

Dura fibrosis and adhesions caused by an atlas malformation in two dogs

DITTE SKYTTE¹, HUGO SCHMÖKEL¹, YVONNE LANG² OG ULRIK WESTRUP¹

¹INSTITUT FOR MINDRE HUSDYRS SYGDOMME, KØBENHAVNS UNIVERSITET, DANMARK

²KLEINTIERKLINIK YVONNE LANG, ALZENAU-HÖRSTEIN, TYSKLAND

Introduction

Adhesion formation and fibrotic scarring is a rare finding in chronic spinal cord compression in the dog. To the knowledge of the authors there is no report of clinical cases in the literature. Spinal decompressive surgery with durotomies in dogs have been used in experimental settings to investigate different anti-adhesion strategies for human surgical treatment of spinal conditions, where post-operative adhesion between the dura and the extra-epidural soft tissues is a major problem (1,2,3). Extensive formation of scar tissue can follow after a laminectomy/hemilaminectomy in dogs, but it is not reported to be caused by dura mater irritations with-

out previous surgical intervention (4,5). Malformations of the cervical spine are most commonly found at the occipital-C1 junction leading to the Chiari-like syndrome mainly seen in Cavalier King Charles Spaniels, but also in other small breeds (6), however malformations of the atlas (C1) vertebra are a rare finding.

Case 1: An 11 months old female Cavalier King Charles Spaniel was referred for the evaluation of neck pain of several weeks duration. The owner reported that the dog was reluctant to move and vocalized in pain when bending the neck. All clinical findings including body temperature,

blood hematology and chemistry were normal with exception of severe neck pain during the manipulation of the head/neck. No neurological deficits were present. A MRI scan was performed and a sample of cerebrospinal fluid was collected to exclude meningitis.

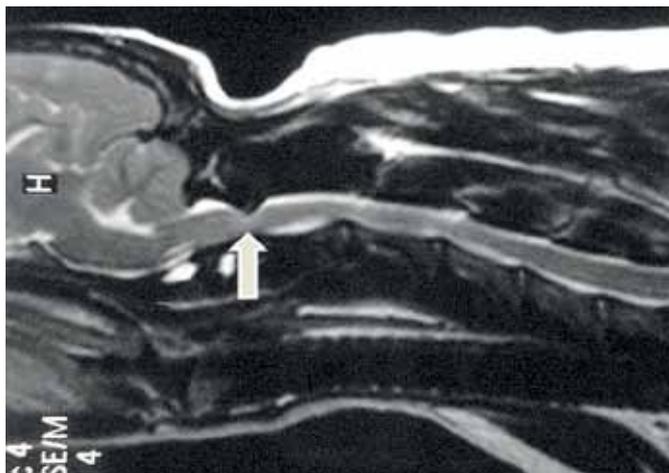
Case 2: A six year old female Norwich terrier was presented because of reluctance to run and play for the duration of several months. There were no significant clinical findings except for pain during the examination of the neck. No neurological deficits were present, and no abnormalities were detected on radiographs of the cervical

Resumé

To hunde blev præsenteret med kroniske nakkesmerter af non-traumatisk oprindelse. Ved den kliniske undersøgelse fandtes ingen neurologiske deficits, men der kunne udløses kraftige smerter ved manipulation af nakken. En MRI scanning afslørede hos begge hunde en malformation af den dorsale lamina af atlas (C1) med kronisk kompression af dura mater og rygmær. Der blev foretaget dekompressiv kirurgi med en laminectomi af den caudale atlas og den craniale C2. Under operationen kunne erkendes misfarving og fibrotisk væv på dura mater ved kompressionsstedet på case 1. Ved case 2 fandtes fibrosering og adhærencer mellem dura mater og ligamentum flavum og den spinale nerve. I begge tilfælde resulterede det kirurgiske indgreb i smertelindring.

Abstract

Two dogs were presented with signs of chronic neck pain of non-traumatic origin. On clinical examination there were no neurological deficits, but severe pain during manipulation of the neck. An MRI scan showed in both dogs a malformation of the dorsal lamina of atlas (C1) causing chronic compression of the dura mater and spinal cord. Decompressive surgery with a laminectomy of the caudal atlas and the cranial C2 revealed discoloration and fibrotic tissue on the dura mater at the site of the compression in case 1. In case 2 fibrosis and adhesions between the dura mater to the ligamentum flavum and the spinal nerve were encountered. The surgical treatment was successful in both patients alleviating the pain.



Picture 1. MRI of case 1 showing the dorsal malformation of the C1 (arrow) and mild signs of a Chiari-like malformation of the foramen magnum.



Picture 2. MRI of case 2 showing the dorsal malformation of the C1 (arrow).

spine. Conservative treatment with NSAID and gabapentin resulted in clinical improvement of the dog's activity and neck pain, but the symptoms returned when the medication was stopped. An MRI scan was then performed (Picture 2).

MRI findings: Case 1: In the T2 sagittal images, at the level of intervertebral space C1-C2, the spinal canal is decreased in diameter due to a hypointense dorsal opacity. An occipital hypoplasia and mild caudal herniation of cerebellum can be appreciated as well. In T2 transversal, at level C1-C2, the same hypointense opacity can be detected, reducing the vertical diameter of the spinal canal, forcing the spinal cord into an oval shape and reducing the subarachnoid space (Picture 1).

Case 2: In the T2 sagittal images, at the level of intervertebral space C1-C2, the spinal canal is decreased in diameter due to a hypointense dorsal opacity like in case 1. But the compression of the spinal cord is less pronounced. No signs of a Chiari-like malformation can be seen (Picture 2).

Analgesia and anesthesia: Both dogs received methadon for premedication (0.3 mg/kg im), midazolam (0.2 mg/kg iv) and propofol (40 mg/kg iv) for induction and intubation followed by isoflurane/O₂ for maintenance. After the surgery the dogs were treated with methadone (0.3 mg/kg im) every 4-6 hours before discharge the next day; and

they received NSAID (carprofen 4mg/kg po) and tramadol (3 mg/kg po every 8-12 hours) after discharge.

Surgery: In both dogs an approach to the dorsal C1-C2 junction was performed. The caudal part of the dorsal lamina of C1 and the cranial part of the dorsal lamina of the C2 including the most cranial part of the spinal process was removed with a small Smith-Kerrison Punch Forceps; the dorsal fibrous tissue (ligamentum flavum) between C1-C2 was carefully resected (Figure 1).

In case 1 the dura mater (DM) under the compressing malformed end of the C1 was showing a yellowish discoloration caused by the formation of fibrotic tissue (Picture 3). No adhesion of the DM to the liga-

mentum flavum was found, the spinal nerves were free of scar tissue.

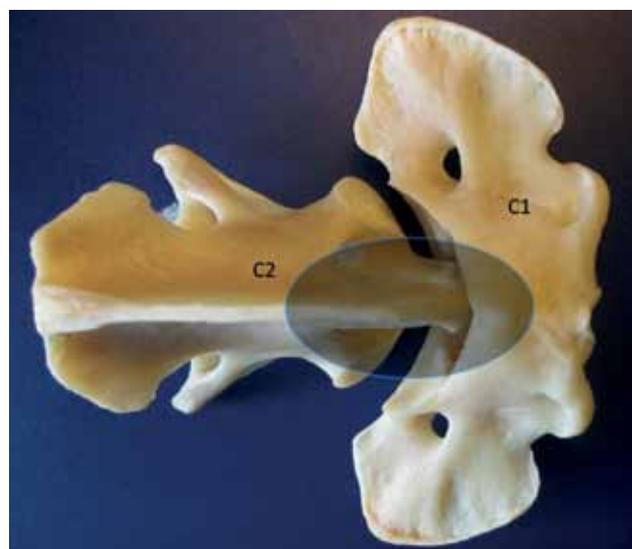
In case 2, the extent and amount of newly formed fibrotic tissue was more pronounced, the DM was strongly adhered to the ligamentum flavum, and the adhesions involved the dorso-lateral aspects of the DM including the spinal nerves on both sides (Picture 4). In this case, the adhesions were carefully dissected until the spinal nerves were visible.

An autogenous fat graft was placed in the laminectomy defects to prevent further adhesions (4,5), and the muscle/fascia and subcutis/intradermal layers were closed with absorbable interrupted sutures.

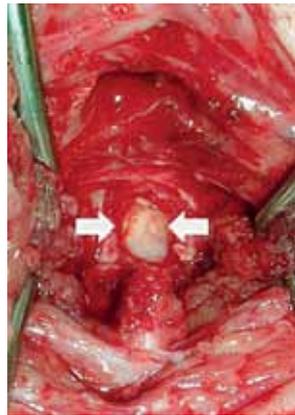
Both dogs recovered well from anesthesia and showed no neurological deficits



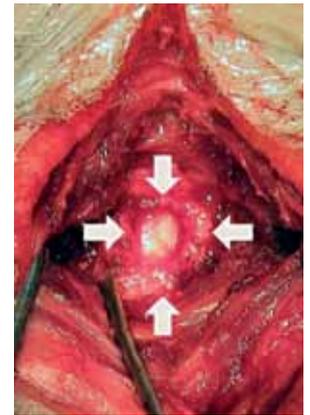
Figure 1. Extend of the dorsal laminectomy C1-C2.



Picture 3. Intraoperative findings of case 1 showing the fibrotic changes of the DM (arrows).



Picture 4. Intraoperative findings of case 2 showing DM fibrosis and extensive adhesions (arrows) of the DM to the (removed) ligamentum flavum and the bone involving the spinal nerve ganglions.



caused by the intervention. Case 1 had immediately after the surgery reduced pain according to the owner and was normal at the following examinations by the referring veterinarian. Case 2 showed 7 days after the surgery reduced pain, was walking normally, but still reacted during manipulation of the neck. However over the next four weeks the dog continued to improve and eventually was moving normally, no longer showing signs of pain in the neck.

Discussion

Chronic compression of the spinal cord/dura mater (DM) is a common neurological diagnosis in dogs and is mainly caused by vertebral malformations, tumors or disc protrusions Hansen type II (7). Surgical removal of the compression leads in most cases to a good result. The DM is rarely severely changed by the compression in the cervical and thoraco-lumbar spine; more commonly fibrotic changes and adhesions have been found by the authors during decompressive surgery of a cauda equina syndrome (L7-S1). In experimental laminectomies/hemilaminectomies fibrosis of the DM and adhesions to the sur-

rounding soft tissues have been found at the surgical site (4,5). Therefore placing an autogenous fat graft into the bone defect to cover the DM has been recommended based on histologic examinations (4,5). In the here described cases the compression was massive enough in case 1 to cause the clinical symptoms, but in case 2 the compression of the spinal cord was minimal. It can be assumed that the fibrosis/adhesion were also a source of the strong pain in this patient. The DM moves in relation to the bony spinal canal during bending and extending the neck, facilitated by the epidural fat; adhesion of the DM limits this shifting and causes stretching of the DM and involved spinal nerves like in case 2. In human medicine post-laminectomy adhesions are a common complication inflicting chronic pain (1,2,3). The extent of movement between the DM and the spinal canal is maximally in the flexible cranial cervical spine. The changes of the DM in the here described dogs could be the result of continuous friction between the malformed end of the C1 causing compression and the moving DM after the epidural fat was pushed away by the compression. The more ex-

tended formation of fibrotic tissue in case 2 could be explained by the longer duration of the compression (age of case 2 was six years compared to the age of 11 months in case 1). The owner of case 2 had not noticed any signs of neck pain in the first 5 years of their dog's life, therefore it is likely that the pain was also inflicted by the adhesions, and not only by the mild spinal cord compression visible on the MRI scan.

In case 1 a syringo-hydromyelia (Chiari-like syndrome) was the differential diagnosis number one, Cavalier King Charles Spaniel being affected very often by this malformation in the area of occipital bone-C1 (6). There were no signs of an intramedullary cyst formation on the MRI in case 1, it is assumed that the visible compression was causing the clinical symptoms in this young Cavalier King Charles Spaniel.

In both cases the dorsal laminectomy was successful in removing the compression and the adhesions, and as result the pain was alleviated in these two patients and should be considered as a therapeutical approach to these cases. ■

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