CANINE LUMBOSACRAL DISEASE

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Brent Higgins discusses differing approaches to this group of diseases, and argues that while conservative management can help, it can only go so far.

CANINE lumbosacral disease is the term used for a group of diseases affecting the lumbosacral intervertebral joint and the surrounding tissues.

These diseases can occur in any dog, but large breeds, especially German shepherd dogs, are most commonly affected. This article discusses this group of diseases from a practical and surgical viewpoint.

The lumbosacral joint is made up of three joints – the lumbosacral disc and two synovial facets (Figure 1). It is known as the lower back’s hinge joint, as it has a larger range of motion than the rest of the lower spinal vertebrae.

The cauda equina (Figure 2) travels through the lumbosacral vertebral canal. This is a collection of nerve roots that innervate the pelvic limbs, tail, urinary system and anus.

The spinal cord itself usually terminates at the fifth or sixth lumbar vertebrae, although the dural sac often extends further caudally, especially in smaller dog breeds. From the cauda equina, the seventh lumbar (L7) nerve courses through the lumbosacral intervertebral foramen (Figure 3) to the lumbosacral nerve plexus, becoming part of the sciatic nerve to the pelvic limb.

Conditions involved
Lumbosacral disease includes the following conditions:

- degenerative lumbosacral stenosis (DLSS);
- lumbosacral intervertebral disc disease;
- lumbosacral discospondylitis;
- lumbosacral vertebral fractures;
- lumbosacral neoplasia;
- congenital lumbosacral stenosis; and
- lumbosacral fibrocartilagenous embolism.

DLSS is the most common of these diseases, and will be explored in this article.

The large mobility of the lumbosacral joint results in extreme stresses being placed upon it. This leads to joint degeneration, including disc degeneration and subluxation of the sacrum, thus causing further instability. As a result, osteophytosis occurs (seen as spondylosis on a lateral radiograph) and the soft tissues hypertrophy (disc protrusion, thickening of the joint capsules of both the disc and facets, and thickening of the ligamentum flavum). These changes lead to compression of the cauda equina as it passes through the lumbosacrum, and compression of the L7 nerve as it exits its intervertebral foramen (Figure 3).

Compression of these nerves will initially cause lower back pain, which is the most common clinical sign of lumbosacral disease. Owners may give a history of the patient not wanting to jump or climb stairs, stiffness, pelvic limb lameness and, as the disease progresses, urinary or faecal incontinence.

On clinical examination, lower back pain is elicited by pressing firmly on the dorsal aspect of the lumbosacrum (Figure 4), pressing dorsally per rectum or by jacking the tail dorsally. Generalised muscle atrophy affecting the pelvic limbs may also be present.

In the early stages, or in mild cases of DLSS, the neurological examination is often normal. Dogs may have reduced hock flexion on the pedal withdrawal test, or have increased patellar reflexes due to pseudohyperreflexia. This occurs when the hamstring muscles (innervated by the sciatic nerve – nerve roots L6-S2) have atrophied due to nerve root compression. In contrast, the femoral nerve is not affected by lumbosacral disease as its nerve roots (L4-6) exit the vertebral column, cranial to the lumbosacrum. Therefore, a normal reflex from the quadriceps muscle group (innervated by the femoral nerve) during testing of the patellar reflex can be perceived as
exaggerated because quadriceps action is not limited by hamstring tone.

Pseudohyperreflexia must be differentiated from true hyperreflexia, which occurs in thoracolumbar spinal disease. These dogs have strong pedal withdrawal reflexes and, if acute, no muscle atrophy. The foot may also “resonate” after the patellar tendon is struck.

These clinical signs are not all exclusive to lumbosacral disease, and other common differential diagnoses to consider include hip dysplasia (which is common in large-breed dogs), degenerative myelopathy (which is common in German shepherd dogs), thoracolumbar intervertebral disc disease (common in small and large-breed dogs) and bilateral cruciate disease (one of the most common canine orthopaedic diseases).

Diagnosis, therefore, requires careful evaluation of the history, clinical signs and imaging findings. Plain ventrodorsal and lateral radiography of the caudal lumbar spine and pelvis will allow evaluation for fractures (Figure 5), bony tumours (Figure 6), hip dysplasia and possibly discospondylitis (Figure 10). Beware of overinterpreting spondylosis deformans (new bone formation ventral to the vertebral bodies) as many normal dogs will have spondylosis.

The ideal advanced imaging modality is computed tomography (CT) or magnetic resonance imaging (MRI), but myelography can be helpful in some dogs. This is performed by injecting a lowosmolar, non-ionic, iodinated positive-contrast agent (such as iohexol), at ideally the L5-6 subarachnoid space, or at the cerebello-medullary cistern (and tilting the table for 10 minutes so that the contrast flows caudally).

Myelography must be done carefully to avoid damage to the spinal cord and, if a referral is being contemplated, it is best done by the referral surgeons themselves. Myelography is limited by the fact that the dural sac does not travel into the lumbosacrum in all dogs. CT or MRI (Figures 7, 8 and 9) is often favoured, but imaging findings do not always correlate well with clinical or surgical findings. Novel imaging methods are currently under further investigation at the University of Liverpool (supported by the RCVS Trust) to improve diagnostic accuracy.

Treatment depends on accurate diagnosis. DLSS, intervertebral disc disease and congenital lumbosacral stenosis cause chronic nerve root compression. In the case of DLSS, 50 per cent of dogs may respond positively to conservative management – exercise restriction (lead-only exercise of five to 10 minutes daily for at least six weeks, followed by very gradual increases in lead-only exercise).

However, the clinical signs may recur following the onset of unrestricted exercise. Antiinflammatory drugs and weight loss may also be helpful. Surgical management requires neurosurgical experience or specialised training. Dorsal laminectomy is the procedure of choice to decompress the cauda equina, and around 80 per cent of dogs will have improved postoperative function. With the patient positioned in sternal recumbency and with the pelvic limbs flexed to open the
lumbosacral space, a dorsal midline incision is made over the lumbosacrum and the soft tissues are retracted. The caudal aspect of the L7 laminae and the cranial aspect of the S1 laminae are burrowed to eggshell thickness and rongeurs or kerrisons are used to complete the laminectomy. Nerve roots are retracted laterally and the protruding thickened lumbosacral annulus is excised. Complications include nerve injury, haemorrhage and infection. Fibrosis at the site of laminectomy may cause a recurrence of clinical signs in the long term. Case selection of dogs for surgery is important, as animals with pre-operative urinary and faecal incontinence have a poor prognosis. Other surgical techniques, including stabilisation of the lumbosacrum using implants and decompression of the intervertebral foramen, are also available.

Dogs with discospondylitis (Figures 10 and 11) will present either acutely or chronically. The area will be very painful and the patients will have bouts of pyrexia and anorexia. Treatment involves identification and treatment of the underlying cause, which may include immune dysfunction (such as hyperadrenocorticism or diabetes mellitus) or infection elsewhere in the body (dental, urinary or skin disease). If no aetiology can be found, it is worth performing urine bacteriology, taken by cystocentesis, as subclinical urinary tract infections are a common source of haematogenous infection. These dogs usually require aggressive antibiotic treatment for at least six weeks, and often much longer.

Dogs with fibrocartilagenous embolism present acutely with paraparesis or paraplegia, but the site is not painful. These dogs may recover after days or weeks of nursing care. Dogs with neoplasia (Figures 6 and 7) or traumatic injuries (Figure 5) have a more guarded prognosis.

Summary

This article aimed to highlight lumbosacral disease as a differential diagnosis to consider in lower back pain cases. More common diseases, like hip dysplasia, cruciate disease and degenerative myelopathy, may cause similar signs. However, a thorough history, clinical and neurological examination and further imaging will rule out these diseases from the differential list. Conservative management may be helpful in some cases, depending on the aetiology of the disease, but surgery is often required for a satisfactory outcome.

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Further reading

Figure 1. Dorsolateral view of the seventh lumbar vertebrae (L7), sacrum and paired lumbosacral intervertebral facets (arrows).
Figure 10. Lateral spinal radiograph of a dog’s lumbosacrum showing widening of the lumbosacral disc space, loss of disc end plate definition and sclerosis of the vertebral bodies around the disc. This dog was diagnosed with discospondylitis.
Figure 11. Sagittal T2-weighted MRI of the lumbosacrum of a boxer dog with lumbosacral discospondylitis. This image shows disruption of the lumbosacral disc and irregular destruction of the adjacent vertebral endplates. This dog did not completely respond to medical management and surgery was required.
Figure 2. The cauda equina.

Photo courtesy of FAY PENROSE/UNIVERSITY OF LIVERPOOL.
Figure 3. A ventrolateral view of the lumbar spine and pelvis, focusing on the intervertebral foramina (arrowed), from which the nerve roots exit to become the femoral, sciatic, obturator and pudendal nerves.

Figure 4. The lordosis test involves the extension of the lumbosacral joint. Pressure is put
on the lumbosacrum from a dorsal direction. Note that the dog is supported under the pelvis, so that the hips are not extended – this helps to avoid coxofemoral pain during the test and subsequent misdiagnosis of coxofemoral osteoarthritis.

Figure 5 (left). Lateral spinal radiograph of a German shepherd dog’s lumbosacrum, showing a fracture of the L7 vertebral body. There is cranioventral displacement of the caudoventral aspect of the L7 vertebral body.
Figure 6 (right). Lateral spinal radiograph of a dog’s lumbosacrum, showing altered margination of the ventral surface of the L7 vertebral body and subtle lysis associated with a vertebral tumour. Note also the irregular, wispy soft tissue mineralisation and marked soft tissue swelling ventral to the lumbosacrum.
Figure 7 (right). Transverse T2-weighted MRI of the same dog exhibited in Figure 6, showing an L7 vertebral body tumour (horizontal arrow) present ventral to the cauda equina (vertical arrow). There is extensive bone destruction and a large soft tissue component to the mass (hyperintense on this sequence).
Figure 8 (above). Sagittal MRI of the lumbosacrum of a dog showing dorsal protrusion of the lumbosacral disc secondary to Hansen type-two degeneration, resulting in pinching of the cauda equina. There is, additionally, mild ventral subluxation of the body of S1 relative to L7.
Figure 9 (left). A transverse T2-weighted MRI at the level of the lumbosacral disc space, showing unilateral compression of the left L7 nerve, due to foraminal stenosis (large arrow). Note the loss of the normal hyperintense peri-radicular fat on the left compared to the right. The cauda equina (thin arrow) is not being compressed in this view.