Dura mater marsupialisation and outcome in a cat with a spinal subarachnoid pseudocyst: a case report

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ABSTRACT: A six-month-old male domestic shorthair cat was referred with a history of acute-onset paraplegia, over the previous two months. The neurological examination revealed a thoracolumbar lesion. After myelography and myelo-computed tomography (myelo-CT), the diagnosis of a T13–L1 subarachnoid pseudocyst potentially related to a previous L1 vertebral body fracture or malformation was made. Surgical decompression consisted in dorsal laminectomy followed by durotomy and marsupialisation. Immediately after surgery the cat improved neurologically and showed progressive improvement of his neurological signs over the next few months, until he died, from unrelated causes, approximately 18 months after surgery.

Keywords: cat; subarachnoid pseudocyst; marsupialisation; myelo-CT; myelography

List of abbreviations

CBC = cell blood count, CSF = cerebrospinal fluid, CT = computed tomography, myelo-CT = myelo-computed tomography

Subarachnoid pseudocysts are uncommon spinal pathologies in animals and are usually associated with chronic and focal neurological signs (Jaggy and Platt 2010). The aetiology and pathophysiology of this disease are not entirely clear, although some authors describe the alteration of cerebrospinal fluid (CSF) flow patterns along the spinal subarachnoid space as the major cause for the formation of these pseudocysts (DeLahunta and Glass 2009a; DeLahunta and Glass 2009b). The surgical approach is complicated and durotomy with the marsupialisation technique has met with major clinical success (Skeen et al. 2003). This case report describes the treatment of a possibly acquired subarachnoid pseudocyst associated to a vertebral abnormality in a paraplegic cat.

Case description

A six-month-old male domestic short hair cat with a two month history of acute paraplegia, was referred to the Teaching Hospital of the Faculty of Veterinary Medicine of the University of Bologna, Italy. According to the owners, the cat was found paraplegic after he was left alone in the house and, due to the presence of back pain noted by the referring veterinarian, a trauma could not be excluded. In the two months before our examination, conservative treatment was performed consisting of cage rest, manual bladder compression, oral antibiotics and prednisone. General physical examination was unremarkable. The neurological examination was performed by a board-certified neurologist and neurological findings included sternal recumbency and paraplegia with normal spinal reflexes. The cutaneous trunci reflex was absent caudally to the L2 vertebral segment. Nociception was markedly decreased in both hind limbs. The bladder was overdistended and only manual compression allowed micturition. No signs of spinal pain were detected. The lesion was localised to the T3-L3 spinal cord segments and the differential diagnoses included traumatic, anomalous, vascular or, less likely, infectious/inflammatory disease. Advanced diagnostic imaging was suggested but, due to the financial limitations of the owner, only cell blood count (CBC), biochemical profile and medio-lateral

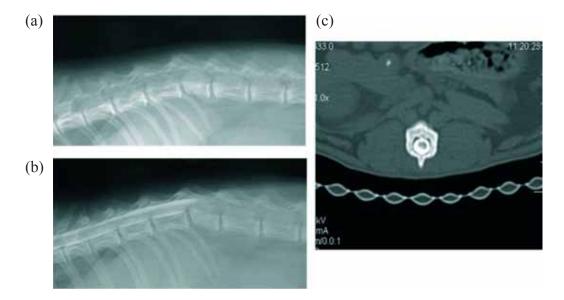


Figure 1. X-ray image, myelography, myelo-CT. X-ray, showed an abnormal size and profile of the L1 vertebral body, consistent with a previous vertebral fracture or malformation (a). Myelography showed an abrupt interruption of the progression of the contrast medium, associated with thickening of the dorsal contrast column in correspondence of the T13–L1 intervertebral space (b). Myelo-CT confirmed the compression of the spinal cord of the T13–L1 and the presence of L1 vertebral body alterations, consistent with a previous fracture or malformation (c)

X-ray of the thoracolumbar spine were performed. Blood results were normal while the X-ray revealed an abnormal size and profile of the L1 vertebral body (Figure 1a). The owners refused further investigations and the cat was lost to the follow-up. The cat was re-examined after a further four months, after being adopted by a shelter. His neurological condition was unchanged and myelography and myelo-computed tomography (myelo-CT) of the thoracolumbar region were planned. After general anaesthesia, 0.35 ml/kg of contrast medium (iodixanol-320 – Visipaque®) was injected into the subarachnoid space from the cisterna magna and the myelogram showed an abrupt interruption of the progression of the contrast medium associated with thickening of the dorsal contrast column in correspondence of the T13-L1 intervertebral space, causing severe dorsal extradural compression of the spinal cord (Figure 1b). Myelo-CT of the T10-L3 area confirmed the presence of L1 vertebral body shape changes, consistent with a previous fracture or malformation, and the dorsal compression of the spinal cord due to the presence of focal accumulation of cerebrospinal fluid in the subarachnoid space (Figure 1c). A final diagnosis of spinal cord compression due to a suspected subarachnoid pseudocyst was made. Surgical treatment, consisting of dorsal laminectomy, durotomy and dura mater marsupialisation, was planned. After general anaesthesia using a standard protocol, dorsal laminectomy was performed at the level of the T13-L1 space. In correspondence of L1, due to the possible abnormal biomechanical resistance of the vertebral body, the surgeon preferred to maintain part of the spinal arch and the articular processes of T13-L1 to avoid destabilisation of the spine (Figure 2). The dura mater was abnormally thin and transparent, and the spinal cord appeared to be displaced ventrally. During durotomy, a colourless cerebrospinal-like fluid drained from the pseudocyst, allowing decompression of the spinal cord, which immediately resumed its normal position. The marsupialisation was made by suturing the dura mater edges at the level of L1 (using the non-absorbable suture 6/0 ProleneTM) to the laminar periosteum and the articular facet joint capsules (Figure 2). Specific care was made in avoiding excessive detachment of the periosteum and soft tissues in order to have enough space to anchor the suture and avoid excessive traction on the meningeal flaps. The surgical wound was closed using routine procedures. Shortly after surgical decompression the nociception improved and the cat began to show minimal voluntary movements in the hind limbs. The neurological examination was repeated on a weekly basis, by the same boardcertified neurologist. Over the next months the cat showed progressive improvement in the hind







Figure 2. Intraoperative image of pseudocyst marsupialisation. Dura mater marsupialisation at the level of the L1 vertebra (a, b)

limbs movement leading, and approximately three months after surgery, had recovered the ability to walk unassisted and to urinate spontaneously. His condition remained unchanged until he died, hit by car approximately 18 months after surgery.

DISCUSSION AND CONCLUSIONS

Subarachnoid pseudocysts, also known as "spinal subarachnoid pseudocysts", are usually associated with chronic and focal neurological signs with possible congenital, vascular, inflammatory or traumatic aetiology (Jaggy and Platt 2010). In the past, subarachnoid pseudocysts were defined as "subarachnoid cysts" but, although on myelograms and MR images they have the appearance of cysts, the subarachnoid space readily fills with contrast during myelography. This finding supports the idea that they are pseudocysts of the subarachnoid space. Therefore, the term "cyst" is a misnomer (DeLahunta and Glass 2009b). An altered cerebrospinal fluid (CSF) flow pattern along the spinal subarachnoid space may contribute to the formation of these pseudocysts (DeLahunta and Glass 2009b), although their pathophysiology and the associated myelopathy are poorly understood. Spynal subarachnoid pseudocysts are well known and have previously been described in dogs (Bently et al. 1991; Dyce et al. 1991; McKeen and Renwick 1994; Hardie et al. 1996; Bagley et al. 1997; Mauler et al. 2012). However, there are only a few reports which describe their occurrence in cats (Shamir et al. 1997; Vignoli et al. 1999; Schmidt et al. 2007; Sugiyama and Simpson 2009). In our case, the acute and severe onset of neurological signs along with the presence of initial back pain were suggestive of an external trauma which may have played a significant role in the spinal subarachnoid pseudocyst formation. Since the pseudocyst was detected at the same level as the L1 vertebral body alteration, we assumed a close relationship between the trauma, the abnormality of the L1 shape (considered as a possible previous fracture), and the formation of the pseudocyst. In a recent study, a similar correlation was also reported in 13 out of 122 dogs with spinal arachnoid pseudocysts (Mauler et al. 2012). Nevertheless, due to the chronicity of the suspected trauma in our case, CT findings could not rule out that L1 abnormalities were the result of a congenital malformation. Considering the good recovery starting immediately after the surgery, the severe neurological condition observed was probably principally due to the spinal cord compression maintained by the CSF accumulation in the pseudocysts. Although MRI is considered the gold standard for diagnosis of subarachnoid pseudocysts, myelography and myelo-CT still represent useful diagnostic techniques in most instances (Galloway et al. 1999; Vignoli et al. 1999; DeLahunta and Glass 2009b; Sharp and Wheeler 2009; Sugiyama and Simpson 2009; Ricci et al. 2011). In our case the CT scan was essential to investigate in more detail the vertebral body structure and the spinal cord compression. This allowed the identification of the previous fracture of the L1 vertebra as a possible causative factor. The treatment of subarachnoid pseudocysts typically requires a surgical approach (Sharp and Wheeler 2009) that considers two different entries to the spinal cord: dorsal laminectomy (McKeen and Renwick 1994; Frykman 1999; Sugiyama and Simpson 2009; Ricci et al. 2011) or hemilaminectomy (Shamir et al. 1997; Gnirs et al. 2003; Schmidt et al. 2007). Two different treatments can be performed, the simple fenestration technique (durotomy or durectomy) (Shamir et al. 1997; Frykman 1999; Jurina and Grevel 2004; Sugiyama and Simpson 2009; Ricci et al. 2011), or durotomy associated with dura mater marsupialisation (McKeen and Renwick 1994; Jurina and Grevel 2004; Schmidt et al. 2007; Sharp and Wheeler 2009). As many authors suggest (McKeen and Renwick 1994; Sugiyama and Simpson 2009; Ricci et al. 2011), we performed a dorsal laminectomy and marsupialisation, with consideration of the better reported outcome (McKeen and Renwick 1994; Jurina and Grevel 2004). Simple durotomy or durectomy represent good techniques for treatment of subarachnoid pseudocysts but, in the authors' opinion, neither maintain persistent drainage of the pseudocysts since they often show recurrence (Frykman 1999; Sugiyama and Simpson 2009). Marsupialisation of the pseudocysts may provide permanent drainage of cerebrospinal fluid and often prevents recurrence (McKeen and Renwick 1994; Frykman 1999; Schmidt et al. 2007; Sugiyama and Simpson 2009; Sharp and Wheeler 2009). There are several descriptions of the marsupialisation technique in dogs (McKeen and Renwick 1994; Jurina and Grevel 2004) and in humans (Palmer 1974), whilst in cats, to our knowledge, only one case was briefly described (Schmidt et al. 2007). According to some authors (McKeen and Renwick 1994), this technique may represent a challenge for the surgeon, because the small size of the patient may make complicate the fixing of the dura mater edges to the periarticular soft tissues. To overcome this problem, it is crucial not to detach the soft tissues and periosteum too widely. In this way sufficient space is provided for the suture anchors to prevent excessive traction on the meningeal flaps by the suture, in order to prevent relapse and spinal stress. In dogs, the time of recurrence ranges from five months to four years (McKeen and Renwick 1994; Frykman 1999; Gnirs et al. 2003; Jurina and Grevel 2004) and in cats it ranges from 10 months to four years (Schmidt et al. 2007; Sugiyama and Simpson 2009). Although recurrence following utilisation of the marsupialisation technique was described (Schmidt et al. 2007), this procedure still represents the best treatment for this spinal disease (Skeen et al. 2003). Our follow-up was not long enough to exclude recurrence in the long term because, unfortunately, the cat died 18 months after the surgery from unrelated causes. In this cat, the prompt improvement of neurological signs start-

ing immediately after surgery, suggests that the decompression of the spinal cord using this type of surgery may be relevant for a positive outcome and should always be encouraged and performed, even in patients presenting with chronic severe neurological dysfunction.

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