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Lumbosacral instability and foraminal stenosis: solved or unsolved problems?

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ETIOLOGY AND PATHOGENESIS

Lumbosacral vertebral canal stenosis (also called lumbosacral instability, lumbosacral malformation/malarticulation, lumbar spinal stenosis, lumbosacral spondylolisthesis, and cauda equina syndrome) is a term that encompasses a spectrum of disorders that result in narrowing of the lumbosacral vertebral canal with resulting compression of the cauda equina. The term "cauda equina syndrome" describes a group of neurologic signs that results from compression, destruction, or displacement of those nerve roots and spinal nerves that form the cauda equina by a variety of causes, including lumbosacral vertebral canal stenosis.

The term lumbosacral vertebral canal stenosis is used by this author to describe an acquired disorder of large breeds of dog that results from several or all of the following: type II disk protrusion (dorsal bulging of the annulus fibrosus), hypertrophy and/or hyperplasia of the interarcuate ligament, thickening of vertebral arches or articular facets, and (infrequently) subluxation/instability of the lumbosacral junction. It is likely that several separate disorders currently are included within this single syndrome. Other terms have been used to describe this syndrome, including lumbosacral spondylolisthesis, lumbar spinal stenosis, and lumbosacral instability. In humans, the term spondylolisthesis refers specifically to a forward (anterior) movement of a lower lumbar vertebra relative to a lumbar vertebra or sacrum directly below it. This problem rarely occurs in dogs, in which the most frequently encountered problem is a ventral "slippage" of the sacrum relative to the body of the L7 vertebra. The term retroolisthesis has been proposed to describe this "reverse spondylolisthesis" of dogs. Lumbar spinal stenosis is a term that perhaps is best used to describe a congenital ("idiopathic") syndrome reported to occur in young dogs. Lumbosacral instability is a misleading term, as instability is not demonstrated consistently in association with lumbosacral vertebral canal stenosis.

Certain similarities between vertebral and soft tissue alterations seen in dogs with lumbosacral vertebral canal stenosis and Doberman pinscher dogs with caudal cervical spondylomyelopathy have been noted. As the etiology and pathogenesis for either condition are incompletely understood, such comparisons are of little significance at the present time. Recently, an association has been reported between lumbosacral stenosis and transitional vertebrae in German shepherd dogs. In another recent report, more than 30 per cent of German shepherd dogs with clinical signs of cauda equina compression had radiographic and pathologic abnormalities compatible with osteochondrosis of the sacral end-plate.

CLINICAL FINDINGS

Acquired degenerative lumbosacral vertebral canal stenosis occurs most commonly in largebreed dogs especially German shepherd dogs. Males appear to be affected more frequently than females. Dogs with the congenital ("idiopathic") form appear to be of the smaller breeds. Affected dogs in both categories are between 3 and 7 years of age, although the problem may occur at any age. Degenerative lumbosacral vertebral canal stenosis rarely is recognized in cats.

Signs of cauda equina compression seen frequently in affected dogs include the following: apparent pain on palpation of the lumbosacral region, on caudal extension of the pelvic limbs, or on elevation of the tail; difficulty rising; pelvic limb lameness (often unilateral); pelvic limb muscle atrophy; paresis of the tail; and rarely, paraphimosis. These signs most often are insidious in onset and progress gradually over months, and they are easily confused with those of hip dysplasia or degenerative myelopathy.

Abnormalities detected on neurologic examination include gait deficits related to sciatic nerve paresis (e.g., dragging of toes). In addition, depression or loss of conscious proprioception, normal or slightly exaggerated patellar reflexes ("pseudoxaggeration" related to loss of antagonism to femoral nerve-innervated muscles by sciatic nerve-innervated muscles), depressed or absent flexion reflexes in pelvic limbs, decreased anal tone and anal sphincter reflexes, atonic bladder, hypesthesia of the perineum and tail, and muscle atrophy may be seen. These abnormalities relate to deficits of the sciatic, pudendal, caudal, and pelvic nerves, whose nerve roots comprise the cauda equina.

DIAGNOSIS

Characteristic clinical findings may be consistent with a diagnosis of degenerative lumbosacral vertebral canal stenosis. Careful mapping of areas of loss of cutaneous sensation may assist in determining involved nerve roots. However,
presence of this syndrome must be confirmed by means of plain radiographs and special radiographic procedures. Rarely can this condition be diagnosed on the basis of plain radiographic findings alone.

Plain radiographic findings include spondylosis deformans ventral and lateral to the lumbosacral articulation, sclerosis of vertebral end-plates, "wedging" or narrowing of the L7-S1 disk space, and secondary degenerative joint disease in the region of L7-S1 articular facets. Ventral displacement of the sacrum with respect to L7 ("retrolisthesis") and diminished dorsoventral dimensions of the lumbosacral spinal canal may be seen; however, such findings must be interpreted with caution, as they may be seen in normal dogs in association with slight rotation of the vertebral column on lateral radiographs. Every effort must be made to ensure that such rotation does not occur during exposures for lateral radiographic projections. General anesthesia is mandatory for obtaining radiographs of the lumbosacral vertebral column. A ventrodorsal projection also is recommended.

"Stressed" plain radiographic projections (flexed and extended views), completed with careful attention to avoid rotation, often assist in determining the presence of instability or "retrolisthesis." Several attempts to separate normal dogs from dogs with lumbosacral vertebral canal stenosis by means of objective measurements made from radiographs have not been successful. Appearance on plain radiographs helps to eliminate other causes of cauda equina syndrome (e.g., diskospondylitis or vertebral neoplasia). Linear tomography, when available, may provide specific information regarding the diameter of the lumbosacral vertebral canal that cannot be obtained from plain radiographs.

Electromyography may complement information available from a neurologic examination and from plain spinal radiographs by confirming denervation in muscles innervated by the nerves of the cauda equina. Motor nerve conduction velocity determinations in sciatic and tibial nerves and measurement of evoked spinal cord potentials may also provide indirect evidence of cauda equina dysfunction.

Several contrast radiographic techniques exist for examination of the lumbosacral vertebral canal. Use of such techniques is necessary for demonstration of soft tissue vertebral canal stenosis.

Myelography most often is useful in the diagnosis of lumbosacral problems, as the terminal portion of the subarachnoid space of dogs may fill with contrast material at this level. Transosseous vertebral sinus venography (filling of vertebral sinuses with contrast material) and epidurography (filling of the lumbosacral epidural space with contrast material) have been used by many investigators in an attempt to outline soft tissue stenosis of the lumbosacral vertebral canal. Results obtained with either of these techniques must be interpreted cautiously, as falsely positive studies occur with both.

A technique that is useful for confirmation of lumbosacral soft tissue stenosis is diskography. Diskography consists of radiography completed following the injection of contrast material into the nucleus pulposus of an intervertebral disc.

This technique has special application to the lumbosacral disk space.

Computed tomography, either alone or combined with the contrast techniques listed above, and MRI, may provide further information regarding soft tissue stenosis of the lumbosacral vertebral canal, particularly with regard to the L7-S1 intervertebral foramen.

Surgical exploration may be indicated in dogs (with appropriate history and clinical signs) in which results of ancillary diagnostic tests do not provide a definite diagnosis of soft tissue stenosis.

TREATMENT

Some affected dogs in which clinical signs are mild or in which apparent lumbosacral pain is the sole problem improve temporarily after strict confinement and restricted leash exercise for a period of 4 to 6 weeks. Use of analgesic drugs or corticosteroids has been recommended; however, their use must be accompanied by strict confinement.

Clinical signs commonly recur in affected dogs treated only by means of medical therapy. Dogs with recurrence of signs, or dogs that are moderately to severely affected at the time of initial presentation (especially those with urinary/ fecal incontinence), should be considered candidates for surgical therapy. Dorsal decompressive laminectomy of L7 and S1 vertebrae is recommended. This procedure may be combined with foraminotomy or facetectomy in dogs in which compression of spinal nerves at the level of the intervertebral foramina is suspected. In animals with radiographically confirmed instability or significant retrolisthesis, fusion of the lumbosacral articulation may be necessary. A dorsal approach for fusion has been recommended.

Dogs should be confined for 2 to 4 weeks postoperatively. Postoperative complications include seroma formation at the surgical site and formation of a laminectomy scar at the site of the laminectomy. Both may be avoided by use of appropriate surgical technique and postoperative patient management.

Attention to bladder emptying may be necessary in dogs with bladder atony prior to surgery. The bladder should be manually expressed three times daily in such dogs. Urine should be submitted for culture and sensitivity testing prior to and 2 weeks after completion of surgery, and appropriate antibiotic therapy instituted as indicated by results.

Prognosis for affected dogs is dependent on the severity of signs prior to surgery. Return to normal function may be expected in dogs that are mildly affected prior to surgery. Dogs with bladder atony or a flaccid anal sphincter prior to surgery have the poorest prognosis.

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