

Peritumoral (pseudo)cystic meningioma in a cat

Peritumoraal (pseudo)cysteus meningeoom bij een kat

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ABSTRACT

A nine-year-old, male, neutered domestic shorthair cat was referred with a three-month history of abnormal behavior. On neurological examination, the cat showed circling towards the left. Magnetic resonance imaging of the brain revealed an extra-axial mass at the level of the left forebrain surrounded by a large peritumoral (pseudo)cyst. A left-sided rostral tentorial craniectomy to drain the fluid and to remove the tumor was performed. On histopathologic examination, the presence of a meningioma was confirmed. The owners did not report any clinical signs one year postoperatively.

SAMENVATTING

Een negen jaar oude, mannelijke, gecastreerde Europese korthaar werd doorverwezen omwille van abnormaal gedrag dat al drie maanden duurde. Op neurologisch onderzoek vertoonde de kat cirkelgang naar links. Een MR-scan van de hersenen toonde een extra-axiale massa ter hoogte van de linkerhersen helft aan, omringd door een grote peritumorale (pseudo)cyste. Via een rostral tentoriale craniëctomie aan de linkerkant werd de peritumorale (pseudo)cyste gedraineerd en werd de tumor verwijderd. Op histopathologisch onderzoek werd de aanwezigheid van een meningeoom bevestigd. Een jaar na de operatie deed de kat het nog steeds goed en vertoonde volgens de eigenaar geen klinische tekenen.

INTRODUCTION

Meningioma is the most commonly reported primary brain tumor in cats (Troxel et al., 2003; Troxel et al., 2004; Tomek et al., 2006). Feline meningiomas are considered benign, slow-growing, mostly solitary tumors arising from the arachnoid cells of the meninges (Cameron et al., 2015). In cats, the most common locations are the tela choroidea of the third ventricle and the supratentorial meninges (Troxel et al., 2003). Multiple meningiomas are not uncommon in cats and have been reported in 10% of cats diagnosed with intracranial neoplasia, and in 17.2% of cats diagnosed with intracranial meningioma (Troxel et al., 2003; Forterre et al., 2007). The most common neurological signs are behavioral changes, ataxia and seizures, although some cats do not show any neurological signs. Nonspecific clinical signs of lethargy and anorexia are commonly present (Troxel et al., 2003; Cameron et al., 2015). Magnetic resonance

imaging (MRI) is considered the imaging modality of choice for the investigation of brain neoplasia (Troxel et al., 2004). The treatment of choice for cats with meningiomas is surgical excision, as in this species meningiomas tend to be well-encapsulated and can easily be separated from the normal brain (Gallagher et al., 1993; Gordon et al., 1994; Troxel et al., 2003; Cameron et al. 2015). Surgical treatment is generally associated with a good clinical outcome, and a median survival time of up to 37 months has been reported (Gallagher et al., 1993; Gordon et al., 1994; Troxel et al., 2003; Cameron et al. 2015). In this case report, the clinical signs, MRI findings and treatment of a cat with a peritumoral (pseudo)cystic meningioma are described. A cystic meningioma has only been reported in one cat in the literature (Troxel et al., 2004) and to the authors' knowledge, this is the first case report of a peritumoral (pseudo)cystic meningioma in a cat.

CASE

A nine-year-old, male, neutered domestic shorthair cat was presented with a three-month history of circling to the left, which was gradually increasing in frequency. General physical examination was unremarkable. The cat was circling compulsively to the left but no other abnormalities were found on neurological examination. The neuroanatomical localization was determined to be left forebrain and the main differential diagnoses were neoplasia, infection, inflammation or degenerative disease. A vascular event or trauma were considered less likely due to the chronic and progressive nature of the neurological signs. Blood tests including hematology and biochemistry revealed no significant abnormalities.

An MRI scan (0.4T Aperto, Hitachi, Tokyo, Japan) of the brain was performed under general anesthesia. The cat was premedicated intramuscularly with butorphanol (0.2 mg/kg) and medetomidine (6 µg/kg). Anesthesia was induced with propofol (3 mg/kg intravenously (IV)) and maintained with sevoflurane (2%) vaporized in a combination of medical air and oxygen with an inspired oxygen fraction of 0.5. The MRI protocol included a sagittal T2-weighted (T2w; TE 120, TR 3690; 3 mm slice thickness), a transverse T2-weighted (T2w; TE 112, TR 2505), FLAIR (fluid attenuated inversion recovery; TE 87, TR 8359), T2* GRE (gradient echo; TE 50, TR 656), and transverse T1-weighted (T1w) pre- and postcontrast sequences (TE 13.1, TR 640; Gadovist®, gadobutrol 1.0 mmol/mL, 0.1 mL/kg IV). All transverse sequences were performed using a 4 mm-slice thickness.

The MRI scan revealed a large (1.25 x 0.75 cm in transverse cross-section and 1.35 cm in length), well-delineated, broad based, ovoid-shaped, extra-axial mass at the level of the left frontal and parietal lobes. The mass was mildly hyperintense on T2w and FLAIR, and isointense to the brain on T1w images. Mild hyperostosis of the adjacent parietal bone was present. The mass was surrounded by a large fluid accumulation in a presumed dilated subarachnoid space, causing mild dilation of a few cerebral sulci rostrally and moderate displacement of the adjacent brain parenchyma. The fluid surrounding the mass was hyperintense on T2w and FLAIR images, and hypointense to the brain but hyperintense to the cerebrospinal fluid (CSF) in the lateral ventricles on T1w images. After contrast administration, the mass showed marked homogeneous contrast enhancement, with meningeal enhancement and a small dural tail sign medially. Neither the fluid surrounding the mass, nor the presumed meningeal lining or wall of the fluid-filled cavity showed contrast uptake (Figure 1). There was a significant mass effect present with compression of the ventricular system, subfalcine, caudal transtentorial and foramen magnum herniation, and there was mild dilation of the cranial cervical central canal. The MRI findings were consistent with an extra-axial mass in the area of the left frontal and parietal lobes,

most likely representing a meningioma with a large peritumoral (pseudo)cyst.

As the MRI showed cerebellar herniation through the foramen magnum, a CSF sample could not be safely acquired. Prednisolone therapy (0.5 mg/kg once daily, orally) was started following the MRI scan. The patient showed mild improvement with the circling episodes becoming less frequent. Surgery was planned six days later.

On the day of the surgery, levetiracetam (20 mg/kg IV) was administered prior to anesthesia. The patient was premedicated with methadone (0.2 mg/kg IV); general anesthesia was induced with propofol (3 mg/kg IV) and midazolam (0.3 mg/kg IV) and maintained with sevoflurane (2%) vaporized in a combination of medical air and oxygen with an inspired oxygen fraction of 0.5. Mannitol (0.5 g/kg slowly IV) was given at the time of induction in order to reduce the intracranial pressure. A continuous rate infusion of alfentanil (0.4 mcg/kg/min IV) was given during the surgery for analgesia. A left-sided rostrotentorial craniectomy was performed. Before opening the dura mater, a needle was inserted to relieve some of the fluid and avoid a possible sudden drop in intracranial pressure. Macroscopically, the fluid had a similar appearance to CSF. Subsequently, a durotomy was performed and a large amount of fluid was released. The mass appeared grossly tan to yellow in color and was firm on palpation. It was removed as a single entity and a wall of the peritumoral (pseudo)cyst could not be identified. The surrounding dura mater and cerebral surface were macroscopically within normal limits. Minor hemorrhage occurred during surgery, which was successfully treated using hemostats (Surgicel®; ethicon SARM, Neuchatel, Switzerland and Surgiflo®; Ethicon, Somerville, New Jersey, USA). The surgical site was routinely closed using 3-0 polydioxanone (PDS plus®; Ethicon, Somerville, New Jersey, USA) for closure of the fascia of the temporal muscle and subcutaneous tissue. Staples were used for skin closure. Histopathology of the mass was consistent with a meningioma.

No anesthetic complications occurred and the patient made an uneventful recovery from anesthesia. The cat was neurologically normal immediately following surgery and analgesia was slowly tapered. Levetiracetam (20 mg/kg, three times daily) was continued for another two days postoperatively and the cat was discharged three days after surgery. The owner was contacted by telephone 14 months after the surgery and reported the cat to be clinically normal.

DISCUSSION

The sensitivity of MRI to correctly identify intracranial meningiomas has been estimated to be 96% in cats (Troxel et al., 2004). Meningiomas are typically reported to be well-defined, extra-axial, broad based masses that are T2w hyperintense and T1w iso- or hypo-

intense on MRI. They often show marked contrast enhancement and the presence of a dural tail sign. The contrast-uptake pattern may be homogeneous or heterogeneous and in some cases, hyperostosis of the overlying calvarium and cystic lesions or mineralizations within the mass may be present (Graham et al., 1998; Kitagawa et al., 2002; Troxel et al., 2004).

Cystic meningiomas are uncommonly reported in cats. In a study including 33 cats with meningiomas, only one cat (3%) was confirmed to have a cystic meningioma (Troxel et al., 2004). In dogs, more cases of cystic meningiomas have been reported (Bagley et al., 1996; Bagley et al., 2000; Kitagawa et al., 2002; Sturges et al., 2008; Ródenas et al., 2011; James et al., 2012). In a large study of 112 dogs, 26% of the meningiomas contained cystic lesions (Sturges et al., 2008).

The extra-axial mass detected on MRI in this patient was expected to be a meningioma based on the

typical MRI characteristics compatible with those reported in the literature; however, the large peritumoral (pseudo)cystic component was unusual. Cysts in canine meningiomas can occur intratumoral or peritumoral (Bagley et al., 1996; Bagley et al., 2000; Kitagawa et al., 2002; Sturges et al., 2008; James et al., 2012). In humans, cystic meningiomas have been classified according to the location of the cyst and the cyst content (Nauta et al., 1979; El-Fiki et al., 1996). Four different types have been described in the first classification system: in type 1, the cyst is an intratumoral cyst, in type 2, the cyst is located peripherally in the mass but within the tumor margins, in type 3, the cyst lies adjacent to the tumor but within the brain parenchyma, and in type 4, the cyst is actually a loculation of CSF in the subarachnoid space adjacent to the tumor (Nauta et al., 1979).

In the classification based on cyst content, also four

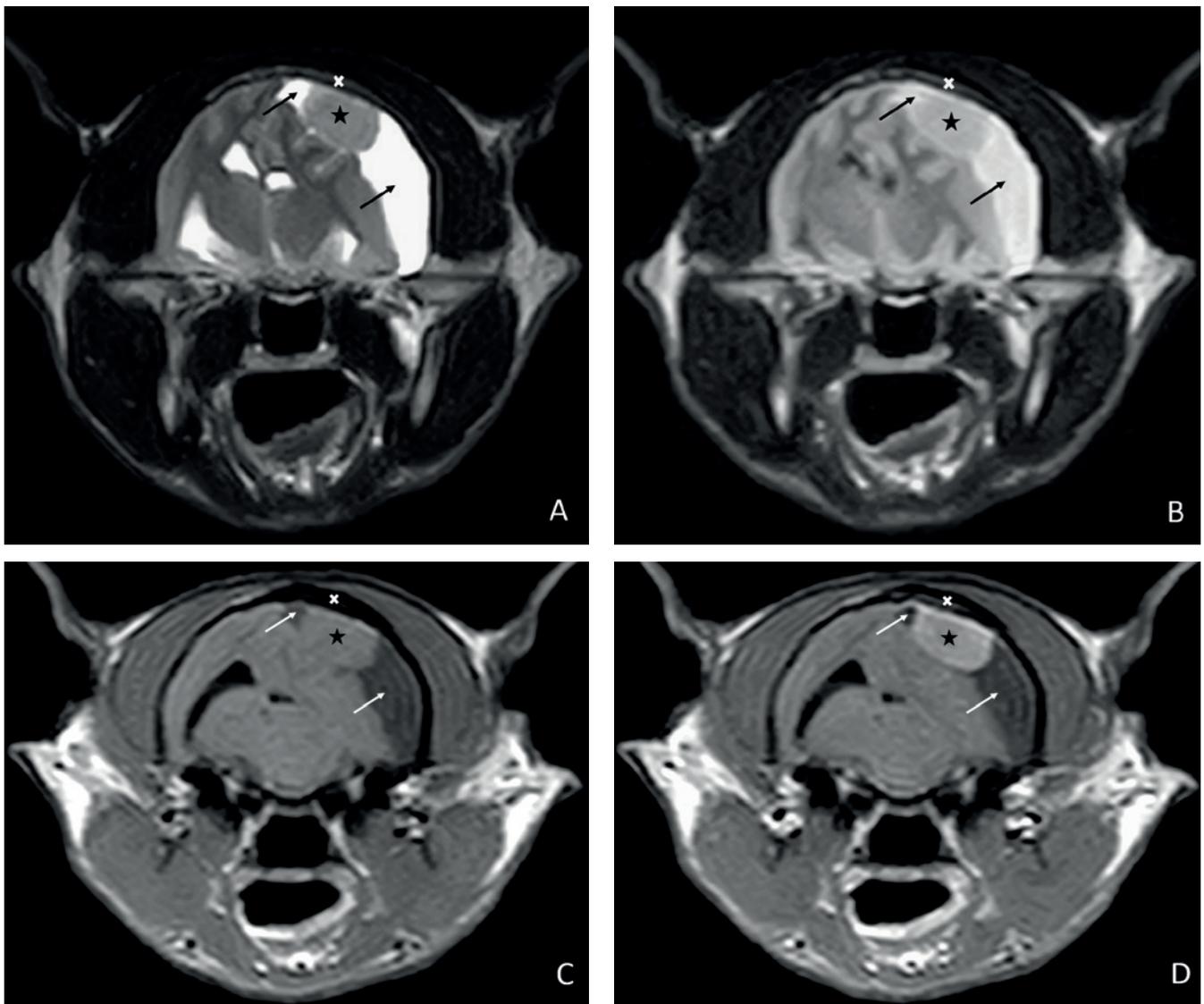


Figure 1. A. Transverse T2-weighted, B. FLAIR, C. T1-weighted and D. T1-weighted postcontrast magnetic resonance images. Images A and B are at a slightly different level than images C and D due to patient movement during the scan and repositioning that was required. Notice the large, mildly T2-weighted and FLAIR hyperintense and T1-weighted isointense, extra-axial mass with marked, uniform contrast enhancement at the level of the left parietal lobe (star), the large peritumoral (pseudo)cyst (arrows), which is hyperintense to the CSF on FLAIR and T1-weighted images, and hyperostosis of the parietal bone (cross).

types are described, of which the first two types are subdivided according to the location of the cyst (El-Fiki et al., 1996). In type A, the cyst contains clear, watery or CSF-like fluid and the cyst is located outside of the tumor. Type A is further subdivided in two categories: type A1, where entrapped CSF is surrounded by normal arachnoid membranes separating the cyst from the tumor and type A2, where clear cyst fluid is not surrounded by an arachnoid membrane separation and is likely resulting from tumor cell excretion or exudation. Also type B, where the cyst fluid is xanthochromic fluid, is subdivided in two categories; type B1 where the cyst is located extratumorally and no clearly defined wall is present, and type B2 where the cyst is located intratumorally and surrounded by a thin rim of enhancing tumor tissue. In type C, the cyst is located intratumorally and the cyst fluid is colored dark brown or dirty yellow. In type D, a combination of cysts is present, with both clear fluid containing extratumoral or peritumoral cysts and small intratumoral cysts containing a dark brown fluid.

El-Fiki et al. (1996) also reported a difference regarding the degree of peritumoral edema associated with cystic meningiomas. In their study, minimal peritumoral edema was typically present with clear fluid containing peritumoral cysts (type A), while moderate, variable (type B) to severe (type C) peritumoral edema was seen with xanthochromic fluid-filled peritumoral cysts and dark brown fluid-filled intratumoral cysts, respectively.

In the presented case, only the solid mass showed contrast uptake, with a small dural tail sign present, while the large peritumoral fluid accumulation showed no obvious capsule on the pre- and postcontrast images. Although the fluid was not loculated, it was presumably present within the subarachnoid space and was clear, CSF-like. This is consistent with a type-4 cyst according to the classification system of Nauta et al. (1979) and with type A1 or A2 in the classification of El-Fiki et al. (1996). Although moderate compression of the adjacent brain parenchyma by the large peritumoral (pseudo)cyst was present in this case, there was no evidence of brain edema. This is again compatible with a type-A cyst according to the classification of El-Fiki et al. (1996).

Although no histology of the surrounding tissues was performed, the peritumoral fluid accumulation in this case most likely did not show an epithelial lining and was therefore more likely to be a pseudocyst. Based on the MRI images, it was suspected that the fluid accumulation was actually a dilation of the subarachnoid space rather than an enclosed fluid-filled cavity. This raises the question whether the (pseudo)cyst could also be referred to as a subarachnoid diverticulum (Buetow et al., 1991; De Lahunta et al., 2015; Da Costa and Cook, 2016).

Although not in combination with a mass, intracranial and spinal (sub)arachnoid diverticula have been reported in cats (Milner et al., 1996; Galloway et al.,

1999; Vignoli et al., 1999; Schmidt et al., 2007; Lowrie et al., 2009; Adams et al., 2015; Taroni et al., 2015; Alcoverro et al., 2018). However, the lack of complete signal suppression on FLAIR and T1w images in this case suggests that this was not a simple CSF-containing subarachnoid diverticulum, and that the fluid accumulation was likely secondary to an inflammatory reaction, tumor fluid production, or compression of the subarachnoid space by the tumor (Worthington et al., 1985; Pinna et al., 1986; Zee et al., 1995).

The peritumoral (pseudo)cystic fluid in this case was hyperintense on T2w and hypointense on T1w images, consistent with a fluid nature, but was hyperintense on FLAIR and hyperintense to the CSF on T1w images. This has also been described in one of the dogs by James et al. (2012). In the canine cases described by Bagley et al. (1996, 2000), Kitagawa et al. (2002) and Sturges et al. (2008), FLAIR sequences were not performed. A lack of CSF signal suppression on FLAIR has been described before and can be caused by an increased total protein concentration of the CSF, hemorrhage, leptomeningeal metastasis, ischemic infarction, intracranial space-occupying lesions, a high fraction of inspired oxygen, or by artefacts such as the chemical shift artefact (Tha et al., 2009a; Tha et al., 2009b; Crawford et al., 2017; Moiola et al., 2017). Based on the MRI findings, the (pseudo)cyst fluid was considered to likely represent CSF. However, the lack of CSF and (pseudo)cyst fluid analysis is a limitation of this case report and the reason behind the hyperintense signal on FLAIR could therefore not be determined. Troxel et al. (2003) examined the CSF of 15 cats with meningioma. The median CSF total protein concentration in these cats was significantly increased while the median cell count was mildly increased (Troxel et al., 2003). Similar changes in this case could have been responsible for the altered appearance of the peritumoral fluid accumulation on FLAIR and T1w images (Cakirer et al., 2003).

There was no cyst wall nor contrast enhancement of a possible cyst wall visible on MRI. Neither could a cyst wall be identified during surgery, and the dura mater and sublesional cerebral surface were macroscopically normal. Only the solid contrast enhancing mass was therefore removed while the fluid from the peritumoral (pseudo)cyst was drained. El fiki et al. (1996) reported that there is no need to remove the cyst wall if it is not contrast enhancing and located outside of the tumor. Nevertheless, others have recommended surgical removal of the cyst wall despite a lack of contrast enhancement (Wang et al., 2016).

Another limitation of this case is that histopathology of the surrounding meninges was not performed. Similar cases have been described in dogs where only the tumor showed contrast uptake on MRI, but not the cyst wall (Bagley et al., 2000; Kitagawa et al., 2002; James et al., 2012). The lack of contrast uptake and removal of the cyst wall have also been described in

two dogs with cystic meningioma (Bagley et al., 2000; Kitagawa et al., 2002). Follow-up MRI was available for both of these cases. In the first case, follow-up MRI three months after surgery showed no recurrence and the dog was clinically doing well (Bagley et al., 2000). In the other case, a follow-up MRI was performed 77 days after surgery and showed contrast uptake at the level of the falx cerebri, suggesting residual or recurrent tumor tissue, even though the patient was clinically doing well. There was no recurrence of the cystic lesion reported (Kitagawa et al., 2002). Only in one dog with a cystic meningioma, histology showed foci of tumor cells bordering the area of the cyst. However, the type of cyst was not described and from the limited information and images available, the described tumor cells possibly represented the primary tumor itself rather than a type of cyst wall (Bagley et al., 1996).

Canine meningiomas are histologically very similar to their human counterparts. They are a heterogeneous group of tumors with different histological subtypes and grades (Sturges et al., 2008). In contrast to dogs and humans, feline meningiomas have a consistent and uniform histological pattern. Feline meningiomas are usually histologically benign and are most consistent with grade-1 tumors (Vandeveldt et al. 2012). Because of these differences between species, it has to be borne in mind that care should be taken when extrapolating information from one species to another.

Studies have shown that the median survival time in cats following meningioma resection is between 22 and 37 months (Gallagher et al., 1993; Gordon et al., 1994; Troxel et al., 2003; Cameron et al. 2015). Although in this case, follow-up was limited to 14 months postsurgery, the cat had a good outcome, with absence of clinical signs. However, the follow-up was only done via a telephone conversation with the owner, lacking a complete follow-up neurological examination and repeat MRI scan. To this day, recurrence of the meningioma can therefore not be excluded.

This is the first case report including MRI imaging of a peritumoral (pseudo)cystic meningioma in a cat. With this case, the need for a classification system for cystic meningiomas in cats and dogs is highlighted, with guidelines for surgery, as more and more pets are referred for further diagnostic work-up and surgery of brain tumors.

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