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CASE REPORT

Companion or pet animals

Dysphonia in a dog with cervical spinal cord injury and suspected progressive myelomalacia caused by a C4-C5 hydrated nucleus pulposus extrusion

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Abstract

A 10.5-year-old dog was presented with acute onset tetraparesis progressing to tetraplegia. The clinical sign of dysphonia was documented in the absence of signs of laryngeal dysfunction. MRI findings of the cervical spinal cord were consistent with a C4-C5 hydrated nucleus pulposus extrusion (HNPE) and overlying spinal cord injury (SCI). Seven days after presentation, a repeat MRI study was performed due to clinical deterioration with respiratory compromise and revealed changes compatible with progressive myelomalacia. The dog was euthanased due to the progression of severe clinical signs. Dysphonia due to acute cervical SCI has not been reported in dogs. Explanations for the clinical sign of dysphonia are discussed, relevant aspects of the literature on HNPE are reviewed and discussion points are mentioned with regard to SCI and myelomalacia.

BACKGROUND

In humans, cervical spinal cord injury (SCI) of various aetiologies is common and literature on this subject is vast.[1–8] Respiratory complications due to the cervical SCI itself or surgical procedures are well documented in human literature.[1,4,8] Speech impairment, dysphonia and dysphagia are reported as clinical signs in humans with cervical SCI, both unrelated and related to surgery.[4,8] Respiratory complications are relatively infrequently reported in canine spinal cord surgery, albeit more so when the cervical spinal cord is involved.[9–13] Recently, bilateral laryngeal paralysis following a ventral slot surgery in a dog was reported.[14] To date, dysphonia has not been reported as a clinical sign in dogs with cervical SCI.

CASE PRESENTATION

A 10.5-year-old male miniature pinscher was presented during emergency hours with signs of acute onset tetraparesis progressing to tetraplegia. Just 4 hours before the presentation, the dog was seen walking normally indoors. General physical examination did not reveal any abnormalities. Briefly, regarding the examination of the respiratory system, a respiratory rate of 20 breaths per minute and a costo-abdominal pattern were noted. Although tetraplegia was evident, a comprehensive neurological examination was not performed at that point. This was partly due to attempts of the dog to bite the examiner when it was handled. Consequently, due to concerns of pain, analgesics were administered (0.23 mg/kg methadone and 0.2 mg/kg meloxicam) and intravenous fluid therapy (balanced crystalloid solution) was started. A comprehensive neurological examination was scheduled to be performed by the neurology service 5 hours later. The dog was presented in lateral recumbency and was alert and responsive; it responded to visual and auditory cues by lifting its head and looking around. The dog was tetraplegic (no voluntary motor activity of the limbs). The thoracic limbs were extended with increased muscle tone. The pelvic limbs and tail were subjectively hypotonic. Spontaneous urination had been witnessed by hospital staff. Bilateral patellar hyporeflexia, intact withdrawal reflexes of pelvic and thoracic limbs and intact cutaneous trunci and perineal reflexes were noted. Cranial nerve examination did not reveal any abnormalities. No signs of cervical hyperesthesia were noticed upon palpation. When testing withdrawal reflexes, the dog turned its head and attempted biting. Therefore, nociception was evaluated as intact. Noticeably, the dog also attempted to bark, but only a barely audible, hoarse sound was produced (Video 1). This was interpreted as dysphonia. The owner was consulted to ensure that the dog was able to bark before the onset of the neurological signs; this was confirmed.

The neuroanatomical localisation was based on the following considerations:

1. Tetraplegia, with increased muscle tone in the thoracic limbs and decreased muscle tone in the pelvic limbs, intact spinal reflexes, but patellar hyporeflexia.

2. Localisation: C1-C5 myelopathy with spinal shock to account for the pelvic limb lower motor neuron signs of hypotonia and patellar hyporeflexia.
3. Dysphonia
   Localisation of a or b or both:
   a. Vocal cord dysfunction: general somatic efferent (GSE) neurons of the accessory nerve (XI) and vagal nerve (X) in the nucleus ambiguus, (cranial) nerve roots of X and XI, vagal nerves, recurrent laryngeal nerves, laryngeal muscles (intrinsic), vocal cords.
   b. Respiratory dysfunction: any disorder that impairs the ability to produce sufficient force of expiration to produce sound, which includes central and peripheral parts of the nervous system: upper motor neuron control of respiration (ventral) respiratory groups in the medulla oblongata (and pons), reticulospinal (bulbospinal) tracts, GSE neurons of the phrenic nerves in the ventral horn of (C4 and) C5-C7 spinal cord segments, (C4 and) C5-C7 nerve roots and spinal nerves, phrenic nerves, neuromuscular junction, diaphragm musculature and other (accessory) respiratory muscles (e.g., intercostal muscles) and their innervation through GSE neurons dispersed in the caudal cervical and thoracic spinal cord segments.

Given the preponderance of neurological signs, cervical myelopathy involving the C1-C5 spinal cord segments was suspected, with involvement of LMN compromising the phrenic nerves and resulting in a degree of diaphragmatic paresis severe enough to inhibit adequate phonation, but not severe enough to impair respiratory function at this point. This hypothesis was formulated in hindsight after consideration of all neuroanatomical structures involved in phonation and those (likely to be) affected in this particular case.

LEARNING POINTS/TAKE HOME MESSAGES

- Dysphonia can be a clinical sign in patients with cervical spinal cord injury (SCI).
- Cervical SCI can result in respiratory dysfunction and patients suffering from this injury merit careful monitoring.
- Repeated diagnostic imaging studies may reveal signs suggestive of ongoing/worsening of myelopathy and provide information on which to base decisions of further management of patients
- The indication for surgery in patients with severe cervical SCI due to hydrated nucleus pulposus extrusion is unclear

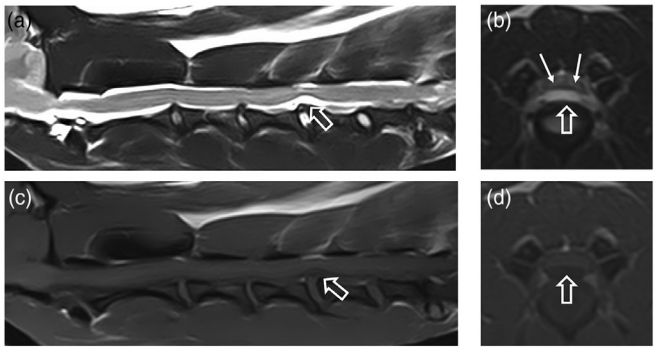

FIGURE 1 Magnetic resonance images acquired approximately 10 hours after signs were noticed by the owner. (a, c) Mid-sagittal T2W and T1W, respectively, (b, d) transverse T2W and T1W, respectively, at the level of C4-C5 intervertebral disc space. The hollow arrows point to the extradural lesion at the level of C4-C5, which is hyperintense in T2W and hypointense in T1W. From the transverse images (b, d) the lesion is seen located on the ventral aspect and affecting both sides, with a typical "seagull appearance", and it causes moderate spinal cord compression. The thin arrow points out the focal overlying intramedullary lesion

INVESTIGATIONS

Haematological and biochemical tests were unremarkable. An MRI study was performed within 1 hour of the onset of neurological examination. The dog was premedicated with 0.23 mg/kg butorphanol and 6 μg/kg dexmedetomidine. The larynx was visualised noting adequate abduction of the vocal cords on inspiration (i.e., no signs of laryngeal paralysis) and sprayed with 10% xylocaine. An intravenous bolus of 2 mg/kg propofol and repeated boluses of 1 mg/kg were administered to achieve an adequate plane of anaesthesia before intubation and transferral to the MRI unit. The dog was placed in dorsal recumbency for the MRI examination and mechanical ventilation was initiated with 2% isoflurane to maintain an adequate plane of anaesthesia. Expiratory CO_2 was kept between 35 and 50 mmHg. After the MRI study, the dog was weaned of mechanical ventilator support, maintaining CO_2 mmHg of about 50–60 after initial higher values required to trigger independent ventilation. The dog had a SpO_2 of 98% (pulsoxymetry) on room air with voluntary respiratory efforts when extubated, and respiratory pattern did not change, raising no concerns of respiration at that time.

The MRI study (1.5T Vantage Elan; Canon Medical Systems, The Netherlands) of the cervical region was performed with a Flex Speeder medium-sized coil. Sequences included were: dorsal STIR, sagittal T1-weighted (T1W), sagittal T2-weighted (T2W), transverse T1W and transverse T2W sequences. The images revealed a well-defined extradural lesion at C4-C5, located ventrally and affecting both sides and showing a 'seagull-shape' (Figure 1). This lesion was homogeneous, hyperintense in T2W and hypointense in T1W when compared to the spinal cord. It caused marked spinal cord compression, with an associated focal T2W hyperintensity within the cord, centred on the grey matter. Measurements on the midsagittal T2W image revealed a ratio between intramedullary hyperintensity and length of C3 vertebral body of 0.57.[15] Additionally, signs consistent with degeneration of multiple intervertebral discs (IVDs), mild protrusions and endplate changes of C6-C7 vertebrae were found. The imaging findings were consistent with a diagnosis of a C4-C5 acute compressive hydrated nucleus pulposus extrusion (HNPE) with intramedullary changes suggestive of oedema or myelomalacia (gliosis, myelitis, haemorrhage and other causes of intramedullary T2W hyperintensity deemed less likely based on the history and findings).

DIFFERENTIAL DIAGNOSIS

Before investigations, differential categories considered included traumatic SCI (internal, e.g., HNPE or acute non-compressive nucleus pulposus extrusion with or without haemorrhage), vascular incidents (ischemic myelopathy and haemorrhagic myelopathy), neoplasia (primary or metastatic) and degenerative disease (IVD disease [IVDD], e.g., IVD extrusion). The latter may also be categorised as internal SCI with underlying IVD degeneration. External causes of SCI were excluded based on the history. Inflammatory, metabolic and other aetiologies were deemed unlikely, mostly based on the history, including the (hyper)acute nature of the clinical signs.

TREATMENT

The dog was hospitalised to monitor for deterioration over 36 hours, in which there was no apparent change in neurological status. In addition to fluid therapy, the dog was treated with 0.1 mg/kg meloxicam, change of position every 2–4 hours with a preference for sternal recumbency with padded support, physiotherapy exercises (e.g., range of motion exercises, assisted standing as far as possible and spinal reflex stimulation) and bladder management (e.g., prevention of contact of skin with voluntarily voided urine in the cage and mild-moderate manual abdominal pressure to assist active voiding outside). After discussion with the owner, the dog was discharged and taken home for further careful monitoring (especially with regard to breathing), instructions for physiotherapy and continued 0.1 mg/kg meloxicam oral.

OUTCOME AND FOLLOW-UP

The owner mentioned the dog being alert and active the next day, trying and failing to bark (dysphonia) when the doorbell rang and remaining able to lift its head, but being tetraplegic still. Three days later (5 days after presentation) the dog was readmitted for concerns of signs of pain (attempts at biting the owner). The neurological examination revealed dysphonia/aphonia, a shallow, weak breathing pattern with a rate of 40 breaths per min and hypotonic thoracic limbs. The dog was still able to lift its head and nociception remained intact. A SpO_2 of 90% (pulsoxymetry) on room air further raised concerns of inadequate respiration. The dog was hospitalised and kept in an oxygen chamber (SpO_2 95% at about 80% oxygen). A repeat MRI scan was performed 7 days after the original presentation. This included the same sequences as the first MRI study, plus post-contrast T1W sequences in transverse and sagittal planes (Figure 2). The MRI revealed that the extradural lesion at C4-C5 previously seen was no longer present. However, the intramedullary lesion was increased in size and severity, centred at the level of C4-C5 and extending cranially to the level of C2-C3 and caudally to C7-T1. This intramedullary lesion affected predominantly the grey matter (although the surrounding white matter was also involved to a lesser extent). This intramedullary lesion was homogeneous, T2W hyperintense and T1W isointense. On T1W images acquired after administration of gadolinium (0.15 mmol/kg), it showed moderate enhancement affecting grey matter at the level of C4-C5 and mild meningeal enhancement. The imaging findings were considered most likely consistent with ascending/descending myelomalacia and/or infarction. The presence of gliosis or other causes of the mentioned signal changes could not be excluded but were deemed unlikely based on the clinical findings and earlier imaging diagnosis. Due to the progressive nature of signs and findings suggestive of progressive myelomalacia as the cause, the owner decided for humane euthanasia. Postmortem examination was not allowed.

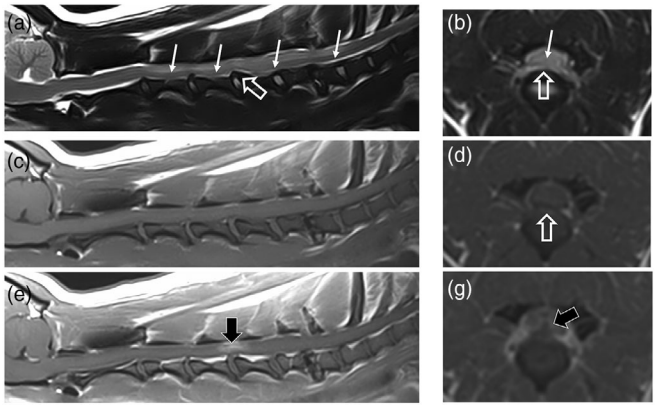

FIGURE 2 Magnetic resonance images acquired approximately 7 days after the MRI study shown in Figure 1. (a, c and d) Mid-sagittal T2W, T1W and T1W post-contrast, respectively. (b, d and f) Transverse T2W, T1W and T1W post-contrast, respectively. The hollow arrows point out the lack of compressive extradural lesion previously seen at C4-C5. The thin white arrows point out the intramedullary lesion, which is more extensive than in the previous MRI study. The black arrows point out the focal intramedullary contrast enhancement

DISCUSSION

Dysphonia is described as a clinical sign in a textbook case of a dog with postmortem confirmed ascending/descending myelomalacia with respiratory compromise resulting from thoracolumbar SCI due to an IVD extrusion.[15] Dysphonia as a clinical sign is not reported in veterinary literature on dogs with primary cervical SCI. The clinical characteristics, diagnostic findings, treatment options, microsurgical findings and outcomes of canine cases with HNPE have been described.[16–22] Respiratory compromises in dogs with HNPE were reported.[16,18,22] A predilection for the cervical region was found in several studies focussing on acute compressive HNPE, with the C4-5 IVD site most often involved.[16–22] In one report, 5/36 dogs displayed non-ambulatory tetraparesis complicated by mild dyspnoea and 5/36 dogs showed tetraplegia that was complicated by severe dyspnoea in two of those five dogs.[18] In that report, it was mentioned that normal respiratory function was regained following anaesthesia in dogs with respiratory impairment. Those dogs were scanned and operated on during the same period of anaesthesia. In those cases with respiratory compromises, some degree of dysphonia is likely to be present and is presumably under-recognised and under-reported. It was a conspicuous finding in the case reported here which led to the authors specifically paying attention to vocal cord abduction while the dog was

premedicated with sedatives previously reported to be suitable for the evaluation of vocal cord movements by laryngoscopy in dogs.[23] That, and the lack of inspiratory stridor, which would be expected in a case of laryngeal paresis/paralysis, led us to conclude with confidence that vocal cord dysfunction was not a plausible cause of dysphonia in this dog. It was thus concluded that inadequate velocity of expiratory airflow to induce the production of sound (phonation) at the level of the vocal cords was the cause of dysphonia in this case. As the C4-C5 IVD underlies the C5-C6 spinal cord segments and the canine phrenic nerves have their GSE neuronal cell bodies in the (sometimes C4 and) C5-C7 spinal cord segments,[24,25] the most likely explanation for the insufficient force of expiration is diaphragmatic paresis. Although in this case, it did not lead to clinically recognised respiratory pattern nor rate abnormalities, mild-moderate diaphragmatic paresis is likely to have been present. Blood gas analysis was not performed and may have shown abnormalities not recognised clinically.

In dogs, other respiratory muscles may compensate in case of diaphragmatic paresis/paralysis.[26] As human patients are able to report symptoms such as the relative difficulty of breathing and having to consciously pay attention to breathing and veterinary patients are not, diaphragmatic paresis in canine SCI is likely under-recognised. Although we cannot draw strong conclusions from only this single case, the clinical sign of dysphonia may function as a signal to the clinician to employ more rigorous testing and support of respiratory function in cases of cervical SCI. Dysphonia is not specifically included in any of the employed grading systems of cervical SCI due to HNPE, with the worst grade often being variably defined as, for instance, 'tetraplegia with a respiratory compromise', 'tetraplegia with absent nociception' or 'tetraplegia with neurogenic hypoventilation or sensory impairment'.[16–22] Dysphonia may be interpreted as a sign of respiratory compromise and may be a reflection of injury severity or simply the localisation of the injury.

Myelomalacia is defined as gross softening of the spinal cord characterised by haemorrhagic necrosis and liquefaction of spinal cord tissue,[27] carries a very grave prognosis for recovery of function and may result in death.[27–29] It is the result of primary and secondary injury to the structures of the spinal cord.[30] Secondary injury is still a subject of research but seems to be the result of a combination of several factors including excitotoxicity, ischemia and ischemic-reperfusion injury.[30] This process may be contained at the site of the primary injury or may spread cranially/caudally for which the reasons are not quite clear. Clinical deterioration usually ensues quickly when this process is started, but a delay in clinically noticeable signs of progressive myelomalacia has been reported in dogs, usually pertaining to a variable number of days (up to 5 days) after acute SCI.[27–31] Myelomalacia (focal, ascending/descending or progressive) has been mostly reported in dogs with acute IVDD and several risk factors have been reported, including the presenting neurological grade (i.e., the more severe the deficits, the more likely is the development of myelomalacia) and localisation (lumbar intumescence involvement).[27–29]

In this case, clinical deterioration was not seen in the 36 hours following neurological assessment. In 7 reports focussing on HNPE in dogs, presumptive myelomalacia was a reported sequela in five dogs (including one specifically suspected based on MRI findings, one with consistent surgical findings and three that died or were euthanased because of cardiorespiratory issues or worsening of signs but lacking any other form of diagnostic findings specifically supporting a diagnosis of myelomalacia) of a combined total of 154 cases, equal to 1.3%–3.2% of cases.[16–22]

It must be reiterated that progressive myelomalacia was solely presumptively diagnosed in the case reported here, based on clinical deterioration, MRI findings and lack of plausible other causes for both of these. Since changes were centred on grey matter, (polio)myelomalacia could be suspected.[32] Other causes for signal change such as those encountered on MRI (i.e., haemorrhage, oedema, myelitis and gliosis) were deemed unlikely, due to the nature of the signal changes, the history, the findings on the earlier MRI and the clinical development. Haemorrhage could have been excluded with more confidence if gradient-echo T2* sequences were acquired. In dogs with thoracolumbar IVD extrusion, MRI findings have a prognostic value related to the incidence of myelomalacia.[27–30,33] The ratio between intramedullary hyperintensity and length of C3 vertebral body is reported to be associated with outcome in dogs with HNPE[21]; a possible association to the occurrence of progressive myelomalacia is yet to be confirmed. Intramedullary contrast enhancement was evident on the second MRI in this case. A contrast medium was not administered during the first MRI. Thus, we do not know if contrast enhancement may have been present early on as well. MRI findings of (polio)myelomalacia are not specific. Visual inspection (and preferably palpation to evaluate 'softening') of the spinal cord is required to definitively diagnose myelomalacia, with transverse sectioning (postmortem) and histopathological examination providing further details, such as which specific spinal cord structures are involved. Surgical confirmation of myelomalacia by visual inspection of the spinal cord after durotomy and palpation with a probe may be considered sufficient to definitively diagnose myelomalacia.[34,35] Although irreversible, myelomalacia may remain focal from the start or halt after initial ascending/descending progression for largely unknown reasons, though measures to lower intraspinal pressure seem promising.

Since extensive discussion is out of the scope of this case report, the authors would instead like to point out some specifically interesting questions and considerations with (possible) relevance to veterinary patients with cervical SCI caused by HNPE and suspected myelomalacia:

- The basics of treatment of SCI remain largely unchanged, ensuring that[2,3,7,30]:
  1. A patent airway is present,
  2. Adequate respiration and blood oxygenation are maintained,
  3. Tissue perfusion (i.e., blood pressure and local pressure) is optimized and
  4. Further SCI is prevented (i.e., immobilisation in case of unstable vertebral fractures).
- The effect of extensive decompressive surgery and durotomy in dogs with severe SCI or myelomalacia is a subject of interest in recent literature.[34–37]
- Dural surgery as a measure to lower intrathecal pressure and monitoring thereof are two of the key points of

interest in the latest human neurosurgery reviews on the current state of the art in SCI.[1–3,5–7,30] Animal studies form the backbone of these developments.

- There is no clear evidence to support the indication for surgery in dogs with HNPE with severe SCI, but it is good to be mindful of some facts[16–22]:
  1. In a study where all dogs were treated surgically reported that several dogs with respiratory compromise had normal respiratory function immediately after procedures,
  2. HNPE compressive lesions may resolve without surgical intervention, even within a short span of days and
  3. The number of patients with severe cervical SCI due to HNPE in studies comparing surgical versus conservative management is low.
- Anecdotally, detrimental outcomes of conservative therapy of dogs with severe cervical SCI due to HNPE have been seen by experienced neurologists/neurosurgeons, while surgical intervention typically results in good outcomes in such cases.[38] Some may say that it could be considered prudent to perform emergency decompressive surgery in these patients as long as we assume those procedures to have no detrimental effects on the recovery of these patients, even if it is unclear, as of yet if these procedures provide an overriding benefit to the patient.

In the case reported here, surgery was put forward as an option when clinical deterioration was noticed. However, progressive myelomalacia was (suspected to be) responsible for the clinical deterioration based on MRI findings. Thus, repeat imaging should be performed before the decision is made to surgically treat these patients to be sure that the planned surgery would be of any possibly expected benefit (i.e., if there is still a compressive lesion) and assess if complicating factors are present (i.e., intramedullary changes). In one study, two dogs with HNPE started on a conservative treatment were treated surgically due to a perceived unsatisfactory response to medical management.[17] There is no information on the nature of dissatisfaction nor on the surgical findings (i.e., if there was any material still to be removed upon performing the ventral slot technique). Both dogs nevertheless experienced complete neurological recovery, because of or despite this surgery; this is unknown as mentioned in the discussion of that article.

To be clear, the authors would like to state that some of the decisions made in the management of this case are subject to scrutiny. Although no deterioration was seen over the course of 36 hours and clinically recognisable respiratory compromise (other than dysphonia) was not evident or was not recognised at that time, the lack of specific diagnostic tests (e.g., blood gas analysis to assess the presence of subclinical hypoventillation[13]) and discharge at that point prevented us from recognising the eventual deterioration sooner. We, therefore, conclude that we should advise clients more strongly to the extent of the period of monitoring and keep such patients admitted for a longer period in the future. For instance, until improvement is seen, with or without surgery. We do not know if this would have changed the outcome in this case or not, but it is likely to be of benefit to the patient. Financial considerations and wishes of the owner are factors to be taken into account.

This case illustrates the importance of repeated clinical examination, repeat diagnostic imaging studies and careful assessment of the need for monitoring of neurological patients. We report the clinical sign of dysphonia in a dog with cervical SCI. The relevance of the clinical sign of dysphonia can be evaluated by taking this sign into the account, in future cases of cervical SCI. The benefit of surgical or medical treatment for dogs with severe cervical SCI due to HNPE is unclear. The possibility of the benefit of surgical treatment cannot yet be dismissed.

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CONFLICT OF INTEREST

The authors declare they have no conflicts of interest.

ETHICS STATEMENT

Ethical approval was not required for the article.

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SUPPORTING INFORMATION

Additional supporting information may be found in the online version of the article at the publisher's website.

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