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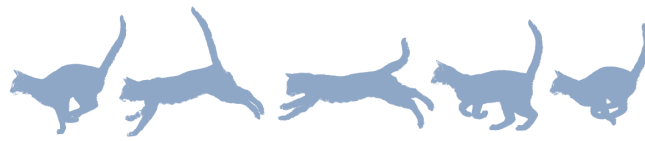
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Symptomatic lateral ventricular cystic lesion in a young cat

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Abstract

Case summary A 1.5-year-old male neutered Persian cat was referred for acute deterioration of chronic left head tilt and ataxia. A lateral intraventricular cystic lesion, closely associated with the left choroid plexus, was identified on MRI. The intralesional signal intensity and cytological analysis of the fluid revealed a liquid similar to cerebrospinal fluid. After trepanation, an endoscopic-assisted fenestration and aspiration of the cyst were performed to temporally relieve the high intracranial pressure while waiting for surgical cystoperitoneal shunt placement. Three weeks after surgery, clinical relapse and recurrence of the lesion were noted on the pre-cystoperitoneal shunting MRI. During anaesthesia, the cat arrested. Cardiac resuscitation was successfully performed and cystoperitoneal shunting was postponed. Global brain ischaemia was then suspected, based on major forebrain clinical signs and MRI abnormalities. During a 6-month recovery period, a further three fine-needle CT-guided aspirations of the lesion were required, owing to clinical recurrence and increased cyst size. Cystoperitoneal shunting was eventually performed, allowing persistent reduction of the lesion and long-term improvement of the cat's neurological status.

Relevance and novel information This is the first report of a symptomatic lateral intraventricular cystic lesion in a cat. A left lateral intraventricular choroid plexus cyst was suspected based on the MRI features. Our case suggests that endoscopic fenestration and CT-guided aspiration are not adequate treatments for long-term management. Cystoperitoneal shunting may be a safe procedure, allowing significant and stable reduction of the cystic lesion, associated with improvement in the cat's neurological status by preventing high intracranial pressure.

Keywords: Vestibular; intracranial; intraventricular; choroid plexus; cyst; cystoperitoneal shunt; lateral ventricle

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Introduction

Non-neoplastic intraventricular cystic lesions are rarely described in veterinary medicine and no details exists on their MRI appearance or therapeutic management. Based on human classification considering only cerebrospinal fluid (CSF)-filled lesions, congenital midline cysts, arachnoid, choroid plexus or ependymal cysts can be considered.¹ None has been described in cats. Herein, we describe a case of a symptomatic lateral intraventricular cystic lesion, suspected to be a choroid plexus cyst, successfully treated with a permanent cystoperitoneal shunting.

Case description

A 1.5-year-old male neutered Persian cat was referred with a 3-month history of waxing and waning left head

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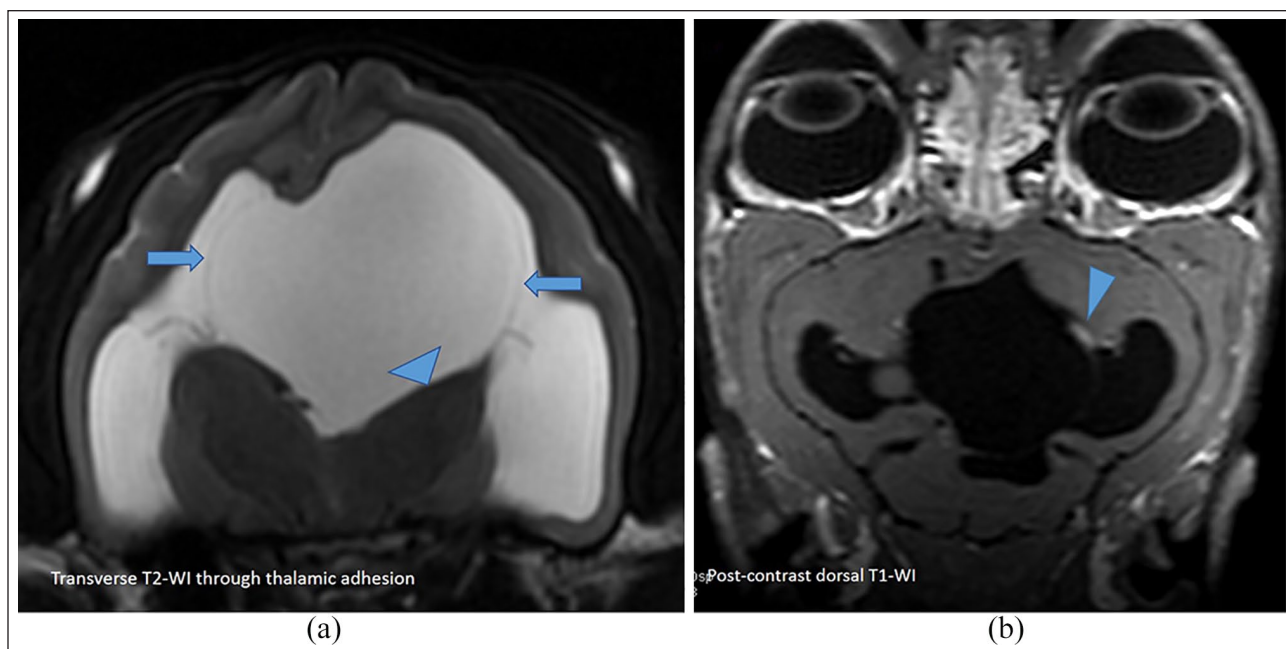


Figure 1 Initial MRI. (a) Large homogeneous hyperintense asymmetric cystic lesion in the lateral ventricles, delineated by a thin wall (arrows). Severe dorsal left thalamic compression (arrowhead), generalised cortical thinning, effacement of the cerebral sulci and subarachnoid space are observed. (b) The cystic lesion appears to be closely related to the left choroid plexus (arrowhead). T2-WI = T2-weighted image; T1-WI = T1-weighted image

tilt and ataxia with acute deterioration over the past 3 days. Improvement was noted following corticosteroid therapy (prednisolone 0.5 mg/kg q12h PO). The cat had been rehomed 1 year previously, had no history of neurological signs initially and was up to date on vaccinations. There was no history of trauma or exposure to toxins. Physical examination was unremarkable. Neurological examination revealed moderate decreased mentation, left vestibular ataxia and head tilt, and delayed postural reactions (hopping responses and tactile placing reactions) on the left side with normal spinal reflexes. These signs were consistent with a left vestibular syndrome. A central origin was considered, given the left-sided proprioceptive deficit and altered mentation. A left brainstem lesion (including vestibular nuclei) or, less likely, a thalamic lesion (including medial geniculate nuclei), were suspected based on the neurological signs. Differentials included an inflammatory or infectious process or, less likely, a neoplasm or a progressive congenital disease.

MRI of the brain was performed using a high-field scanner (1.5-T magnet; Signa HD23 optima 1.5T [General Electric Healthcare]). The protocol included T2-weighted (T2W), T1-weighted (T1W), fast-spin echo (FSE) sequences, T2W fluid-attenuated inversion recovery (FLAIR), susceptibility-weighted magnetic resonance sequence (SWAN sequence), diffusion-weighted imaging, and pre- and post-contrast three-dimensional FSE (cube) T1 sequences. Gadoteric acid contrast medium

intravenous injection was performed (0.1 mmol/kg). All intensities were compared with normal grey matter. A large, well-demarcated lesion was observed within the lateral ventricles, closely associated with the left choroid plexus. Measured dimensions were $27.7 \times 29.1 \times 24.3$ mm (length \times width \times height) (Figure 1). The intralesional signal intensity was uniformly hyperintense on T2W and hypointense on T2-FLAIR and T1W images, similar to CSF. This lesion was asymmetric, delimited by a thin wall and extended caudally, causing secondary non-communicating hypertensive hydrocephalus with dilation of the lateral ventricles, causing left thalamic, brainstem and cerebellar compressions. Minimal wall cyst contrast enhancement was noted on the T1W post-contrast images. Magnetic resonance signs of increased intracranial pressure were present, with caudotentorial brain herniation and severe cerebellar foraminal herniation. Generalised thinning of the cerebral cortex and effacement of the cerebral sulci and subarachnoid space were also observed. Differential diagnoses based on the magnetic resonance characteristics of the lesion included choroid plexus cyst, arachnoid diverticula or ependymal cyst.

Following MRI study, left rostral tentorial trepanation and transparenchymal endoscopic-assisted fenestration and aspiration of the lesion were performed, to temporally relieve the neurological signs associated with high intracranial pressure, prior to cystoperitoneal shunting. The intralesional fluid collected was similar to normal CSF; biopsy of the lesion wall was attempted

but was not successful. Corticosteroid therapy was initiated (prednisolone 0.5 mg/kg body weight PO q12h). Progressive clinical improvement was noted over the 3 days following the drainage of the lesion. On the 2 days post-decompression MRI, marked decreased size of the lesion, apparent subarachnoid CSF and absence of brain herniation were observed (Figure 2).

Three weeks post-decompression, relapse of the initial clinical signs was observed and lesion recurrence was

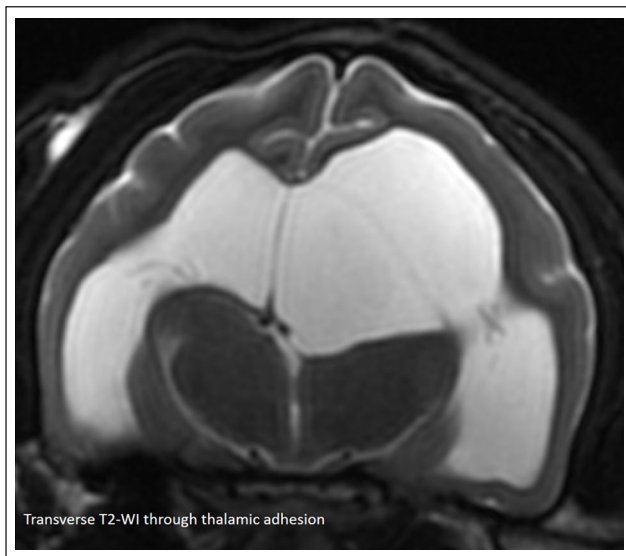


Figure 2 Two days post-trepanation control MRI. The cyst appears decreased compared with Figure 1a. Subarachnoid cerebrospinal fluid is visible and brain herniations were no longer observed (data not shown). T2-WI = T2-weighted image

noted on the pre-cystoperitoneal shunting MRI. General anaesthesia was complicated by cardiac arrest. Successful cardiopulmonary resuscitation (CPR) and a CT-guided aspiration of the cyst, through the trepanation hole, were performed. Cystoperitoneal shunting surgery was postponed. Major forebrain clinical signs were observed. MRI was repeated 10 days post-CPR, demonstrating recurrence of the cystic lesion, diffuse cortical and thalamic T2W and T2-FLAIR hyperintensity of the grey matter and heterogeneous contrast enhancement (Figure 3). Based on these abnormalities and the clinical signs, global brain ischaemia was suspected.

The cat slowly improved over the next 6 months. During this period, three fine-needle CT-guided aspirations of the lesion were performed through the initial craniectomy defect, because of the recurrence of clinical signs associated with increased cyst size, while waiting for the cat to recover and be able to withstand surgical cystoperitoneal shunting. The cat remained bilaterally blind but regained good levels of consciousness, urinary and faecal continence, and motility. Quality of life was judged to be acceptable for the owners and the medical team in charge.

Successful surgical placement of a cystoperitoneal shunt in the cystic intraventricular lesion was eventually performed. Repeat MRI, 4 days after surgery, revealed a marked reduction of the cyst and adequate placement of the cystoperitoneal shunting device (Figure 4). The cat's neurological status improved shortly after surgery and had been stable overtime, 7 months post-shunting. The size and appearance of the cystic lesion were unchanged on follow-up MRIs, 2 and 7 months postoperatively.

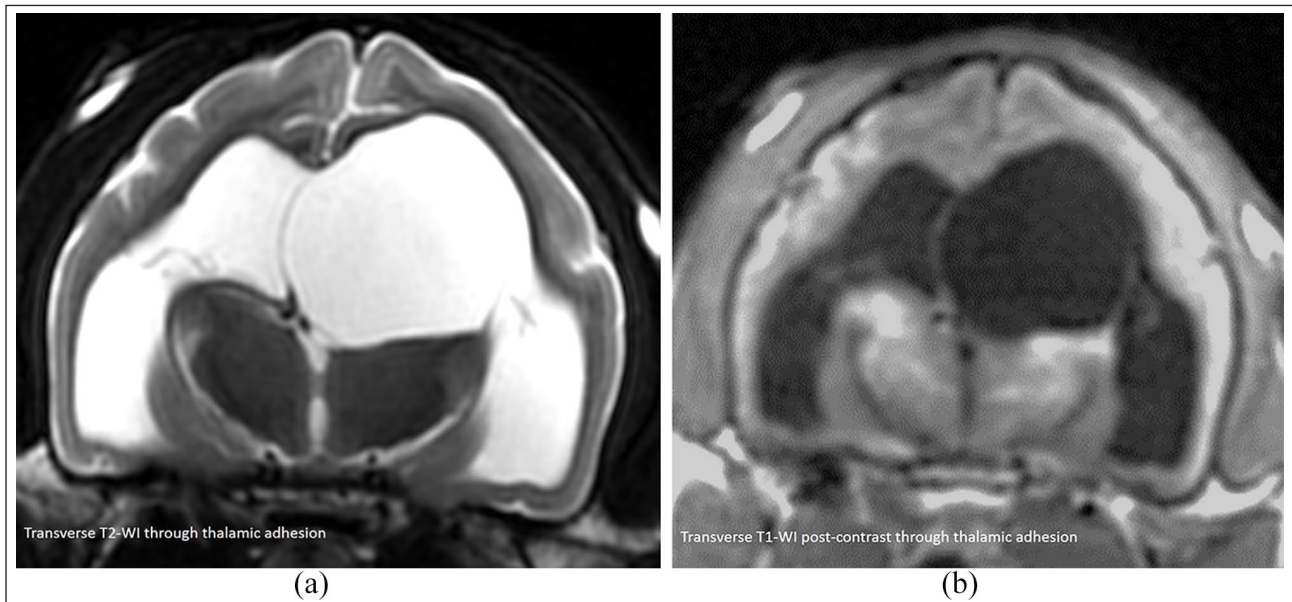


Figure 3 Ten days post-cardiorespiratory arrest. (a) Marked enlargement of the cyst, compared with Figure 2, showing recurrence of the lesion. Diffuse cortical and thalamic T2-weighted hyperintensity of the grey matter, compatible with global brain ischaemia. (b) Diffuse post-contrast signal enhancement of the cortical and thalamic grey matter. T2-WI = T2-weighted image; T1-WI = T1-weighted image

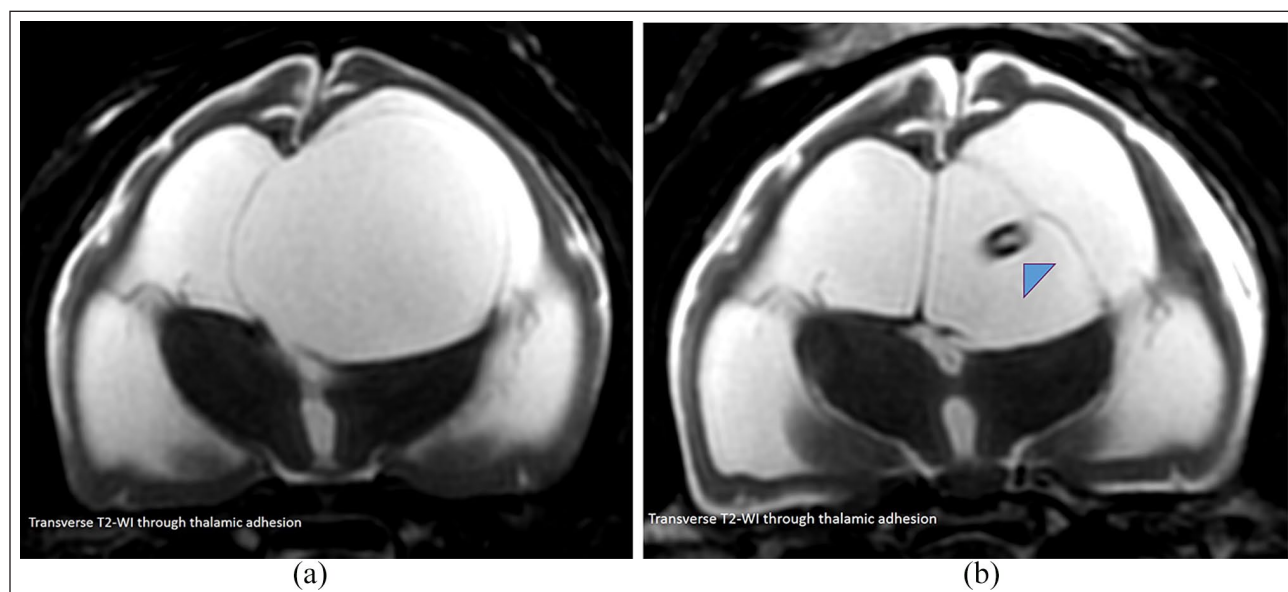


Figure 4 (a) Pre-cystoperitoneal shunting MRI. Increased size of the cystic lesion, 1 month after the last CT-guided aspiration. The subarachnoid cerebrospinal fluid is visible contrary to the initial diagnosis, although the cyst size is similar. This may be due to cortical atrophy secondary to the global brain ischaemia. (b) Post-cystoperitoneal shunting MRI. The shunt tip is visible inside the cystic lesion (arrowhead) and a significant reduction of the cyst is observed with widening of the subarachnoid space

Discussion

Strictly defined, a cyst is an epithelial-lined structure filled with fluid.¹ The term 'cyst' can be misleading as some lesions are not delineated by a wall, and should therefore be identified as cyst-like lesions, pseudocysts or diverticula. Because of this confusion, and frequent lack of definitive histological diagnosis, the classification of intracranial cystic lesions in the literature is not consistent, especially regarding intraventricular lesions. In cats, the main differential diagnoses for intracranial cyst-like lesions are parasitic cysts (cerebral coenurosis or cysticercosis),^{2,3} arachnoid cysts⁴⁻⁷ and abscesses.⁸⁻¹⁰ Cystic neoplasms or other congenital and developmental cystic lesions (hydranencephaly and porencephaly) are very rarely described.^{8,11,12} All cases have been intra-axial or quadrigeminal, except one intraventricular parasitic cystic lesion.³ Although the MRI features of the current case could be compatible with a parasitic cyst,² this hypothesis was excluded due to the cat living only indoors, the chronic history of the case and the absence of parasite detection in the cystic fluid.

To our knowledge, this is the first description of a lateral intraventricular cystic lesion in a cat. Intraventricular cystic lesions are infrequently reported in domestic animals,¹³⁻¹⁷ and real consensus classification is lacking. In the human literature, these lesions are mainly non-neoplastic: dermoid, epidermoid, ependymal, neuroendodermal, choroid plexus, colloid or arachnoid cysts, and congenital midline cysts.^{18,19} When only considering CSF-filled lesions, the list of differential diagnoses should be restricted to congenital midline cysts, arachnoid cysts,

choroid plexus or ependymal cysts.²⁰ A congenital midline cyst was considered unlikely in our case, as it is usually triangular, circular or elliptical interhemispheric.¹⁸ Based on the asymmetric CSF-filled cystic lesion in our case, an arachnoid cyst, or an ependymal or choroid plexus cyst were favoured hypotheses.

Choroid plexus cysts are rare in humans,²¹ and have only been reported in three canine cases, to our knowledge, with only two of them being intraventricular in the fourth ventricle. In humans, they are most commonly located in the lateral ventricles and are often incidental findings and asymptomatic.²¹ Depending on their size and location, they can cause hypothalamic dysfunction and obstructive hydrocephalus by compromising CSF flow in the ventricular system and prolapsing through the inter-ventricular foramen, as was observed in this cat.^{14,21} Cystic content usually appears similar to CSF on human MRI, although FLAIR suppression may be incomplete because of elevated protein concentration or gelatinous characteristics within the cyst.^{1,14} Variable contrast enhancement is described. In the two cases published in the veterinary literature with MRI descriptions,^{14,22} one showed strong homogeneous contrast enhancement of the lesion and the second only weak contrast enhancement of the membranous tissue in the cystic lesion. Contrast enhancement may be related to vascularity of the cysts, but the cause of homogeneous enhancement is not fully understood.¹ In our case, MRI follow-up demonstrated recurrence in expansion of the cystic lesion during the period between diagnosis and the cystoperitoneal shunting, despite multiple fine-needle aspirations of the cyst. Furthermore, the

absence of neurological signs at rehoming and the progressive waxing and waning clinical course demonstrate the progressive nature of this lesion. The ongoing enlargement of the cyst may have resulted from active fluid secretion from choroid plexus tissue, as was suspected in a previous case.²² Several theories have been formulated about their origin, and most authors suggest an embryogenetic origin,²¹ even if some cases may have been secondary to head trauma or following intraventricular shunting.¹⁴ In our cat, an embryogenetic origin is suspected as no head trauma or shunting occurred.

Owing to the lack of histological analysis, an intraventricular arachnoid cyst or an ependymal cyst could not be excluded. Intracranial arachnoid cysts are collections of CSF contained by a thin wall of arachnoid cells and collagen. However, because cases may not have evidence of complete epithelial lining, the lesions are more appropriately termed diverticula.^{1,23} The majority are congenital, and a splitting or duplication of the arachnoid membrane during embryonic development can be considered.¹ Because of the intra-arachnoid nature of the fluid accumulation, these lesions may not communicate freely with the subarachnoid space or the ventricular system. In cats, none of the published cases is intraventricular. In veterinary medicine, most of them are located in the caudal fossa, in a region comparable with the quadrigeminal cistern.^{1,4,17,24} In human medicine, intraventricular arachnoid cysts are rare entities, and only concern 0.3% of the arachnoid cysts diagnosed in children.²² Progressive enlargement has been infrequently reported in human studies.²² The osmotic gradient between the intra- and extracystic medium, and fluid hypersecretion by the lining cells of the cyst wall, were suggested.²³

Finally, no description of feline ependymal cysts has been published previously and only two cases are reported in dogs,^{25,26} including one cerebellar, in the fourth ventricle,²⁵ and one suprapituitary in the third ventricle for the more recent case.²⁶ In humans, they are rare and often located deep within brain parenchyma, in the central white matter, or are juxtaventricular.⁸ The origin of these cysts is also controversial, but an embryological abnormality is suspected subsequent to sequestration of developing neuroectoderm. In our case, the continuity of the cyst and the choroid plexus makes an ependymal cyst less probable.

The main limitation of this report was the lack of definitive diagnosis with histopathological and immunohistochemical studies. Indeed, biopsies of the cystic wall during the endoscopic procedure were unsuccessful, as the cystic wall collapsed when introducing the endoscopic device. However, according to the close relation with the left lateral choroid plexus and the presumed continuous CSF secretion, a left lateral choroid plexus cyst was highly suspected in our case.

Owing to the paucity of cases, no treatment consensus exists in veterinary medicine for the management of choroid plexus cysts or even for intracranial cystic lesions. In humans, even if conservative clinical management with MRI follow-up is adequate for asymptomatic brain cysts, no consensus exists for benign symptomatic cystic lesions.¹⁹ Various therapeutic procedures have been described, such as stereotactic or endoscopic aspiration (ipsi- or contralateral transcortical), placement of an internal cystosubarachnoid/cystoventricular or cystoperitoneal shunt, craniotomy with cyst resection or open fenestration into subarachnoid space or lateral ventricle.¹⁹ Aspiration, and even fenestration, usually only allow temporary relief of clinical signs and are not an adequate long-term definitive treatment, as seen in our case. In veterinary medicine, two feline quadrigeminal cysts have been successfully treated with cystoperitoneal shunt.^{4,5} In dogs, surgical resection has been described for intracranial cystic lesions,^{14,17,22,25–27} but a cystoperitoneal shunt also appears to be an effective treatment method.²⁸ Among canine cases of intracranial cystic lesions, two cases of choroid plexus cysts have been surgically treated, with no recurrence documented after follow-ups of 3 and 18 months, follow-up.^{15,22} However, local recurrence of ependymal or neuroendodermal cysts were reported after incomplete surgical excision.^{15,26} Therapeutic decisions must be based on cystic location or accessibility, but permanent procedures should be promoted.

Conclusions

To our knowledge, this is the first case of a symptomatic intraventricular cystic lesion in a cat. A choroid plexus cyst was considered the main diagnosis, with findings similar to human literature and MRI features. This case illustrates that endoscopic fenestration and CT-guided drainage of an intraventricular cystic lesion are not therapeutic options for adequate long-term management but can be used for temporary relief of the high intracranial pressure and risk of death. Permanent shunting should be considered whenever possible, and can be linked to a good prognosis, allowing for long-term control of cyst size.

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Conflict of interest The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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Ethical approval This work involved the use of non-experimental animals only (including owned or unowned animals and data from prospective or retrospective studies). Established internationally recognised high standards ('best practice') of individual veterinary clinical patient care were followed. Ethical approval from a committee was therefore not necessarily required.

Informed consent Informed consent (either verbal or written) was obtained from the owner or legal custodian of all animal(s) described in this work (either experimental or non-experimental animals) for the procedure(s) undertaken (either prospective or retrospective studies). For any animals or humans individually identifiable within this publication, informed consent (either verbal or written) for their use in the publication was obtained from the people involved.

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