Case report  

A 6-year-old male neutered crossbreed dog presented with acute onset paraparesis and was diagnosed with an L1–L2 intervertebral disc extrusion. A right-sided T13–L2 hemilaminectomy was performed. However, the dog deteriorated and became paraplegic with marked thoracolumbar hyperaesthesia 48 h after surgery. A computed tomography scan of the thoracolumbar vertebral column revealed the presence of pneumorrhachis (PR) at the level of T13, possibly embedded in a haematoma, and causing marked spinal cord compression. Revision surgery confirmed the presence of a haematoma, which was removed. The dog gradually improved and was neurologically normal 6 weeks after surgery.

Conclusion  

Although PR is a rare condition, it may be considered a possible cause for early postoperative neurological deterioration in dogs undergoing decompressive spinal surgery. Surgical revision resulted in a good outcome in the presented case.

Keywords  

hemilaminectomy; intervertebral disc extrusion; pneumorrhachis; postoperative complications; vertebral canal

Abbreviations  

CT, computed tomography; MRI, magnetic resonance imaging; PR, pneumorrhachis; TR, repetition time

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Postoperative symptomatic haematoma and pneumorrhachis in a dog with a thoracolumbar intervertebral disc extrusion

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Pneumorrhachis (PR) is the presence of extradural or intradural gas in the vertebral canal.1–4 It has been reported three times in veterinary literature,2,4,5 but has been reported more commonly in human medicine where it is mostly considered an incidental finding. Several underlying aetiologies have been suggested, with most cases occurring secondary to trauma or degenerative disc disease. Other causes include iatrogenic, infectious, spontaneous or toxic conditions and decompression sickness.1–3 Iatrogenic PR is reported secondary to epidural analgesia, thoracostomy tube placement, spinal surgery and radiation therapy.2,6 Spontaneous PR denotes the occurrence of non-traumatic, non-iatrogenic gas within the vertebral canal. This has been described in association with the vacuum phenomenon, which is characterised by gas accumulation in a degenerated intervertebral disc.2,6 In human medicine, computed tomography (CT) is considered the imaging modality of choice to diagnose PR, but magnetic resonance imaging (MRI) allows evaluation of potential coexisting aetiologies.2 Because PR is a rare and mostly incidental finding, no empirical treatment guidelines exist.1

In veterinary medicine, a case of spontaneous thoracic PR and a case of PR in conjunction with tension pneumocephalus after a transfrontal craniotomy or rhinotomy have been described.2,6 To the best of our knowledge, this is the first report describing the development of postoperative PR after a hemilaminectomy for a thoracolumbar intervertebral disc extrusion in a dog.

Case report

A 6-year-old, 13-kg, male neutered crossbreed dog was referred for further evaluation of acute onset, progressive ambulatory paraparesis that occurred 12 h before presentation. General physical examination was within normal limits.

Neurological examination identified marked paresis and ataxia of the pelvic limbs, absent proprioceptive placement and hopping in both pelvic limbs, increased patellar reflexes and marked thoracolumbar hyperaesthesia on spinal palpation. The remainder of the neurological examination was within normal limits. The findings were consistent with a lesion affecting the T3–L3 spinal cord segments. A complete blood count and serum biochemistry profile were within normal ranges.

The dog was premedicated with 0.2 mg/kg methadone (Physeptone, Martindale Pharma) administered intravenously. Anaesthesia was induced with 3 mg/kg of intravenous propofol (PropoFlo Plus, Abbott Laboratories), immediately followed by 0.2 mg/kg diazepam (Diazepam Injections, Hameln Pharmaceuticals Ltd), and maintained with sevoflurane (SevoFlo, Abbott Laboratories) vaporised in oxygen.

MRI (Intera 1.5 T, Philips Medical Systems) of the thoracolumbar vertebral column was performed with the dog in dorsal recumbency. The imaging protocol included sagittal and transverse plane T2-weighted (repetition time (TR)/echo time (TE), 3000/120), T1-weighted (TR/TE, 400/8) and transverse plane T2-weighted BAL TGRAD (TR/TE, 7.9/3.9) sequences. The presence of a right-sided extradural spinal cord compression mainly at the level of L1–L2, extending cranially over the vertebral body of T13, was demonstrated.

A presumptive diagnosis of a right-sided L1–L2 intervertebral disc extrusion was made and a routine right-sided T13–L2 hemilaminectomy6 was performed. Intraoperatively, analgesia was provided by a constant-rate infusion of fentanyl (Fentadon, Eurovet Animal

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Health) at 0.1–0.3 μg/kg/min and ketamine (Anesketin, Dechra) at 10 μg/kg/min. Prophylactic antibiotic therapy was given every 2 h throughout the surgery with 20 mg/kg of cefuroxime (Cefuroxime, Stavencon Ltd). An absorbable bovine collagen fleece (Lyostypt, B. Braun Surgical S.A.) was placed over the surgical defect and the wound was closed using a routine 3-layer closure.

Surgery and recovery from anaesthesia were uneventful. One day after surgery, the dog demonstrated slight neurological deterioration, being non-ambulatory paraparetic, but became paraplegic with intact nociception and marked thoracolumbar hyperaesthesia 48 h after surgery. The dog was anaesthetised using the same protocol as for the surgery. CT of the thoracolumbar vertebral column with a 16-slice scanner (PQ 500, Universal Systems) was performed (helical mode, 2-mm slice thickness, 1 mm overlap between slices, 140 kVp, 120 mA, 110-mm acquisition field of view, bone and soft tissue reconstruction algorithms, 512 × 512 matrix). This confirmed the presence of a right-sided T13–L2 hemilaminectomy defect and complete removal of the extruded disc material.

There were multiple, rounded, well-demarcated, spherical, hypoattenuating structures consistent with gas bubbles (Hounsfield units = −1020) present in the soft tissues overlying the surgical defect. From the caudal level of T13 to mid-L1, there was poorly demarcated hyperattenuating tissue extending from the surrounding tissues entering the vertebral canal via the surgical defect, resulting in mild spinal cord compression. In the vertebral canal, at the caudal level of T13, there was a large, spherical, hypoattenuating lesion consistent with a gas bubble on the left side (Figure 1A). This structure had a maximum diameter of 4.8 mm, filling up approximately 50% of the vertebral canal, and was associated with marked spinal cord compression. Similar structures were present within the vertebral canal at the cranial level of L1 (2.1 mm diameter) and the cranial level of L2 (3 mm diameter), both on the right side and causing mild spinal cord compression (Figure 1B).

Based on these imaging findings, diagnosis of a postoperative mildly compressive haematoma and extradural markedly compressive PR was made. Revision surgery was performed, confirming the presence of the haematoma, which was removed. The gas bubble could not be visualised. Identical intraoperative analgesia was administered as during the first surgery. Recovery from anaesthesia was uneventful. Postoperative analgesia consisted of 0.1 mg/kg of methadone (Physeptone, Martindale Pharma, IV q4h), gabapentin (Gabapentin Zentiva, Zentiva; 10 mg/kg PO q8h) and paracetamol (Pardale-V, Dechra 10 mg/kg IV q6h).

The dog improved gradually and was discharged 3 days after surgery. He was ambulatory 5 days after the revision surgery and neurologically normal 6 weeks after surgery. Neurological examination 5 months after surgery did not reveal any abnormalities.

**Discussion**

This case report describes the occurrence and successful management of PR after spinal surgery as the cause of early postoperative neurological deterioration. Although postoperative or iatrogenic PR has been reported in human medicine, it is considered very rare.7–9

![Figure 1. Postoperative transverse (A, B) and sagittal reconstruction (C) CT images of the thoracolumbar vertebral column at the level of T13 (A) and L2 (B) following decompressive spinal surgery in a dog. There are multiple, well-demarcated, spherical, hypoattenuating structures representing gas bubbles in the vertebral canal and the soft tissues surrounding the right-sided hemilaminectomy defect. CT, computed tomography.](image_url)
Although the exact pathophysiology of postoperative PR is still unknown, three different hypotheses have been suggested. First, air that enters the surgical site during surgery can get trapped when blood binds the soft tissue margins of the wound, forming a membrane that encases the gas collection. Second, spontaneous and post-operative PR can be associated with the ‘vacuum phenomenon’, which is produced by the liberation of gas from the extracellular fluid that accumulates within the cracks, clefts or crevices that form in degenerated intervertebral discs. Third, the ‘inverted bottle’ mechanism, whereby an arachnoid tear created by the primary aetiology (intervertebral disc extrusion) causes cerebrospinal fluid leakage, resulting in a negative pressure gradient and allowing gas to flow into the subarachnoid space.

In the present case, given that neither the MRI nor CT study demonstrated the vacuum phenomenon in the affected intervertebral disc, it is unlikely that the vacuum phenomenon was associated with PR. Neither dural tear nor cerebrospinal fluid leakage was observed during the initial spinal surgery and the observed gas bubble was extradural instead of intradural. Therefore, it is also unlikely the inverted bottle mechanism was involved in the development of PR in the dog presented here. There was, however, a haematoma present on postoperative CT imaging, which possibly sealed the vertebral canal and entrapped gas in the surgical site. We assume the clinical signs in the presented case are reflecting a combination of the formation of a haematoma and entrapment of a large gas bubble.

Conservative management consisting of bed rest, hyperhydration and inhalation of 100% oxygen are adequate for the majority of human patients with postoperative PR. In human medicine, conservative management is usually attempted first and surgical treatment is generally reserved for patients not responding adequately to medical management. Surgical intervention typically consists of needle aspiration or open surgical removal of the intraspinal air. Surgery was elected in the case presented here for two reasons. First, the dog demonstrated severe neurological signs and rapid deterioration of his condition. It was therefore decided to pursue direct decompression of the spinal cord. Second, although the PR occupied approximately half of the transverse vertebral canal area, it was likely that the haematoma itself was also causing some of the spinal cord compression and therefore contributing to the dog’s clinical signs.

Although uncommon, several mechanisms of early neurological deterioration after decompressive surgery for thoracolumbar intervertebral disc disease in dogs have been described. These include progressive myelomalacia, inadequate spinal cord decompression, early reherniation at the previous hemilaminectomy site, a second disc extrusion, a fat graft reaction and vertebral instability. Although PR is a rare condition, it can be considered a possible cause for early postoperative neurological deterioration in dogs undergoing decompressive spinal surgery. Surgical revision in this case resulted in a good outcome.

References


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