DEGENERATIVE LUMBOSACRAL STENOSIS IN DOGS: CURRENT CONCEPTS OF DIAGNOSIS AND TREATMENT

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This paper has been modified from an article appearing the New Zealand Veterinary Journal. *Worth AJ, Thompson DJ, Hartman AC. Degenerative lumbosacral stenosis in working dogs: Current concepts and review. NZVJ, 57(6), 319-30, 2009*

Degenerative lumbosacral stenosis (DLSS) is a common condition amongst active large breed dogs with a complex aetiopathogenesis. Large-breed dogs, especially the German Shepherd (GSD), Retrievers and active or working dogs (Police, military or sporting dogs), are the most commonly affected by DLSS. Affected dogs are generally mature (mean age 5 ½ years), and are predominantly heavier than 25 kg bodyweight. Males are reported to be over-represented.

Pathogenesis

The term degenerative lumbosacral stenosis (DLSS) defines an acquired narrowing of the vertebral canal +/- intervertebral foraminae resulting in compressive radiculopathy of the cauda equina. The L/S joint of large-breed dogs is prone to degenerative change, seemingly related to concurrent degeneration of the intervertebral disc of a form which is remarkably similar to the most common spinal problem in humans. One or more nerves of the cauda equina become compressed by alterations of the soft and bony tissues associated with the spine, coupled with or caused by suspected instability of the L7-S1 intervertebral motion segment. In humans, and presumably canines, the degenerative process that leads to DLSS starts in the intervertebral disc as the result of prolonged stress associated with activity and age, and by changes which occur from strain beyond normal physiological limits. The eventual outcome is fibroid metaplasia and the once gelatinous nucleus begins to protrude through tears in the weakened annulus, resulting in a Hansen Type II disc protrusion. The degenerating annulus cannot absorb and distribute loading, and the resultant tearing of Sharpey's fibres leads to circumferential formation of osteophytes around the lumbosacral joint.

The intervertebral disc space is narrowed as the disc protrudes dorsally, and this loss of spacing affects the size of the foramen. The L7 nerve root, having left the dura at the level of L6, passes through the lateral recess within the vertebral canal and exits the foramen lateral to the disc and the other nerves of the cauda equina. The foramen is not a simple aperture, but rather a tunnel, with entrance, middle and exit zones. Protrusion of disc material in a dorsolateral direction can affect the entrance and middle zones of the foramen, while impingement of the exit zone can be the result of narrowing of the intervertebral disc space, osteophytosis or hypertrophy of soft tissue in the region of the facets.

The degree of anatomical change affects the nature and severity of the clinical signs detected. Central protrusion causes dysfunction of the sacral and caudal nerves, leading to abnormal carriage of the tail and faecal, and/or urinary incontinence. In contrast, lateral (foraminal) compression of the L7 nerve roots leads to nerve root signature (referred pain down a limb, causing lameness or elevation of the limb, resulting from entrapment of the spinal nerve), with intermittent to progressive lameness and sciatic deficits in severe cases.

Predisposing factors in the development of DLSS

A lesion resembling osteochondrosis has been reported to affect the dorsal end-plate of S1 in the GSD and to occur with increased incidence in dogs with cauda equina syndrome *vs* normal dogs.

GSDs with transitional vertebrae have a higher risk of developing cauda equina syndrome putatively due to the abnormal rotational forces induced by malalignment and malarticulation of the asymmetrical lumbosacral junction. The incidence of transitional vertebrae in GSD has been reported to range from 3.5 to 29%. In a study of more than 4,000 pelvic radiographs there was no association between the presence of a transitional vertebra and hip dysplasia in the GSD. The heritability of a transitional vertebra was estimated to be 0.2 to 0.3. Dogs with lumbar transitional vertebrae were eight times more likely to develop cauda equina syndrome than dogs without lumbar transitional vertebrae, and GSDs were eight times more likely to develop cauda equina syndrome than other breeds, and at a significantly younger age dogs with lumbosacral disease have decreased range of motion and increased malalignment of the lumbosacral junction. A significant decrease in the mean foraminal area in an extended *vs* flexed position has been detected in clinically affected dogs. It is hypothesised that one of the normal functions of supportive structures of ventral and dorsal compartment is to maintain the foraminal dimensions of L7-S1 independent of the angle of the lumbosacral.

Principles of diagnosis

The diagnosis of DLSS is based on the presence of pain and dysfunction associated with the lumbosacral joint, combined with supportive imaging findings and the exclusion of alternative differential diagnoses. Diagnosis is hampered by subtle clinical signs which may be confused for other neurological or orthopaedic conditions. Signalment and specific manipulative tests allow the clinician to form a high suspicion for DLSS and initiate investigation however plain radiographic findings are not pathognomic. Reluctance to jump, or pain when jumping or rising from a prone position or climbing stairs and signs of pain or stiffness during physical activity are the clinical observations most frequently cited.

Low back pain on direct digital palpation in the lumbosacral area and associated with hyperextension, plus hindlimb lameness are reported as the most important presenting clinical signs of DLSS. Pain may originate from entrapment of nerve root (radicular pain); degeneration or tearing of soft tissues surrounding the joint such as the annulus fibrosus (discogenic pain), longitudinal ligaments, joint capsules, and periosteum; or irritation of the overlying dura mater (meningeal pain).

Other clinical signs include hindlimb weakness and/or ataxia, urinary and/or faecal incontinence, and a flaccid tail. Careful palpation may reveal atrophy of the gluteal or stifle flexor muscles. Examination should include finger pressure applied dorsally over the lumbosacral space with and without extension of the hips, tail jack, and the 'lordosis test'. Pain elicited on extension of the hip is not specific for lumbosacral disease as it can be associated with either spinal or coxofemoral joint pain. Pain on extension and abduction or rotation of the hip is more suggestive of coxofemoral joint pain.

Presence of pain during lordosis and extension of the hip with absence of pain on abduction or rotation is suggestive of lumbosacral or lumbar spinal pain. Neurological examination may reveal depressed cranial tibial, ischiatic and withdrawal reflexes, and normal to exaggerated patellar reflexes. This 'pseudo-exaggeration' is the result of flaccidity of the flexor muscles of the stifle, which antagonise the patellar reflex and should not be confused with upper motor neuron disease.

Diagnostic Imaging

Diagnostic investigation of cauda equina syndrome begins with plain radiographs of the lumbosacral joint to rule out bone-associated neoplasia, discospondylitis, trauma, and vertebral abnormalities. Plain radiographic signs of DLSS include ventral spondylosis, narrowing of the intervertebral disc space, vertebral sclerosis at the caudal aspect of L7 and the cranial aspect of S1, and subluxation of the vertebral canal at the lumbosacral joint. However, plain radiography has poor accuracy due to both false-positive (presence of degenerative changes without clinical signs) and false-negative (due to inability to image soft tissue structures) diagnoses being common.

Advanced imaging is considered essential for an accurate diagnosis but may highlight non-symptomatic lesions, therefore clinical judgement remains paramount. *Computed tomography (CT)* offers many advantages including better contrast resolution of soft tissue, cross-sectional images and the ability of the computer to reformat dorsal and sagittal images from transverse planes. With the advent of multi-slice helical scanning devices, the generation of high-resolution volume datasets have remarkably improved the ability to three-dimensionally reconstruct the lumbosacral region, allowing more precise assessment of the fine detail and reducing the amount of volume-averaging artefact. CT allows evaluation of the lateral recesses of L7, intervertebral foramenae, articular processes, and the extent of any bulge or prolapse of the dorsal annulus.

However, it must be remembered that CT abnormalities may be clinically insignificant, especially in older dogs. Bulging of the dorsal disc as a proportion of the height of the spinal canal has been reported to average 27% in normal dogs therefore care must be taken not to over-interpret the degree of disc protrusion. In patients with clinical signs consistent with cauda equina syndrome, compression should be suspected at the locations where there is an increase in opacity of perineural soft tissue together with the absence of epidural fat.

Magnetic resonance imaging (MRI) is the most sensitive imaging modality for detecting degeneration of the nucleus pulposus due to fibroid metaplasia, which characterises disc degeneration in large-breed dogs. Loss of signal due to dehydration of the disc is best seen in T2-weighted images. MRI can document attenuation of the normal epidural fat signal indicative of foraminal compression of nerve roots.

Despite these advances in imaging, the results of imaging studies, including CT and MRI, may be less important than clinical or surgical factors in predicting an outcome in any affected dog with DLSS.

Treatment

Both surgical and conservative treatment has been advocated for DLSS. Conservative management has been recommended for those dogs with pain only and whose lifestyle can be modified to avoid strenuous exercise. Non-steroidal anti-inflammatory drugs (NSAIDs) can be effective for temporary pain relief, and recommendations for exercise restriction vary from 8 to 10 weeks. Relapse is common after conservative therapy and most if not all of the surgical candidates in retrospective studies have had unsatisfactory conservative management prior to surgical treatment. There is anecdotal evidence that corticosteroids are more effective than NSAIDs but clinical signs typically return when either medical therapy is discontinued. Nevertheless, repeated epidural infiltration of methylprednisolone was beneficial at reducing clinical signs in 30/38 (79%) dogs in a preliminary study, and 20 were considered by their owners to be free of signs (median follow-up 48 months) after a median of five injections (Janssens *et al.* 2009).

Surgical therapy for DLSS is directed at decompression of the cauda equina and/or nerve roots and/or stabilisation of the lumbosacral joint. The most widely reported decompressive surgical technique involves a dorsal laminectomy of L7 and S1, with a dorsal annulectomy to remove any prolapsed disc annulus. Other studies have used dorsal decompression alone, or with fenestration and partial discectomy. Traditional decompression by dorsal laminectomy alone does not provide good access to the L7 foramen, which may result in continuation of clinical signs in dogs with impingement of the foramen. Techniques for enlargement on the foramen include facetectomy, an extension of the dorsal laminectomy beneath the articular facet of L7, and a dorsolateral approach from the outside of the foramen. Facetectomy is no longer recommended due to its potential to induce instability, and a dorsal approach runs the risk of incomplete decompression of the middle and exit zones of the L7 foramen. The recently described dorsolateral foraminotomy involves removing bone from the pedicle, directly over the lateral recess, allowing decompression of the exit and middle zones of the neuroforamen (Godde and Steffen 2007). This new approach offers a means of foraminal decompression with less destabilisation of the caudal facets of L7. Initial results are encouraging.

In 1986 Slocum and Devine advanced a technique for facet joint fixation and bone grafting to permanently fixate the lumbosacral joint in a neutral position. They proposed that hypertrophy of soft tissue causing compression of neural structures was secondary to instability and would regress once the lumbosacral joint was rigidly fixated. In their technique, partially threaded pins or screws are placed through the articulation of the facet joint and an autogenous bone graft is placed dorsally, to promote spondylosis.

Screw fracture and failure of the fixation of the screw have been observed, and concurrent dorsal laminectomy of the caudal aspect of L7 results in facet weakening, making the facet prone to fracture. For this reason, some surgeons limit the laminectomy to the cranial aspect of S1 or do not perform a laminectomy in conjunction with fixation. Others have used specialised medical devices or the 'string of pearls' (Orthomed, Huddersfield, UK) locking plate for dorsal fixation/fusion. This method avoids loading the facets, instead placing screws in the pedicles, allowing more aggressive laminectomy/dorsomedial foraminotomy, with decreased concern over weakening of the facet joint.

Prognosis

The prognosis for dogs with DLSS decreases with age, intended use, and presence of neurological deficits. The results of several studies using dorsal decompression and annulectomy yielded a mean total percentage cure of 55%, and improved the clinical signs in a further 25% of dogs with DLSS. Recurrence of clinical signs resembling DLSS is reported by the owner or diagnosed by clinical examination in 18-37% of dogs. Development of a laminectomy scar membrane or failure to address foraminal stenosis may have contributed to those failures. In some studies there were no significant differences in outcome between those treated with dorsal laminenctomy alone and those that underwent an additional procedure. It is likely that annulectomy or fenestration further destabilises the already degenerate lumbosacral disc and induces greater collapse of the disc space. A significant correlation has been found between the presence of urinary or faecal incontinence prior to surgery and a poorer outcome. The prognosis for surgical treatment of working dogs with DLSS is apparently poorer than for pet animals.

The lateral foramenotomy procedure recently described may represent a significant improvement in the management of DLSS. In a retrospective clinical study, Godde and Steffen (Vet Surgery 2007) reported that 19/20 dogs with DLSS had a good to excellent outcome after surgical intervention, at a mean follow-up of 15 months. The improvement in outcome over previous studies was attributed to the effect of either unilateral or bilateral lateral foramenotomy, which was performed on each dog as dictated by MRI findings. Osteophyte formation at the exit zone and proliferations of soft tissue were the most common findings at surgery.

The utility of lateral foramenotomy has yet to be proven in a prospective clinical study, or in a cohort of working dogs, and it remains to be seen if long-term constriction of the neuro-foramen will recur from the formation of osteophytes or scar following foramenotomy. The long-term results of dorsal fixation/fusion have only been reported in two small-scale studies (Slocum and Devine 1986; Meheust *et al.* 2000), with a total of 13 dogs, all reportedly having good post-operative outcomes. Surgical decision-making should therefore be based on the presence or absence of ventral midline disc protrusion, foraminal involvement by changes in discs/osteophytes/articular facets, and the presence of instability/subluxation. The main controversy remains as to the desirability of fusion and the safest method with which to produce it.

Physical examination of patients with weakness of the pelvic limb/s

1. Examination of the gait, including distance examination, walking/trotting, ascending/descending stairs, turning corners
2. General physical examination, looking for other concurrent/related conditions
3. Orthopaedic and spinal neurological examination

NB An absence of neurological signs does not rule out degenerative lumbosacral stenosis

1. Test postural reactions in the standing patient (knuckling, hemi-walk, hopping)
2. Perform palpation of the pelvic limbs for muscle mass, and the tail for tone
3. Test perineal sensation and anal tone
4. Test myotactic and withdrawal reflexes in lateral recumbency
5. Complete an orthopaedic examination below the hip, with the dog in lateral recumbency
6. Return the patient to standing, and abduct and flex the hip
7. Palpate for spinal pain, with the patient standing, starting away from the lumbosacral joint
8. Palpate the lumbosacral joint - first without, then with, extension of the hip
9. Perform the lordosis test, hyperextending both hips, with concurrent pressure on the lumbosacral joint (standing or recumbent techniques)
10. Perform a tail jack; lift the dog gently until the hindlegs lift off the ground, or upon eliciting a painful response