Complications in a dog with disc associated Wobbler syndrome

Complicaties bij een hond met discusgeassocieerd Wobblersyndroom

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ABSTRACT

This case report describes the occurrence of a second episode of clinical signs of disc associated Wobbler syndrome almost 2 years following ventral decompressive surgery and a rather rare complication after myelographic examination in the same dog.

SAMENVATTING

In deze casuïstiek worden het ontstaan van een tweede episode van klinische klachten van het discusgeassocieerd Wobblersyndroom bijna 2 jaar na een ventrale decompressieve chirurgische behandeling beschreven en het optreden van een eerder zeldzame complicatie na het myelografisch onderzoek bij dezelfde hond.

INTRODUCTION

Disc associated Wobbler syndrome (DAWS) is the most predominant and typical Wobbler syndrome in the dog, and it typically affects middle-aged large-breed dogs. The clinical presentation can vary from neck pain only to tetraplegia. The most typical clinical signs are a paraparetic wide-based, uncoordinated ataxia of the hind limbs, in combination with a short stilted gait of the front limbs. Myelographic examination is the diagnostic method of choice (Sharp and Wheeler, 2005). Although the incidence of postmyelographic complications has dramatically decreased since the use of the newer generation water-soluble, non-ionic contrast media (e.g. iohexol and iopamidol), this diagnostic technique is not completely without risk and different myelographic complications have been described (Widmer and Blevins, 1991). The most important of these are transient neurological deterioration and seizures (Lewis and Hosgood, 1992). DAWS is considered to be a progressive disease in which early surgical intervention is necessary to halt the progression of symptoms (McKee and Sharp, 2003; Sharp and Wheeler, 2005). Many different surgical techniques have been described and many authors claim between 70 and 80% success rates immediately postoperatively (Seim, 2000; McKee and Sharp, 2003; Sharp and Wheeler, 2005). Despite initial success, 20-30% of these dogs will suffer from a second episode of clinical signs. This can be caused by spinal cord compression at the same intervertebral disc space or compression at one of the adjacent intervertebral disc spaces. The latter is termed a 'domino lesion' and may occur after both ventral decompressive and vertebral distraction-stabilization techniques (Jeffery and McKee, 2001; McKee and Sharp, 2003; Sharp and Wheeler, 2005). The exact cause of this phenomenon is unknown but it might indicate that DAWS is not only a multifactorial but possibly also a multifocal disease (Jeffery and McKee, 2001).

CASE DESCRIPTION

A 4-year-old male Weimaraner dog weighing 30 kg was admitted to the Department of Small Animal Medicine and Clinical Biology with the complaint of nonambulatory tetraparesis, which had started on the previous day. The owners had not noticed any abnormalities in the days or weeks prior to presentation. Physical examination revealed no abnormalities. Abnormalities on neurological examination were conscious proprioceptive deficits in all four limbs, increased spinal reflexes in the hind limbs and decreased spinal reflexes in the right front limb. Neck pain could not be elicited. The neurological examination was suggestive of a cervicothoracic spinal cord lesion. All complete blood count (CBC) and

biochemistry panel values were within normal limits. A cardiologic examination including an echocardiogram was unremarkable. The dog was premedicated with methadone (0,1 mg/kg, IV, Mephenon®). Anesthesia was induced using propofol (100 mg, IV, Rapinovet®) and maintained with isoflurane in oxygen. Survey radiographs of the cervical spine were unremarkable. A cervical myelogram was performed using iomeprol (0.2 ml/kg with a maximum dose of 8-9 ml, 300 mgI/ml, Iomeron 300®) injected via cisternal puncture. In addition to standard lateral and ventrodorsal views, lateral views were obtained with the neck in flexion and gentle extension of the cervical region. A ventral, extradural compressive lesion was observed at the level of the intervertebral disc space between the sixth and seventh cervical vertebra (C6-C7) (Figure 1). The spinal cord compression worsened with gentle extension and improved with flexion. In addition, a slight dorsal deviation without attenuation of the ventral contrast column between C5-C6 and C4-C5 was observed. However, no narrowing of the contrast columns was present. Based on these findings, DAWS affecting C6-C7 was diagnosed. Examination of cerebrospinal fluid (CSF) revealed no abnormalities. Anesthetic recovery was uneventful and the dog was scheduled for decompressive surgery. Ventral decompressive surgery was performed 6 days following diagnosis. The preanesthetic medication included acepromazine (0.01 mg/kg, IV, Placivet®), methadone (0.1 mg/kg, IV, Mephenon®), and atropine (0.02 mg/kg, IM, Stellatropine®). Anesthesia was induced with thiopenthal (350 mg, IV, Pentothal®) and maintained with isoflurane in oxygen. Preoperative corticosteroid (30 mg/kg, IV, Soludeltacortef®) treatment was administered. A standard ventral slot was made at the level of C6-C7. Protruded annulus fibrosus and dorsal longitudinal ligament were removed. Anesthetic recovery was uneventful. Progressive postoperative neurological improvement was observed. Six days after surgery, when the dog could walk unassisted, he was discharged from hospital. The clinical symptoms further improved over the next weeks and months. On follow-up examination 11 months later, the dog had improved greatly but still showed conscious proprioceptive deficits and ataxia in the right hind limb. According to the owners, these symptoms were not progressive in nature and had been present since the dog regained ambulatory status after surgery.

Twenty-two months following surgery, the dog was re-admitted to the Department of Small Animal Medicine and Clinical Biology with the complaint of a progressive deterioration of neurological status in the last two months. The dog showed severe but ambulatory ataxia of the hind limbs and a short stilted gait of the front limbs. Physical examination revealed no abnormalities. Neurological examination demonstrated the aforementioned ataxia and conscious proprioceptive deficits of the right hind limb. Neck pain could not be elicited. A CBC and a biochemistry panel were unremarkable. General anesthesia was performed as during the first myelogram, the only

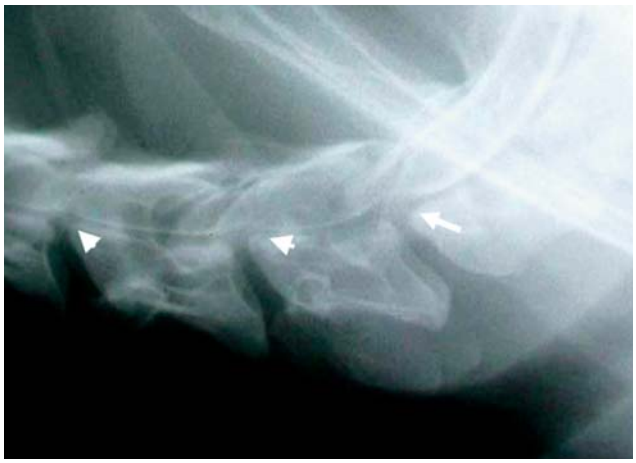

Figure 1. Lateral neutral myelogram at initial presentation showing extradural spinal cord compression at the level of C6-C7 (arrow) and milder lesions at the levels of C5-C6 and C4-C5 (arrowheads).

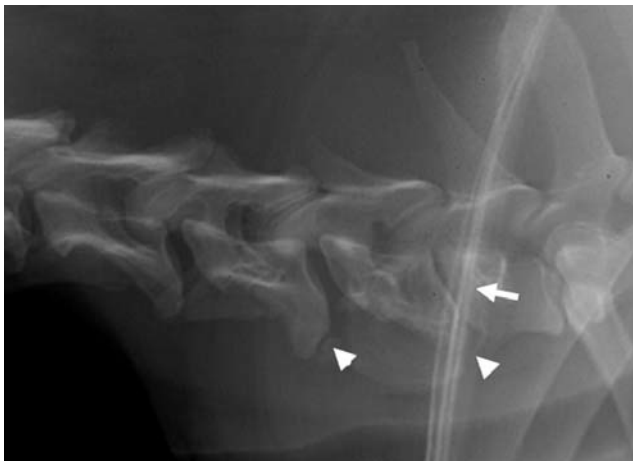

Figure 2. Survey radiograph 22 months following surgery. Severe narrowing of the intervertebral disc space between C6 and C7 (arrow) and new bone formation at the ventral aspects of C6-C7 and C5-C6 can be noticed (arrowheads).

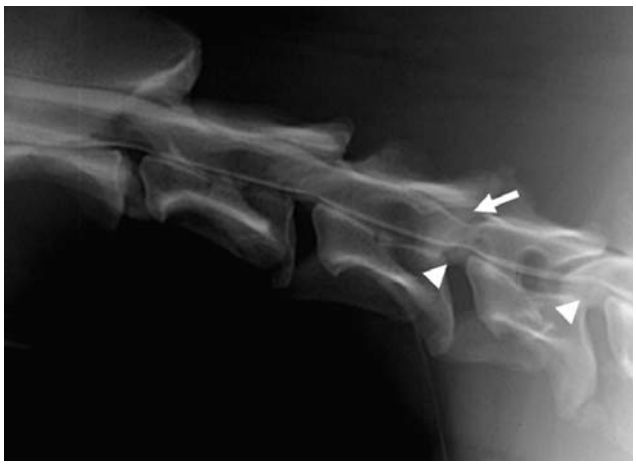

Figure 3. Neutral myelogram of the midcervical region 22 months following surgery. Dorsal compression at the level of C4-C5 (arrow) and milder ventral lesions at the levels of C4-C5 and C5-C6 (arrowheads), resulting in severe narrowing of the spinal cord at the level of C4-C5.

difference being that thiopenthal (400 mg, IV, Pentothal®) was used instead of propofol for the induction of anesthesia. Survey radiographs of the cervical region demonstrated a severe narrowing of the intervertebral disc space between C6 and C7, and new bone formation at the ventral aspects of C6-C7 and C5-C6 (Figure 2). A cervical myelogram was performed using the same protocol as the first time. The myelographic study revealed ventral deviation of the dorsal contrast column at C4-C5 and mild dorsal deviation of the ventral contrast column at the levels of C4-C5, C5-C6 and C6-C7. The spinal cord was clearly attenuated at the level of C4-C5 (Figure 3). These radiographic findings could be explained by ventral compression due to intervertebral disc protrusion and by dorsal compression due to hypertrophy of the ligamentum flavum. DAWS, affecting C4-C5, was diagnosed on the basis of these findings. The owners declined another surgical procedure and preferred a conservative therapy. During anesthetic recovery the dog started shivering and developed hyperthermia (39.9°C). He did not show symptoms compatible with epileptic seizure such as loss of consciousness, tonic-clonic movements or autonomic signs (involuntary salivation, urination and defecation). An adverse reaction to the myelographic contrast medium was suspected. Cooling therapy, fluid therapy (Hartmann's at a rate of 3.5 ml/kg/h), and antibiotic therapy with cefalexine (20 mg/kg, IV, q8h, Cefacidal®) were instituted, and a single dose of corticoids (10 mg/kg, IV, Soludeltacortef®) was given. The dog stopped shivering after a few hours. The hyperthermia disappeared as soon as he stopped shivering. Although the dog was conscious, he appeared exhausted and was hospitalized for observation. He further recovered during the night and was discharged from the clinic the next day. The conservative therapy consisted of cage confinement for 4 weeks and a diminishing oral corticosteroid therapy for 3 weeks (1 week 1 mg/kg sid, 1 week 0.5 mg/kg sid and 1 week 0.25 mg/kg sid, Prednisolone®). The owners were contacted by telephone 46 months after the surgical procedure (24 months following the recurrence of clinical signs). According to the owners the clinical status of the dog had improved, although the dog still showed ambulatory ataxia, which was brought under control with intermittent oral corticosteroid (Prednisolone®) therapy as needed.

DISCUSSION

Disc associated Wobbler syndrome (DAWS) is a relatively common cause of neurological dysfunction in middle-aged, large breed dogs. The most typical presentation is a chronic progressive gait disturbance, mainly affecting the hind limbs. An acute onset of clinical signs, as in this dog, also occurs (McKee and Sharp, 2003; Sharp and Wheeler, 2005). Although myelography is the diagnostic method of choice in veterinary medicine, this diagnostic technique is not entirely without risk. Exacerbation of neurological abnormalities the day after myelography and seizures during recovery are the most important and most common complications (Widmer an Blevins, 1991; Lewis and Hosgood, 1992; Sharp *et al*., 1992). Fortunately, this neurological deterioration is typically transient and resolves spontaneously after a few days. Lewis and Hosgood (1992) demonstrated a higher risk of postmyelographic complications in dogs with DAWS compared to dogs suffering from other cervical spinal cord lesions. In a retrospective study of 66 dogs undergoing cervical myelography, deterioration of neurological status was the most common complication, occurring in 21% of the dogs. Dogs with DAWS, extradural neoplasia and meningitis or myelitis had a significantly greater likelihood of neurological deterioration the day after myelography, compared to other dogs. Seizures occurred in 9% of the dogs after myelography. All of these were Dobermann Pinschers afflicted with DAWS. Other occasionally reported myelographic complications in human and veterinary medicine include headache, nuchal pain, nausea, vomiting, meningeal irritation, hyperthermia, neurobehavioral disturbances (altered mental state), hypotension, bradycardia during injection of contrast medium, and death (Widmer and Blevins, 1991; Lewis and Hosgood, 1992). The complication that occurred in the dog described in this case could possibly have been caused by an adverse reaction to the potentially irritant contrast medium, which is a very rare complication. The veterinary literature lacks comparable data of cases with the same unusual complication of a possible adverse reaction after myelography. The prior list of possible complications illustrates the main disadvantage of myelography: that it is a rather invasive diagnostic procedure. Advanced and less invasive medical imaging techniques, such as computed tomography (CT) and magnetic resonance imaging (MRI), have been developed and introduced into veterinary medicine in the last decades. Little is known about their diagnostic value for dogs with DAWS (Sharp *et al*., 1992; Sharp and Wheeler, 2005).

There is a lot of controversy about the treatment of DAWS (Jeffery and McKee, 2001). Several surgical techniques have been developed and these can be divided into direct decompression by ventral or dorsal decompressive surgery, and indirect decompression by linear traction combined with intervertebral stabilization (Sharp and Wheeler, 2005). Performing direct ventral decompressive surgery by creating a ventral slot is a common way to treat dogs with DAWS. Although this is a standard neurosurgical technique, it can be a technical challenge in dogs with DAWS. Intraoperative bleeding, vertebral malformations, difficult accessibility of the caudal cervical region and difficult removal of all protruding material can complicate the surgical procedure (Seim, 2000; McKee and Sharp, 2003; Sharp and Wheeler, 2005). The dog in the present case improved remarkably after surgery, although he continued to demonstrate residual signs in his right hind limb. Incomplete removal of all protruding annulus fibrosus or dorsal longitudinal ligamentous tissue at the time of decompressive

surgery is the most likely reason. After a ventral slot procedure, there is the risk of a mild collapse of the intervertebral disc space. This will give the opportunity for incompletely removed tissue to prolapse into the vertebral canal and cause residual or even worse spinal cord compression (VanGundy, 1988; McKee and Sharp, 2003; Sharp and Wheeler, 2005). This hypothesis is fed by the fact that the second myelographic examination still showed a mild dorsal deviation of the ventral contrast column at the level of the previously operated site. Twenty-two months after the surgical procedure, the dog experienced a deterioration of the clinical signs. According to the second myelographic examination, a new compressive lesion had developed at the level of C4-C5. This complication, which is termed a domino lesion, is the most common and most important complication after surgery for DAWS (Jeffery and McKee, 2001). It occurs 5 to 60 months after surgery in 20-30% of the cases and is independent of the surgical technique (Wilson *et al*., 1994; Rusbridge *et al*., 1998; Marchevsky and Richardson, 1999). Although several theories have been developed, the exact pathogenesis and etiology of this phenomenon remain unknown. Additional disc protrusions may result when intervertebral fusion or stabilization at one level leads to increased stress at one of the adjacent disc spaces. Degeneration and protrusion of additional discs can also result from continued natural disease progression rather than from the degeneration of a previously normal disc. Another possibility is a combination of these factors (Wilson *et al.*, 1994; Jeffery and McKee, 2001, McKee and Sharp, 2003; Sharp and Wheeler, 2005). In retrospect, a close examination of the preoperative myelogram in the present case suggests that there could already have been disc degeneration and a mild degree of disc protrusion at two other sites (C4-C5 and C5-C6) in the cervical vertebral column at the time of the initial diagnostic evaluation. Therefore, it seems logical that the development of the second episode of clinical signs in our dog can be attributed to the natural progression of the disease. Whether the vertebral fusion caused by the ventral slot technique performed played a cooperative role in the development of this domino lesion is unclear. The same surgical technique is also used to treat other cervical compressive lesions. Acute intervertebral disk disease with extrusion of the nucleus pulposus (Hansen type I) is commonly treated surgically (with excellent results) by ventral decompression through a ventral slot (Sharp and Wheeler, 2005b). Reported complications following the ventral slot procedure for intervertebral disk disease include venous sinus hemorrhage, neurological deterioration, Horner's syndrome, respiratory complications, hypotension and bradycardia, cardiac arrhythmias and instability with subsequent subluxation of the surgical site (Lemarié *et al*., 2000; Sharp and Wheeler, 2005b). A domino lesion is a complication that very seldom occurs after ventral decompressive surgery for acute cervical intervertebral disk disease (Bagley *et al.*, 1996). A recent report described the postsurgical complications, with emphasis on vertebral subluxation, in 9 dogs after a ventral slot procedure in the caudal cervical region. Two of these dogs were older Dobermann Pinschers with DAWS. Only one of these 9 dogs developed a domino lesion. Not surprisingly, this occurred in one of the dogs with DAWS (Lemarié *et al*., 2000). This data supports the hypothesis that the occurrence of a domino lesion is a complication related to the natural progression of this disease rather than to a true postoperative complication. This could be suggestive of a possible multifocal nature of DAWS. Several authors have suggested routinely fusing multiple disc spaces to reduce the incidence of domino lesions (Jeffery and McKee, 2001; McKee and Sharp, 2003; Sharp and Wheeler, 2005). In humans, the risk of new disease at an adjacent level was found to be significantly lower following a multi-level arthrodesis, than it was following a single-level arthrodesis (Hilibrand *et al*., 1999). Results of such an approach in veterinary medicine are currently lacking and should be explored.

CONCLUSION

Although myelography is the method of choice for DAWS, further investigation is warranted to assess the clinical use of less invasive and more advanced medical imaging techniques like CT and MRI. Several surgical techniques have been described to treat DAWS, but none of them can avoid the possible development of a domino lesion. Further investigation is warranted to develop a surgical technique that avoids or decreases the incidence of this surgical complication.

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