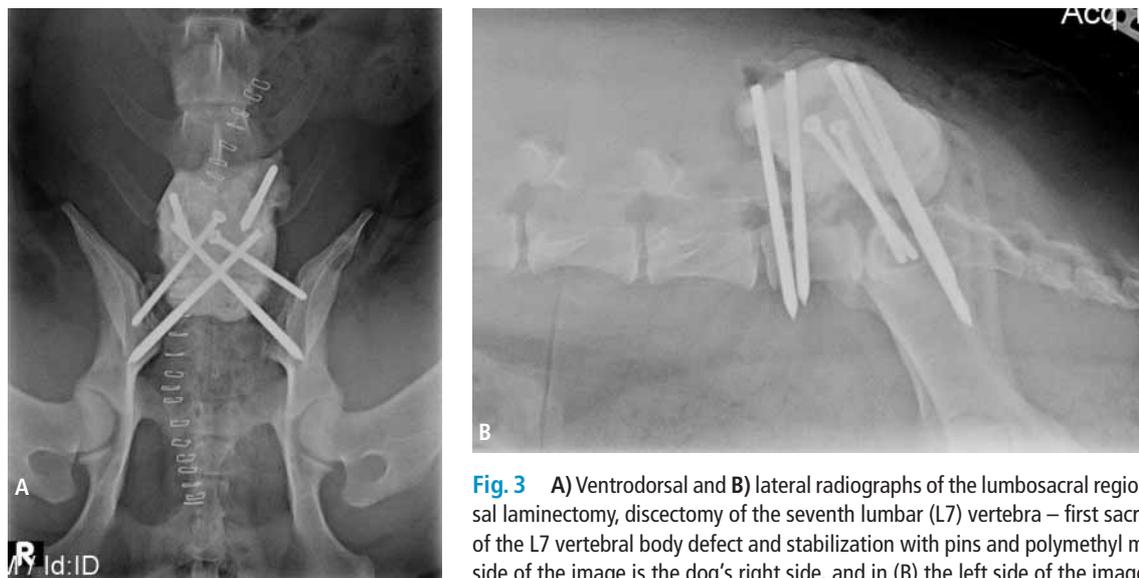


Fig. 3 A) Ventrodorsal and B) lateral radiographs of the lumbosacral region in the dog following dorsal laminectomy, discectomy of the seventh lumbar (L7) vertebra – first sacral (S1) vertebra, curettage of the L7 vertebral body defect and stabilization with pins and polymethyl methacrylate. In (A) the left side of the image is the dog's right side, and in (B) the left side of the image is rostral.

vertebral endplate, theoretically making dogs more resistant to intravertebral disc herniation. In addition, humans are bipeds and thus different biomechanical forces are placed on the discs and vertebral endplates than in dogs (9). Developmental differences of the discovertebral junction exist between the species as well. The human vertebral endplate has developed with a more porous surface while the canine vertebral endplate consists of a thick sheet of bone (10). Due to the low number of reported cases, no breed predisposition can be shown, however German Shepherds are predisposed to osteochondrosis of the vertebral endplate which would, at least theoretically, predispose them to the formation of Schmorl's nodes.

Previous reports of clinical signs in dogs most commonly include signs of back pain and pain upon palpation of the spine, both of which were found in the dog in this report (6). Hindlimb lameness, paresis and paralysis have been less consistently noted in dogs, however with so few reported cases, conclusions regarding onset and clinical presentation cannot be made (6, 8, 9). In humans, symptoms of spinal pain occur only in cases where bone marrow inflammation and oedema are present surrounding the vertebral defect. Inflammation caused by intraosseous fracture and biological reaction to intraspongious disc material is thought to cause the signs of pain (7, 14, 20). In dogs, the cause of spinal pain is unknown. Enlargement of the Schmorl's node on repeat CT imaging in the dog reported here, along with continued clinical signs of spinal pain and muscle fasciculation, resulted in the owner's decision for surgical management.

Deterioration of the neurological status of the dog following dorsal laminectomy, discectomy and spinal fusion may have been due to manipulation of the nerve roots during surgery, infarction of the bone secondary to curettage, or inflammation resulting from curettage of the L7 defect and autologous bone graft placement into the defect. The decreased right patellar reflex postoperatively could have been due to excessive traction placed on the cauda equina during the discectomy or placement of the L7 vertebral body pin deep into the psoas musculature ventral to the vertebral

body (Fig. 3B). This could have resulted in laceration to the iliac vessels that lie within millimeters of the vertebral body of L7 (24). Haemorrhage in the psoas muscle and retroperitoneal space in this area can result in haematoma formation in the psoas, iliacus and iliopsoas muscles. The psoas muscle arises from the transverse processes of L2 and L3 and the ventral vertebral bodies of L4-L7; then it joins with the iliacus muscle (which arises from the ilium) to become the iliopsoas muscle that inserts on the lesser trochanter of the femur (25). The femoral nerve passes through the muscle fibers of the psoas and iliopsoas muscles, and with haemorrhage in this area, compression and stretching of the nerve from the haematoma may cause a femoral neuropathy (25–28). While the exact cause remains unknown, resolution of the decreased reflex over time indicates that inflammation might have been involved. Spinal fusion has been used successfully in a human to resolve spinal pain secondary to a Schmorl's node (13).

Magnetic resonance imaging (MRI) may be useful in differentiating symptomatic from asymptomatic Schmorl's nodes in humans since inflammation and oedema appear to be present in symptomatic patients only and is visible on MRI studies (7, 14, 20). The resolution of signs of pain in these patients seems to correlate with the disappearance of endplate oedema visible on MRI. Unfortunately, the extent of endplate oedema does not correlate with the intensity of pain experienced (20). Magnetic resonance imaging was not available for evaluation of our case, but enlargement of the node and protrusion of the L7-S1 disc into the spinal canal two months following the first CT exam indicated that the node was not static and probably the cause of the signs of spinal pain.

Asymptomatic Schmorl's nodes in humans are often considered to be a self-limiting condition and the defect may regress in size over time, therefore, in those asymptomatic cases, surgical management is not recommended (23). Conservative management in humans has been attempted with repeat injection of the ramus communicans with mepivacaine and triamcinolone with resolution of clinical signs possible, however this technique has

not been reported in dogs (14). Repeated epidural infiltration with methylprednisolone acetate has been successful in 53% of dogs with lumbosacral disease, however, the technique has not been reported in dogs with Schmorl's node (29).

Because of the anatomical and developmental differences between humans and dogs, Schmorl's node is unlikely to be as common a diagnosis in dogs. However, Schmorl's nodes must be considered as part of the differential list in a dog with signs of spinal pain and even paresis since these lesions have been associated with disc herniation dorsally (30).

Conflict of interest

The information in the text has not been previously presented at any scientific meeting. There were no conflicts of interest, nor any financial support for this report.

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