Spinal instability resulting from bilateral mini-hemilaminectomy and pediculectomy

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Introduction

Intervertebral disc extrusion is a common cause of pelvic limb paresis and paralysis in dogs. It mainly affects chondrodystrophic breeds; for the Dachshund, this risk is 10 to 12 times higher than for other breeds (1, 2). Numerous treatment methods have been reported regarding the management of intervertebral disc disease, including conservative management, fenestration, laminectomy, hemilaminectomy, mini-hemilaminectomy, pediculectomy, foraminotomy and corpectomy; it is generally accepted that non-ambulatory dogs are best treated surgically (3–12). Mini-hemilaminectomy and pediculectomy are less invasive techniques for achieving spinal cord decompression and few associated complications have been reported. This case report describes the occurrence of dorsal laminar instability with secondary spinal cord compression as a complication of bilateral mini-hemilaminectomy and pediculectomy surgery with concurrent fenestration. A surgical stabilisation technique that successfully corrected the problem is documented.

Case History

A 7.7kg, 4.5-year-old, male Dachshund was presented as an emergency case with a 24 hour history of pelvic limb dysfunction of unknown cause. On examination, the dog showed bilateral non-ambulatory paraparesis with minimal voluntary movement, negative proprioception and positive deep pain sensation. The dog was assessed as having a grade 3 spinal lesion (10, 11) of the thoracolumbar spinal cord, most likely due to Hansen type I intervertebral disc extrusion.

Orthogonal radiographs of the thoracolumbar spine, which had been taken under general anaesthesia, showed mineralisation of intervertebral discs (IVD) thoracic (T) 10–11, 11–12 and 12–13. Cisternal puncture and myelography showed poor dural contrast opacification from mid body T12 to lumbar (L)1. Dorsal deviation of the ventral contrast column over the body of T12 and the absence of contrast at T12–13 (Figures 1 and 2) suggested an extradural lesion at either or both T12–13 and T13-L1, the myelogram however did not indicate lateralisation.

A left side mini-hemilaminectomy was performed at T12–13, which revealed dorsal protrusion of the IVD, left-sided spinal cord deviation and prominence of the dural vasculature. Following this, a right-side mini-hemilaminectomy was performed at T12–13, and then extended caudally to include a right-side T13 pediculectomy to the level of the caudal articular process of T13; in both locations no abnormality was found. The left side mini-hemilaminectomy of T12–13 was then extended caudally to include a left pediculectomy of T13 and to expose the T13-L1 foramen; a large amount of extruded nucleus pulposus over (and cranial to) the T13-L1 IVD was identified and removed. The T12–13 and T13-L1 IVD were fenestrated from the left side, granular material consistent with degenerate nucleus was recovered from T12–13, and nothing was recovered from T13-L1. The surgical site was flushed and closed.

The dog was hospitalised and initially made a good recovery from surgery; post-operatively he had voluntary movement in both pelvic limbs but could not walk. On the fifth postoperative day, the dog lost voluntary movement of the pelvic limbs, and on the sixth day, the dog lost deep pain perception in both pelvic limbs. The dog showed signs of pain only when the thoracolumbar spine was directly palpated.

Repeat radiographs of the thoracolumbar spine (Fig. 3) showed widening of the ar-
particular facet joints at T12–13, and ventral subluxation of the cranial aspect of the dorsal lamina of T13 with narrowing of the spinal canal by approximately 40% at the cranial aspect of T13 (height of spinal canal measured 4mm at cranial T13, 6.5mm at caudal T13, 5.5mm for T12 and 6.5mm for L1). Repeat cisternal puncture and myelography (Figures 4 and 5) did not show any contrast opacification caudal to the body of T12.

The previous surgical site was re-explored, and a blood clot at the left mini-hemilaminectomy/pediculectomy site was identified; this did not appear to be causing cord compression and was removed. No further abnormal material was found. Spinal stability was assessed by careful manipulation. Spinal extension resulted in subluxation of the T12–13 facet joints with ventral instability of the cranial aspect of the T13 dorsal lamina; the lamina subluxated directly onto the dorsal aspect of the spinal cord and caused visible cord compression. This was compared to the adjacent vertebrae that did not exhibit such instability.

The subluxation was reduced and the normal anatomical relationship of the laminae of T12 and T13 was restored by flexing the spine using bone holding forceps applied to the dorsal spinous processes of T11 to L1 inclusive, and by placement of a single 2mm screw in each of the four dorsal spinous processes. The surgical site was flushed and closed. Postoperative radiographs showed significant improvement of the T13 laminar subluxation and good implant positioning (Figures 6 and 7).

The dog made an uneventful recovery from surgery and was hospitalised with intensive nursing and physiotherapy. Deep pain sensation was regained in the pelvic limbs four days postoperatively, and voluntary movement was recovered in the right pelvic limb at 13 days and in the left pelvic limb at 15 days. At 26 days the dog was weakly ambulatory and ataxic. Two months postoperatively, the dog was walking with moderate pelvic limb ataxia and reduced proprioception. At five months, the dog was taking 45 minute lead walks and exercising normally except he was no longer jumping; on examination he had mild pelvic limb ataxia and reduced proprioception. Radiographs at two and five months postoperatively showed maintained stability of the T12-T13 articulation with anatomically correct position of the dorsal lamina of T13. No changes were associated with the articular facet positional screws, but radiolucency was observed around the screws of the plate indicating loosening of the plate screws. A telephone report from the owner at 20 months postoperatively indicated that the dog was back to normal except for minor pelvic limb in-coordination.

Discussion

Dogs suffering thoracolumbar disc extrusion may be managed medically or conservatively, but it is generally accepted that non-ambulatory dogs should be managed surgically (2). Pre-operative diagnostics are intended to confirm the diagnosis of IVD disease, localise the lesion and exclude other differential diagnoses. Clinical signs allow gross lesion localisation, but may not accurately reflect the location of extruded disc material (13). Myelograms...
Myelography localises the lesion more reliably than clinical signs (13), but may fail to correctly localise the lesion in two to 20% of cases (14–16); using oblique myelographic views reportedly decreases the failure rate to one percent (17). In the present case, myelography did not differentiate the disc protrusion at T12–13 from the disc extrusion at T13-L1, nor did it localise it. This resulted in more surgical approaches to the dog’s spine than was necessary because the most likely lesion location was explored first followed by less likely locations in a logical order until the lesion was identified and resolved, as is recommended (2). Computed tomography and magnetic resonance imaging are superior to myelography for lesion localisation and identification (2, 18). Their use in this case would likely have avoided the unnecessary spinal explorations and the ensuing complication, but they were not performed as they were not available at the time the dog was presented as a neurological emergency.

Mini-hemilaminectomy and pediculectomy are techniques that allow minimal approaches to the thoracolumbar spine but preserve the facet joint (2, 5, 7, 8). Pediculectomy is the removal of the pedicular bone inbetween the intervertebral foramen, but it does not give direct access to the IVD. A mini-hemilaminectomy on the other hand, where the pedicular bone cranial and caudal to the foramen is removed, does allow direct access to the IVD (2, 8). Both techniques, when compared to hemilaminectomy, have been suggested to be less invasive and increase the speed of recovery, as well as decrease surgical time, trauma, possibility of iatrogenic damage, laminectomy membrane formation and destabilisation of the vertebral column (5, 7, 8, 19). As the first myelogram in the present case failed to localise the lesion, the potential need to perform more than one surgical approach to the spine was recognised. Therefore mini-hemilaminectomy with pediculectomy was chosen as this combination has been suggested to maximise spinal visualisation and exploration whilst minimising surgically induced trauma and instability (2, 7, 8).

The reduction in height of the spinal canal of about 40% seen in this case was caused by the ventral subluxation of the cranial, but not the caudal aspect of the dorsal lamina of T13. The author suggests that in order for this to happen, mechanical decoupling (anatomical separation) of the dorsal lamina from the vertebral body of T13 must occur with secondary mechanical failure (subluxation) of the T12–13 facet joints. A bilateral hemilaminectomy extended caudally to include pediculectomy of the vertebral body would anatomically separate the dorsal lamina from the vertebral body if the entire pedicle were removed. In the present case, the entire pedicle of T13 was removed on the left side, and only a small caudal portion remained on the right side, which could have bent or fractured. Either way, the T13 dorsal lamina had minimal structural connection to the vertebral body and was predisposed to becoming un-
stable. The T12–13 articular facet joints probably failed secondarily by stretching or rupture of the joint capsule. Spinal extension would create a compressive force at the T12–13 articular facet and the anatomic conformation of the facets would convert this into a ventral vector component acting on the cranial aspect of the T13 lamina. Given the proposed surgically induced structural deficiencies, this could result in ventral displacement of the T13 lamina.

The literature does not warn of the possibility of such a complication following combined mini-hemilaminectomy and pediculectomy surgery. Conversely, the literature suggests that these procedures have a minimally destabilising effect on the spine (2, 7, 8, 19) and thus their combined use is advocated (2, 7, 8). Pediculectomy has the least destabilising effect on the lumbar spine compared to hemilaminectomy and fenestration (19) and it has been suggested that bilateral mini-hemilaminectomy does not compromise stability of the spine (5), but this has not been proven. Experimental hemilaminectomy has no effect on spinal stability (20), and fenestration and facetectomy (as used in routine laminectomies) have not been shown to cause significant destabilisation (21). Furthermore, hemilaminectomy can be extended bilaterally, and up to two adjacent sites without increasing the risk of a flexion-extension related injury (22).

Given the anatomically destabilising effect (as suggested by the author) of the surgery that was performed in this case, it is perhaps not surprising that this complication occurred. It is interesting however to reflect on the lack of literature documenting similar occurrences of instability following decompressive spinal surgery.

It is possible that the IVD fenestration that was performed at T12–13 (and T13-L1) also contributed to the spinal instability; studies investigating the destabilising effects of spinal surgery have shown that the intervertebral disc is the most important structure for maintaining spinal stability (23) and that fenestration is the single most important destabilising factor (19). However, clinically significant instability resulting from fenestration has never been reported. It seems unlikely that fenestration contributed to instability in this case as gross instability between T13 and the adjacent vertebrae was not identified; conversely structural instability of T13 (lamina vs. vertebral body) plus facet joint subluxation was identified. Another possible explanation of neurological deterioration in this case is that the blood clot found during the second surgery might have been compressing the spinal cord. However, this seems unlikely as the blood clot was not particularly large and nothing was observed when the clot was removed that would suggest it was causing the cord compression.

Many methods of managing spinal instability are available including conservative management, external skeletal fixation, polymethylmethacrylate and pins or screws, and vertebral body plating or stapling (2, 24). In the current case, the unstable articular facet joints were directly immobilised using positional screws as the primary method of stabilisation. A dorsal plate was placed to protect these positional screws against mechanical loading in the early postoperative period, but a veterinary cuttable plate was used rather than a Lubra plate® because the latter was not immediately available. This technique enabled an uncomplicated recovery in this patient.

Conclusions

Bilateral mini-hemilaminectomy extended by pediculectomies can result in spinal insta-
bility and neurological deterioration. Caution should be exercised when combining bi-
ilateral mini-hemilaminectomy with pedi-
culectomy; extensive surgery may result in se-
paration of the dorsal lamina from the ver-
tebrae body and this could result in laminar
instability and secondary cord compression.
Should pathological spinal instability occur,
successful surgical stabilisation and patient
recovery are possible.

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