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Case Report

Neuroendoscopic cyst fenestration for delayed enlargement of perianeurysmal cyst formation through long-term follow-up after endovascular treatment: A case report and review of literature

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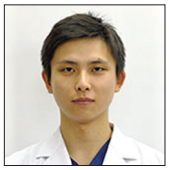

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ABSTRACT

Background: Perianeurysmal cyst formation after endovascular treatment of cerebral aneurysms is a rare complication; however, the number of reports has gradually increased in recent years due to the development of several endovascular treatments.

Case Description: We present a case of delayed perianeurysmal cyst enlargement 8 years after endovascular treatment for multiple recurrences of a large cerebral aneurysm in the anterior communicating artery. The patient presented with obstructive hydrocephalus caused by an enlarged perianeurysmal cyst. The patient underwent cyst fenestration using neuroendoscopy and ventriculoperitoneal shunting, recovered from the clinical symptoms, and had a good prognosis. Histopathological findings showed that the cyst wall contained a fibrotic layer under the monoependymal layer with hemosiderosis without evidence of neovascularization or inflammatory cell infiltration. These findings suggest that the origin of the perianeurysmal cyst wall is not the aneurysm itself but the adjacent brain tissue.

Conclusion: Perianeurysmal cysts can develop during long-term follow-up, and clinicians should consider surgical treatment, including cyst fenestration, using neuro-endoscopy if the cyst presents with clinical symptoms.

Keywords: Complication, Endovascular treatment, Hydrocephalus, Intracranial aneurysm, Neuroendoscopy, Perianeurysmal cyst

INTRODUCTION

Coil embolization has been the standard treatment for ruptured and unruptured cerebral aneurysms since the advent of the detachable coil system in the 1990s.[9] Randomized clinical trials have demonstrated the efficacy and safety of this treatment;[16,17] however, clinicians are well aware of several unique complications, such as thromboembolism, perforation, catheter-induced arterial vasospasm or dissection, and coil migration.[5] Although perianeurysmal cyst formation associated with ruptured or unruptured cerebral aneurysms is rare, the number of such cases has gradually increased in recent years. Several cases of perianeurysmal cyst formation after

coil embolization have been reported. Perianeurysmal cysts can present with various clinical symptoms, resulting in an increase in the cyst size and requiring additional treatment if these clinical symptoms progress. Although the number of reports of perianeurysmal cysts after coil treatment has increased in recent years, the pathogenesis and management of these cysts remain unclear. Here, we describe a case of delayed obstructive hydrocephalus due to a perianeurysmal cyst that developed several years after undergoing multiple endovascular treatments and discusses the pathogenesis and management of this complication.

CASE DESCRIPTION

A 64-year-old man was carried to our hospital, presenting with a sudden headache and severe disturbance of consciousness. Computed tomography revealed a subarachnoid hemorrhage in the interhemispheric and bilateral Sylvian fissures. Cerebral angiography revealed a large aneurysm in the anterior communicating artery (Acom A). Subsequently, we treated the aneurysm with endovascular embolization using a simple coiling technique (Guglielmi Detachable Coil -18 360 [Boston Scientific, Fremont, California, USA] and Target 360 standard [Stryker, Kalamazoo, MI, USA]). Due to the risk of parent artery occlusion, a small neck remnant remained [Figures 1a-c]. After several months of rehabilitation, the patient was discharged without any neurological deficits

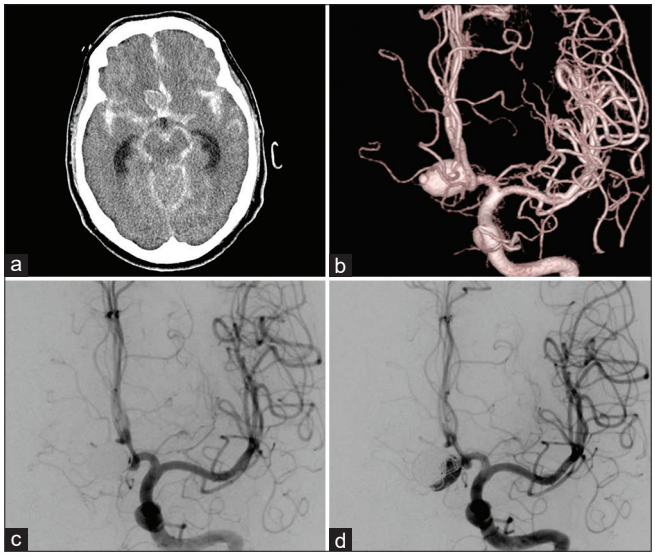

Figure 1: (a and b) Initial computed tomography (CT) shows severe subarachnoid hemorrhage with a mass lesion in the interhemispheric fissure, and CT angiography shows a large ruptured cerebral aneurysm in the anterior communicating artery. (c) Postoperative cerebral angiography reveals that endovascular treatment achieved adequate coiling with a small neck remnant. (d) Follow-up cerebral angiography 27 months after initial treatment shows recurrence of aneurysm and enlargement of neck remnant.

or slight memory disturbances. However, additional endovascular treatments for the enlarged neck remnant were required at 27 and 35 months after the initial treatment [Figure 1d]. The first additional treatment underwent using a simple coiling technique (Target XL 360 Standard [Stryker], Target 360 Soft [Stryker], DELTAMAXX 18 CERECYTE [Codman and Shurtleff, Johnson and Johnson, Raynham, MA, USA], Axium Prime 3D [Medtronic, Minneapolis, MN, USA], and ED Coil-10 Extra Soft [Kaneka Medix Corporation, Osaka, Japan]). After that, the second additional endovascular treatment underwent using a stent-assisted coil technique (HydroFrame 10 [MicroVention Terumo, Tustin, CA, USA], ED coil Complex [Kaneka Medix Corporation], Axium Prime ES [Medtronic], and Enterprise 2 VAD [Codman Neuroendovascular, Johnson and Johnson, Miami, FL, USA]), and complete occlusion of the aneurysm was achieved. Finally, the patient returned to his normal life. Careful follow-up was continued, and the occluded aneurysm remained stable with no recurrence. During this treatment course, a perianeurysmal cyst developed immediately above the aneurysm without any additional clinical symptoms 24 months after the initial treatment [Figures 2a and b]. However, the perianeurysmal cysts enlarged markedly after the second coil embolization, and pericystic edema gradually progressed over time. The patient presented with progressive memory disturbance and perianeurysmal cyst enlargement and was admitted to our hospital 8 years after the initial treatment. Cerebral angiography revealed no blood flow from the cerebral aneurysm to the perianeurysmal cyst [Figure 2c]. Magnetic resonance imaging (MRI) revealed an enlarged perianeurysmal cyst occluding the foramen of Monro, and parenchymal edema was observed on fluid-attenuated inversion recovery imaging [Figures 2d and e]. Due to the progression of clinical symptoms, the patient underwent endoscopic cyst fenestration. Based on the preoperative MRI features, high protein concentration in the cyst fluid was suspected, which may have led to postoperative cyst wall adhesions and subsequent obstructive hydrocephalus recurrence. Furthermore, the patient declined the potential risk of additional surgery in cases of obstructive hydrocephalus recurrence, and additional ventriculoperitoneal shunting was performed simultaneously. Intraoperatively, the cyst wall obstructed the foramen of Monro from the anterior side, and a branch of the anterior septal vein was observed on the cyst wall. Initially, we attempted to perforate the cyst wall using a balloon catheter. However, the cyst wall was tight, and the catheter slipped across its surface of the cyst wall. We then coagulated the upper side of the cyst wall to avoid the vein and gently fenestrated the cyst wall using biopsy forceps. When the cyst wall was fenestrated, yellowish fluid was drained from the cyst. Inside the cyst, a coiled aneurysm and optic nerves were observed. The aneurysm wall was thin, and the internal

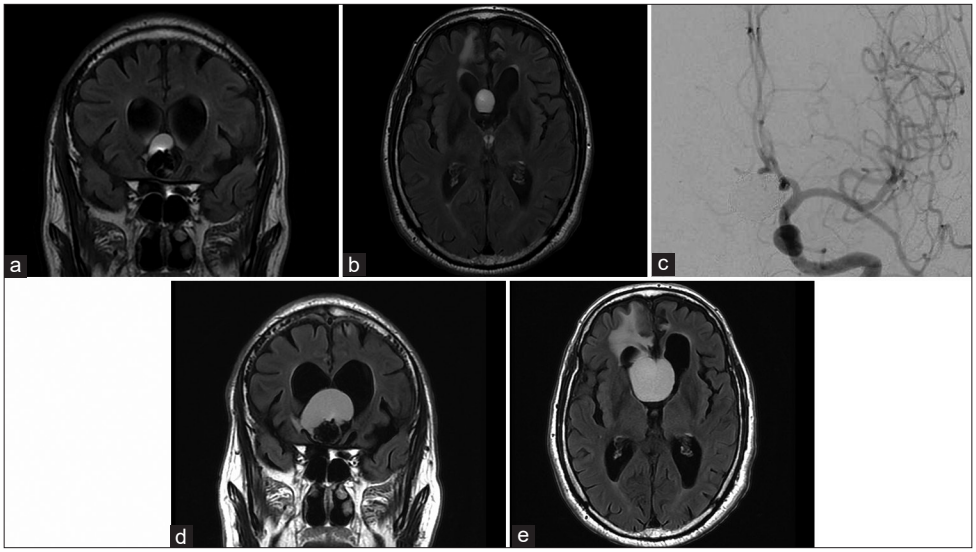

Figure 2: (a and b) Magnetic resonance image (MRI) 24 months after the initial treatment shows that the cystic lesion developed from just above the coiled aneurysm (a and b). (c) Cerebral angiography reveals that there is no blood flow into the perianeurysmal cyst and no recurrence of cerebral aneurysm. (d and e) MRI just before endoscopic treatment shows that an enlarged perianeurysmal cyst led to obstructive hydrocephalus and progressive parenchymal edema.

coil was visible. Finally, in the same manner, two points of fenestration were achieved, avoiding the wall vein and confirming the patency of the Monro foramen [Figures 3a-d]. Histopathological examination revealed that the cyst wall was composed of eosinophilic fibrous tissue covered with a monolayer of ependymal cells, and no neovascularization was observed within the cyst wall [Figures 4a-d]. After treatment, the patient recovered from the obstructive hydrocephalus and returned to normal life. At the 18-month follow-up, there was no significant recurrence of the perianeurysmal cysts or obstructive hydrocephalus [Figure 5].

DISCUSSION

The perianeurysmal cyst is defined as "a structure with signal intensity attenuation characteristics compatible with fluid that lay within the parenchyma adjacent to the aneurysm, and that did not communicate with the ventricles, cisterns, or subarachnoid space."[19] Perianeurysmal cyst formation associated with ruptured or unruptured cerebral aneurysms is extremely rare, with only 22 cases reported in the literature [Table 1].[1-3,6-8,10-14,18-20] Most cases occurred in the elderly average 61.3 years and were slightly more common in men (male: female, 14:8). Most cases were associated with aneurysms in the anterior circulation (internal carotid artery, five cases; middle cerebral artery, five cases; and Acom A, five cases); however, they also occurred in the posterior circulation (posterior cerebral artery, three cases; basilar artery, two cases; and vertebral artery-posterior inferior cerebellar artery, one case). As previously reported, perianeurysmal cysts are not always associated with subarachnoid hemorrhage (seven of 22 cases); however, aneurysms are often large or giant and approximately half of these are associated with thrombosis. The cysts were often large, with a median size of 25.0 mm. The most common clinical symptom is headache, which is associated with increased intracranial pressure. Hydrocephalus and hemiplegia have been observed in several cases, including asymptomatic cases. Notably, ten patients had a history of endovascular treatment for cerebral aneurysms before being diagnosed with perianeurysmal cysts, as in the present case.[1,5,6,10,12-14,18] In addition, nine of these cases showed aneurysm recurrence after endovascular treatment at the time of cyst appearance or during the disease. This suggests that the development of perianeurysmal cysts may be influenced by the endovascular treatment of the aneurysm itself or by changes within the aneurysm, such as thrombosis and recurrence after treatment. In most cases, the duration from endovascular treatment to perianeurysmal cyst diagnosis is relatively long, and the cysts may take time to develop. In the present case, a perianeurysmal cyst was diagnosed 2 years after the initial treatment and gradually enlarged through multiple treatments for recurrent cerebral aneurysm over another 6 years; subsequently, the enlarged cyst caused obstructive hydrocephalus. Several months or years may be needed to develop a perianeurysmal cyst after an aneurysm treatment; therefore, long-term follow-up is important for the diagnosis and treatment of perianeurysmal cysts.

The pathogenesis of perianeurysmal cysts remains unclear. Cyst formation associated with vascular lesions, such as arteriovenous and cavernous malformations, repeated

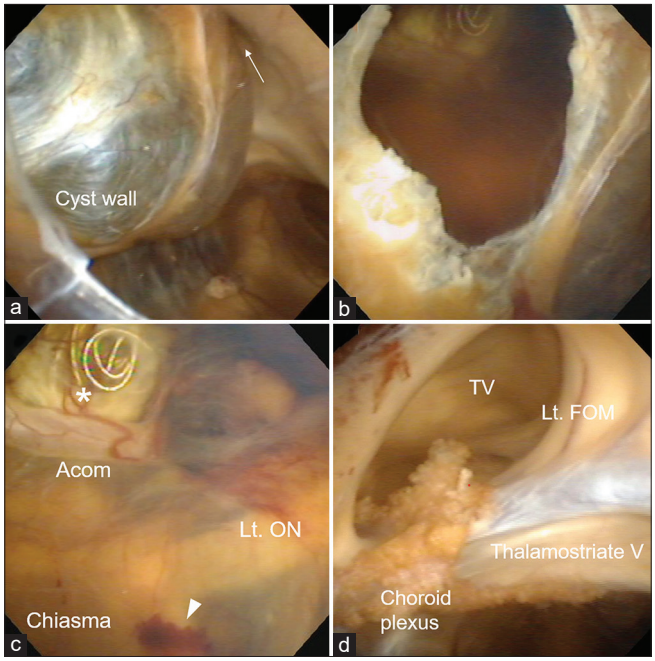

Figure 3: (a) Intraoperative findings. The lesion is revealed through an endoscopic approach from the left anterior horn. The cyst wall was composed of a tight and fibrous layer and obscured the foramen of Monro (white arrow). (b) The fibrous cyst wall is widely opened. A coiled cerebral aneurysm (asterisk), optic chiasma, and left optic nerve are observed from the inside of the perianeurysmal cyst. (c) Hemosiderosis is often seen in the cyst wall (white arrowhead). (d) After cyst fenestration, the left foramen of the monro was opened, and the third ventricle was preserved under the cyst. Acom: Anterior communicating artery, ON: Optic nerve, TV: Third ventricle, FOM: Foramen of Monro, V: vein.

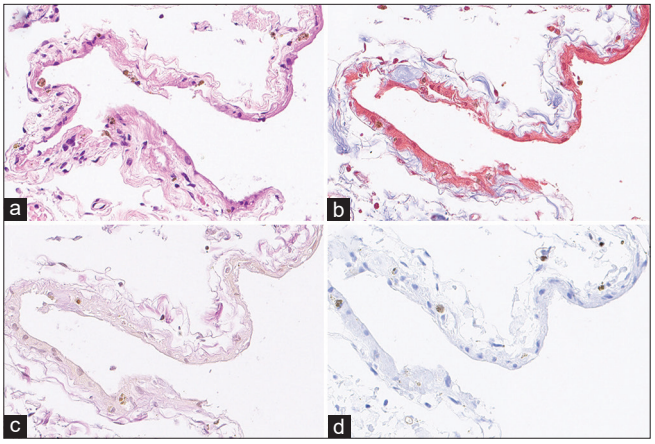

Figure 4: Histopathological findings. (a) Hematoxylin and eosin (HE) staining shows that the cyst wall is focally covered by monolayered flat or columnar epithelium, associated with aggregates of siderophage material. There is no inflammatory cell infiltration into the cyst wall. (b) Immunohistochemistry and Masson-trichrome staining show fibrous changes under the ependymal layer. (c) No blood vessels are detected in the Elastica van Gieson stain. (d) The cyst wall is also positive for Glial fibrillary acidic protein.

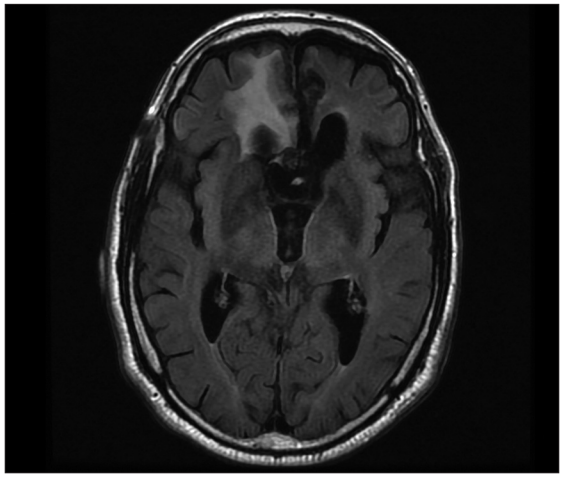

Figure 5: Follow-up MRI 18 months after treatment shows no recurrence of the perianeurysmal cyst and improvement of parenchymal edema.

subclinical or clinical hemorrhage or exudation from the aneurysmal wall, and/or intermittent bleeding or exudation from the neovascular system of the cyst wall, are suggested mechanisms of cyst formation.[1,2] A similar mechanism has been suggested for perianeurysmal cysts, including encephalomalacia associated with local ischemia and bleeding of the surrounding brain parenchyma and increased inflammation associated with thrombosis of the aneurysm, which causes exudation through the aneurysmal wall have been suggested.[1,2,8,11,14] These hypotheses are supported by previous reports on hemosiderin deposition in nonbleeding cases where the cyst content had high protein levels. Moreover, in recent reports of perianeurysmal cysts after endovascular treatment, increasing reactive inflammation of the aneurysmal wall and increasing brain compliance after endovascular treatment lead to cystic encephalomalacia due to direct pressure and pulsatile from the postcoil embolization aneurysm to the adjacent brain tissue are also suggestive mechanisms.[2,8,10] Furthermore, a previous report has suggested an association between cyst formation and hydrogel-coated coils.[1] It is noteworthy that in cases of perianeurysmal cysts following endovascular treatment of aneurysms, most postendovascular cerebral aneurysms recur, requiring additional treatment. Moreover, local disturbances in the cerebrospinal fluid (CSF) circulation due to the mass effect of the aneurysm may also contribute to the enlargement of the perianeurysmal cyst.[2,3,6] In recent years, it has become known that CSF flow synchronized with arterial pulsation and morphological changes in the surrounding brain parenchyma can alter local CSF pressure gradients in the ventricular system.[15] These cysts adjacent to the aneurysm may increase in size over time, resulting in changes in the local pressure gradients

Table 1: Sumarry of intracranial perianeurysmal cyst

No.	Reference	Age	Sex	Symptom	AN location	AN size	SAH	AN treatment	AN recurrence	Duration form initial AN treatment to cyst diagnosis	Cyst size	treatment for cyst	recurrence of the cyst (follow up)
1	Hirota *et. al*, 1999	71	M	hemiplegia	MCA	NM	+	+	NM	30 years	NM	cyst removal	no recurrence (NM)
2	Sato *et. al*, 2000	51	M	seizure	PCA	20mm	-	ET	NM	none	35mm	biopsy of the cyst	unchanged (4 years)
3		62	M	none	ICA	15mm	-	ET	NM	none	15mm	NM	NM
4		35	F	headache	PCA	19mm	-	Clipped	NM	none	25mm	NM	NM
5		46	M	headache	ACA	9mm	-	Clipped	NM	none	20mm	NM	NM
6		57	M	unknown	ACA	40mm	-	none	NM	none	34mm	none	Died of AN rupture
7	Takai *et. al*, 2001	64	F	alexia, aphasia	PCA	30mm	-	ET	NM	none	60mm	cyst fenestration	no recurrence (3 months)
8	Marcoux j *et. al*, 2002	62	F	hydrocephalus	Acom A	NM	+	ET	-	17 months	25mm	cyst fenestration (open)	recurrence (2 months)
9	Fliedman *et. al*, 2003	70	F	hemiplegia, dysarthria	BA	18mm	-	ET	+	20 months	NM	cyst fenestration VP shunt	recurrence (2 motnhs)
10	Benvenuti *et. al*, 2006	54	M	headache	MCA	NM	-	Clipped	NM	none	NM	cyst fenestration	no recurrence (30 months)
11	Martinez GM *et. al*, 2011	74	M	headache	MCA	20mm	-	ET	+	15 months	NM	none	decreased cyst (2 months)
12	Barber *et. al*, 2014	72	F	headache, hemiplegia	BA/ ICA	21/ 3.5mm	-	ET	+	22 months	20mm	ETV	NM
13	Grandhi *et. al*, 2014	51	M	headache	ICA	27mm	+	ET	+	3 years	50mm	Stereotactic aspiration	recurrence (4 weeks)
14	Norris *et. al*, 2015	80	F	hemifacial spasm	ICA	15mm	-	ET	+	2years	NM	levodopa	increased cyst (15 months)
15	Kulwin CG *et. al*, 2015	74	F	headache, gait disturbance	MCA	NM	-	Clipped	NM	none	NM	cyst fenestration (open)	no recurrence (NM)
16	Jayakumar *et. al*, 2019	64	M	hydrocephalus	ICA	NM	-	Clipped	NM	none	NM	cyst fenestration	recurrence (3 months)
17	Birua *et. al*, 2021	50	F	falcian herniation, vomit, headache	MCA	13.2mm	-	Clipped	NM	none	54mm	excision of the cyst	no recurrence (3 months)

Table 1: (*Continued*)

No.	Reference	Age	Sex	Symptom	AN location	AN size	SAH	AN treatment	AN recurrence	Duration form initial AN treatment to cyst diagnosis	Cyst size	treatment for cyst	recurrence of the cyst (follow up)
18	kobayashi *et. al*, 2022	77	F	none	VA-PICA	12mm	+	ET	+	2 years	25mm	cyst fenestration (open), Ommya reservoir	no recurrence (1 year)
19	Liang *et. al.* 2022	52	M	none	Acom A	10mm	+	ET	+	4 years	23mm	additional treatment for AN	unchanged (10 years)
20		54	M	none	Acom A	9.5/ 5mm	+	ET	+	9 months	18mm	none	decreased cyst (4 years)
21		65	M	none	Acom A	19mm	-	-	-	none	10/ 8mm	none	increased cyst (4 years)
22	Present case	64	M	hydrocephalus	Acom A	18mm	+	ET	+	2 years	37mm	cyst fenestration, VP shunt	no recurrence (1.5 years)

Abbreviation: M, male; F, female; ICA, internal carotid artery; PCA, posterior cerebral artery; ACA, anterior cerebral artery; ACoA, anterior communicating artery; BA, basilar artery; MCA, middle cerebral artery; VA, vertebral artery; PICA, posterior inferior celeblum artery; NM, not mentioned; ET, endovascular treatment; ETV, endoscopic third ventriculostomy; VP, ventriculoperitoneal

and morphology of the brain parenchyma associated with arterial pulsation.

Histopathological studies were performed in six cases, including the present case.[2,5,6,19,20] In most cases, reactive gliosis was present without hemorrhage or inflammation. In the present case, neovascularization was not detected in the cyst wall, as confirmed by Elastica van Gieson staining. In addition, fibrotic changes under the ependymal layer were detected in the cyst wall, whereas inflammatory cell infiltration in the cyst wall was not detected. These findings suggest that the origin of the cyst was not the aneurysm itself but rather the adjacent brain tissue. Subclinical hemorrhage or effusion leakage contributes to cyst enlargement and originates from an adjacent aneurysm rather than from the cyst wall. Furthermore, because there were no inflammatory changes in the cyst wall, the inflammation of the aneurysmal wall may have only contributed to the early stages of cyst formation, with the fibrous structure being a secondary change. Other mechanisms, such as impaired local perfusion of CSF, may be involved in the subsequent delayed cyst expansion.

Surgical treatment of cysts has been attempted, including cyst fenestration using a neuroendoscope, cyst wall excision using microsurgery, and aspiration of the cyst contents using stereotactic surgery.[2,6,7,13,20] Many cases have been successfully treated; however, there have been cases of recurrence within a relatively short period, ranging from a few weeks to months. Although there are only a few reported cases with long-term prognoses, adequate excision of the cyst wall and establishing a connection to the CSF space, such as the ventricle or cistern, may be necessary rather than the simple aspiration of the contents. In this regard, neuroendoscopic cyst fenestration has already been performed for other cystic lesions adjacent to the ventricles and is considered highly useful as a treatment for perianeurysmal cysts.[4] However, when performing neuroendoscopic surgery, clinicians should confirm that there is no blood flow from the aneurysm to the cyst by performing preoperative cerebral angiography, as in this case.

CONCLUSION

Perianeurysmal cyst formation is rare in patients with cerebral aneurysms. Particularly, in cases requiring additional treatment for recurrent cerebral aneurysm, including neck remnants after endovascular treatment, perianeurysmal cysts may develop during long-term follow-up, and prompt examination and therapeutic intervention, including opening of the cyst by neuroendoscopy, are necessary when symptoms of suspected hydrocephalus develop. Therefore, clinicians should be aware of the risk of delayed development of perianeurysmal cysts after endovascular treatment for cerebral aneurysms and the need for careful long-term follow-up after treatment.

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Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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