Hansen type I disk disease at T1-2 in a Dachshund

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A 7-year-old Dachshund was presented with chronic left thoracic limb lameness and acute neurological deficits to the hind limbs following trauma. A lesion was suspected between C7 and T2 on the basis of neurological examinations. Radiography and myelography identified a calcified intervertebral disk at C7-T1 and an extradural unilateral compressive lesion at T1-2. Computed tomography scans of the cranial thoracic spine revealed extrusion of disk material from the T1-2 intervertebral space resulting in marked spinal cord compression. Intervertebral disk disease is rarely reported at this location. The neurological condition deteriorated after a second myelogram, which was done to examine the thoracolumbar spine. A modified dorsal decompression of T1-2 was performed. The dog was euthanased due to further neurological deterioration 8 days after surgery.

Case report

A 7-year-old, 12.4 kg, male, neutered Dachshund was referred because of pelvic limb weakness. The dog had a 2 year history of left thoracic limb lameness and recent onset of paraparesis after being kicked by a calf 14 days previously. On neurological examination, the dog was nonambulatory but voluntary movements were present in both pelvic limbs. Conscious proprioception was absent in the left pelvic limb and delayed in the right pelvic limb. Bilateral sciatic, patellar and cranial tibial reflexes were assessed as normal but anal tone was increased. Superficial pain sensation was present in both hind limbs. The cutaneous trunci reflex was absent on the left side but present on the right, which suggested unilateral loss of the lateral thoracic nerve either peripherally or centrally between C8 and T2. The sensory and hyperaesthetic levels were determined to be located at the L2-3 and L1-2 intervertebral spaces respectively, and abdominal splinting was present. Thoracic limb muscle mass, proprioception and spinal reflexes were considered normal on neurological examination. Musculoskeletal examination revealed no significant abnormalities. The presumptive diagnosis was HVDD with an acute lesion at L1-2 and a chronic lesion between C8 and T2.

The Dachshund was anaesthetised for survey spinal radiographs and myelography. Plain radiographs revealed a nondisplaced calcified disk at C7 to T1, intervertebral narrowing at T1-2 and the presence of calcified disk material within the spinal canal at T1-2. There were no abnormalities in the thoracolumbar area. A lumbar myelogram was attempted at L5-6 but was inconclusive. A cisternal myelogram revealed a mineralised mass consistent with disk extrusion at T1-2 (Figure 1). The thoracolumbar spine could not be assessed as the contrast attenuated at T1-2. CT of T1-2 showed dorsoventral, paramedian and lateral extrusion of disk material into the spinal canal with spinal cord compression and left-sided lateralisation (Figure 2). The dog had two seizures during anaesthetic recovery. These were controlled with intravenous diazepam.

Neurological examination of the dog revealed no deterioration but the muscle spasm previously thought to be abdominal guarding was now recognised as thoracic splinting. The dog was reanaesthetised the following day and a second lumbar myelogram performed to rule out thoracolumbar lesions. This confirmed the presence of a single extradural compressive lesion at T1-2. Five hours after recovery from anaesthesia, deterioration of neurological function was detected with bilateral absence of deep pain in the pelvic limbs and bilateral loss of the cutaneous trunci reflex at T2. Neurological deficits were not observed in the thoracic limbs.

The dog was reanaesthetised. Cephalexin 40 mg/kg, and dexamethasone sodium phosphate 0.5 mg/kg were administered intravenously at induction. The dog was positioned in ventral recumbency with the thoracic limbs maintained in a crossed position to allow abduction of the scapulae and better surgical access to the cranial thoracic spine. A dorsal approach to T1-2 was taken, with a skin incision extending from C4 to T6. The tendinous raphe was incised along the left side of the dorsal spinous processes with lateral retraction of the rhomboideus and trapezius muscles and left scapula. The origins of the splenius and serratus dorsalis muscles were incised and retracted laterally to expose the nuchal ligament and dorsal spinous processes of the thoracic vertebrae. The long spinal and multifidus muscles were dissected away bilaterally from the lateral surface of the dorsal spinous processes of C6 to T4 to expose the dorsal laminae.

A modified dorsal laminectomy of T1-2 was performed. The dorsal spinous processes, dorsal laminae and medial aspect of the pedicles of T1 and T2 were removed with rongeurs and a pneumatic burr. The spinal cord had localised purplish discoloration and mild oedema. Duodurotm was performed with a bent, disposable, 26-gauge hypodermic needle. Stay sutures were placed in the dura mater to permit retraction of the spinal cord. Disk material was removed from the ventral aspect of the spinal canal with a nerve root retractor and from the more dorsal aspects of the median spinal canal with an angled curette. A small amount of disk material was removed and this seemed incom-
plete but further retrieval was not attempted due to the risk of iatrogenic cord damage. The disk material appeared gritty and calcified consistent with a chronic Hansen type I disk extrusion. A free fat graft was placed over the spinal cord following removal of the disk material. The tendinous raphe was closed with 2-0 polyglyconate and a suction drain inserted because of haemorrhage and significant dead space. The skin was closed routinely.

Postoperative management consisted of a reducing dose of oral prednisolone, manual bladder expression, positioning on an air mattress and frequent turning to prevent pressure sores. Twice daily neurological examinations showed no clinical or neurological improvement with severe conscious proprioceptive deficits in the pelvic limbs, hyperreflexia, absence of pelvic limb deep pain sensation and a bilateral sensory level at T1-2. The dog was discharged 5 days after operation and it developed left thoracic limb paresis 3 days afterwards. Extrusion of the intervertebral disk at C7-T1 or ascending myelomalacia was suspected and the dog was euthanased at this time. Necropsy was not permitted.

Discussion
Degeneration of intervertebral disks can result in protrusion or extrusion of disk material into the spinal canal resulting in spinal cord compression. Degenerative changes can occur in any of the intervertebral disks between C2-3 and L7-S1 but are more common in the cervical, caudal thoracic and cranial lumbar spine.1 IVDD at T1-2 has been reported in one previous veterinary paper at T1-22 and is rarely reported in human medicine.3 The prevalence of IVDD at the adjacent C7-T1 disk space is 1.5 to 2.0%1,4 This is more common in chondrodystrophic breeds such as the Dachshund and Beagle.4

The cranial thoracic location of the disk extrusion is unusual in this case as the disks between T1-2 and T10-11 are stabilised by intercapital ligaments. The intercapital ligaments originate on one rib head, traverse over the dorsal aspect of the intervertebral disk and insert on the contralateral rib head.5 The intercapital ligament, which is closely associated with the dorsal longitudinal ligament and dorsal annulus fibrosus, provides additional resistance to dorsal extrusion of disk material. The dorsal longitudinal ligament is thicker and stronger in the cervical area than the lumbar area and should provide further protection against dorsal extrusion so that most extrusions occur in a lateral or intraforaminal direction.8 This may result in spinal-root compression rather than cord compression. The 2 year history of left thoracic limb lameness in this dog probably resulted from dorso-lateral disk extrusion causing nerve root compression.

The herniation pattern of intervertebral disks is divided into Hansen types I and II.7 Type I is more common in chondrodystrophic breeds such as the Dachshund. The nucleus pulposus undergoes chondroid degeneration and extrudes through the dorsal annulus fibrosus into the spinal canal.7 Chondroid disk degeneration is characterised by increased collagen content and decreased water content of the disk and alteration in specific glycosaminoglycan concentration of the nucleus pulposus.9

Type I disk extrusion often results in more severe clinical signs than type II. The nucleus pulposus of a chondroid disk is often extruded in an acute or subacute fashion but the severity of signs depends on the velocity at which the compressive force is applied, the degree of compression, the duration of compression and the ratio of spinal cord diameter to vertebral canal diameter.9 Type I disk extrusion is often associated with rupture of the vertebral sinuses and haemorrhage into the epidural space.
which can exacerbate spinal cord compression.\textsuperscript{10} Oedema and necrosis of the spinal cord grey and white matter can also occur.\textsuperscript{11} These changes are not observed with type II disk disease, where gradual or mild compression produces demyelination and axonal degeneration.\textsuperscript{11}

The lesion in this case is believed to be an acute presentation of a chronic progressive Hansen type I disk disease. The left thoracic limb lameness was considered to be caused by a nerve root compression associated with disk extrusion at T1-2. Radiculopathy and associated orthopaedic-like pain can occur in up to 50\% of dogs with cervical disk disease.\textsuperscript{12} Hansen type I partial disk extrusion can also produce chronic progressive spinal cord compression.\textsuperscript{13} Motor fibres are more sensitive to chronic compression than smaller sensory fibres and hence sensory loss is unusual with chronic progressive compression.\textsuperscript{13}

The acute onset of neurological deficits in this case was believed to be caused by trauma. Other possible clinical findings with disk extrusion at T1-2 include lower motor neuron signs in the thoracic limbs and Horner’s syndrome. Lower motor neuron signs were probably not observed in this dog as T1 only contributes 29\% of the fibres to the radial nerve.\textsuperscript{14} In one study, only 39\% of cervical lesions showed forelimb motor deficits.\textsuperscript{15}

Myelography is an invasive procedure which can cause temporary or permanent deterioration in neurological status.\textsuperscript{10} Neurological deterioration can result from puncture of the arachnoid membrane, neurotoxicity caused by the contrast agent or a mass effect resulting from an excessive dose of contrast material.\textsuperscript{16} Myelography should be performed if an intervertebral disk extrusion is suspected but not evident on survey radiographs, the lesion on survey radiographs is not consistent with the neurological examination or multiple lesions are suspected. A second myelogram was performed in this case, as a thoracolumbar lesion was suspected on the original neurological examination, but this may have caused neurological deterioration due to excessive volume of contrast material, resulting in exacerbation of the compression or dislodgment of disk material at T1-2.

A recent study recommended CT for preoperative confirmation of the positional relationship between the spinal cord and the affected intervertebral disk.\textsuperscript{17} The CT scan was very useful in this case to determine the direction and extent of disk extrusion. The most common direction for cervical disk extrusions are dorsolateral, paramedian and dorsomedian.\textsuperscript{18} In this case, the laterally extruded disk caused a chronic radiculopathy and the disk material extruded in dorsomedian and paramedian directions resulted in spinal cord compression.

Surgical management of IVDD is indicated where cases are unresponsive to medical management or have recurrent or progressive clinical signs such as pain, paresis or paralysis with preservation of deep pain or absence of deep pain for less than 24 h.\textsuperscript{19} Treatment in this case involved decompression of the T1-2 disk space using a modified dorsal laminectomy.\textsuperscript{20} A ventral approach via excision of the manubrium may have provided better access to intervertebral disk material and enabled fenestration of the calcified disk at C7-T1. This approach was examined in a cadaver dog but access and visibility were considered poor. In humans, combinations of partial median sternotomy and osteotomy of the manubrium permit excellent exposure from C3 to T4 without interference from the great vessels of the mediastinum.\textsuperscript{17}

In a study of 30 cases of dorsal decompression for cervical disk disease in dogs, it was stated that disk material located ventral to the spinal cord was inaccessible,\textsuperscript{21} but McKee reported a disk retrieval rate of 40\% for dorsal laminectomy in thoracolumbar IVDD.\textsuperscript{22} The ability to retrieve disk material significantly improves recovery as this eliminates spinal cord pressure and restores normal tissue perfusion.\textsuperscript{20} Extruded disk material cannot be completely removed from the vertebral canal using dorsal decompressive procedures without significant risk to further cord damage and hence deterioration in neurological condition.\textsuperscript{22,25} Seventy-five percent of cases in which disk material was removed following dorsal laminectomy for thoracolumbar IVDD made a complete recovery, whereas only 28\% recovered when disk material was not retrieved.\textsuperscript{22} In this case, a modified dorsal laminectomy was performed, which resulted in incomplete removal of disk material, manipulation of the spinal cord during disk retrieval and difficulty in removing disk material from the ventral aspect of the spinal canal. This may have caused further damage to the spinal cord and affected postoperative recovery. Hemilaminectomy may have permitted better access for removal of disk material and minimised iatrogenic spinal cord trauma.\textsuperscript{22} In chronic extrusions, the inflammatory response contributes a significant fibrous component to the compressive mass and fibrous adhesions may develop between the extruded disk material and the dura mater.\textsuperscript{14} Fibrous adhesions were not observed in this case.

The acuteness and severity of neurological deficits, especially deep pain sensation and voluntary motor function, correlate with prognosis for a normal return to function.\textsuperscript{23} Deep pain fibres are located in the dorsal aspect of the ventral commissure of the spinal cord. Deep pain is mediated by nociception transmission via unmyelinated nerves called C-fibres which are small, located deep in the white matter of the spinal cord and do not rely on myelin for transmission.\textsuperscript{24} Loss of deep pain occurs due to disruption of these fibres and, as they are relatively resistant to injury, is considered to indicate a severe if not irreversible injury to the spinal cord due to localised and progressive ischaemia, increased intraneuronal calcium and free radical induced lipid peroxidation.\textsuperscript{11} The location of the extruded disk and retrieval of disk material during decompressive surgery may have exacerbated damage to the deep pain fibres.

The use of deep pain perception for prognostication is controversial, as many have reported excellent recovery following decompressive surgery for dogs with absent deep pain sensation. Case series using decompressive procedures and retrieval of disk material for thoracolumbar IVDD have shown recovery rates to voluntary movement following loss of deep pain sensation range from 50 to 100\%.\textsuperscript{16,23,25,26} The duration of deep pain loss is important with recovery rates of 25 to 38\% if performed between 12 and 24 h,\textsuperscript{25,27} 43\% if between 24 and 48 h\textsuperscript{22,24} and less than 5\% if performed after 48 h.\textsuperscript{27} In
References