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A Ruptured Basilar Tip Aneurysm Showing Repeated Perianeurysmal Edema after Endovascular Coil Embolization: Case Report

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Abstract

The authors present an extremely rare case of a 48-year-old female who developed repeated perianeurysmal edema at 2, 9, and 16 weeks after endovascular coil embolization for the ruptured intracranial aneurysm. Interestingly, the mechanism for this edema could be different at each time point in this case; acute thrombosis formation, chemical inflammation, and aneurysm recanalization. We have to be aware of this potential complication in the long term after endovascular coil embolization for the intracranial aneurysm, especially with large size or buried into the brain parenchyma. The clinical implications of this case are discussed with a review of the literature.

Key words: perianeurysmal edema, aneurysm, coil embolization, mechanism

Introduction

Perianeurysmal edema is rare, but one of the serious complications following endovascular coil embolization for the intracranial aneurysm.[1–11] Proposed mechanism for the perianeurysmal edema are acute thrombosis formation, chemical inflammation, and pulsatile blood flow transmitted to the brain parenchyma due to aneurysm recanalization.[1–4] Recently, some reports have implicated that second generation aneurysm coils including Matrix® (Stryker, Kalamazoo, Michigan, USA), Micrus Coil® (Micrus Endovascular, San Jose, California, USA), and HydroCoil® (MicroVention, Inc., Tustin, California, USA) are easy to induce perianeurysmal edema compared with bare platinum coils, because of an inflammatory response from the implanted materials on the coils.[2,4,10,11] We herein present an extremely rare case showing repeated perianeurysmal edema after endovascular coil embolization for a ruptured aneurysm with different etiologies over time, and discuss its clinical implications with a review of the literature.

Case Report

A 48-year-old woman was referred to our neurosurgery department for a severe headache. Her neurological grade was Hunt & Kosnik grade 2 and World Federation of Neurological Surgeons (WFNS) grade 2. She had no history of high blood pressure or smoking. After admission, neck stiffness was evident without any neurological deficit on physical examination. Computed tomography studies of the brain confirmed subarachnoid hemorrhage (Fisher grade 3) with hydrocephalus, and digital subtraction angiography demonstrated an aneurysm at the basilar bifurcation, which extended superiorly to the midbrain (Fig. 1A, B).

I. Initial treatment and 1st perianeurysmal edema

Endovascular coil embolization for the aneurysm was performed under general anesthesia on the day of admission. Bare platinum coils (GDC®, Boston Scientific, Natick, Massachusetts, USA) were used for embolization with balloon neck remodeling technique, which resulted in a occlusion with a slightly neck remnant (Fig. 1C). The postoperative course was uneventful, and the patient was discharged with no neurologic deficit. Interestingly, asymptomatic perianeurysmal edema was detected in the midbrain on fluid attenuated inversion recovery sequence with magnetic resonance imaging (FLAIR-MRI) 2 weeks after the initial treatment (Fig. 1D). The edema was spontaneously improved in the follow-up examination.

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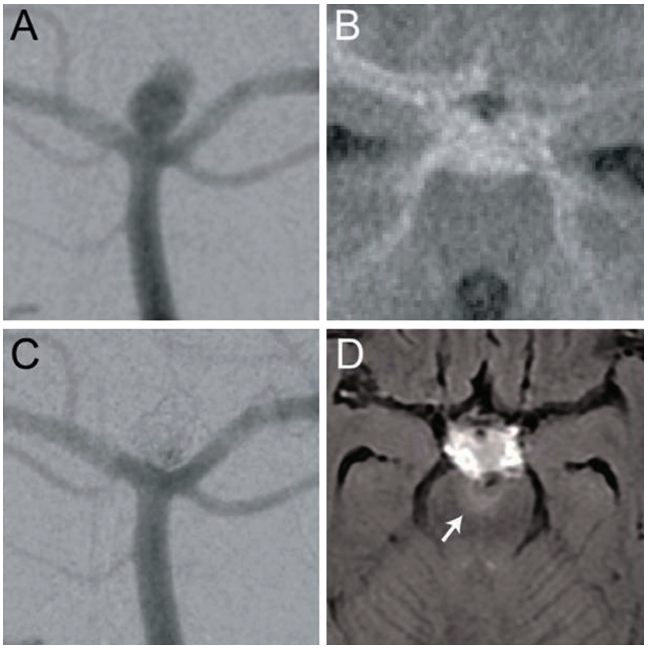

Fig. 1 Preoperative digital subtraction angiography (DSA) showing a ruptured aneurysm at the basilar bifurcation, which extended superiorly to the midbrain (A). Computed tomography showing diffuse subarachnoid hemorrhage in the basal cistern (B). Note that no perianeurysmal edema was detected preoperatively. The aneurysm was obliterated with GDCs®, with a slight neck remnant (C). Postoperative fluid attenuated inversion recovery sequence with magnetic resonance imaging detected perianeurysmal edema, which resolved spontaneously (*arrow* in D).

II. Recanalization, re-embolization, and 2nd perianeurysmal edema

Follow-up angiogram demonstrated aneurysm recanalization and regrowth, with a perianeurysmal edema again on FLAIR-MRI 8 weeks after the initial treatment (Fig. 2A, B), but she presented no obvious neurological symptoms. She underwent re-embolization with bioactive coils and bare platinum coils (Matrix®, Micrus Coil®, and GDC®) 9 weeks after the initial treatment, and complete occlusion was achieved (Fig. 2C). However, perianeurysmal edema massively deteriorated on postoperative FLAIR-MRI at 2 days after the procedure (Fig. 2D), which was spontaneously improved 4 weeks later (Fig. 2E).

III. Recanalization and 3rd perianeurysmal edema

Follow-up angiogram demonstrated aneurysm recanalization and regrowth again in parallel with asymptomatic perianeurysmal edema on FLAIR-MRI 16 weeks after the initial treatment (Fig. 3A, B). She underwent the 3rd embolization with GDC® 31 weeks after the initial treatment, and complete occlusion was achieved (Fig. 3C). FLAIR-MRI 2 days after the third treatment revealed a slight improvement of the edema (Fig. 3C).

Discussion

Endovascular embolization of the cerebral aneurysms has become an accepted therapy for prevention or treatment of aneurysm rupture.[12–14] A prospective, randomized,

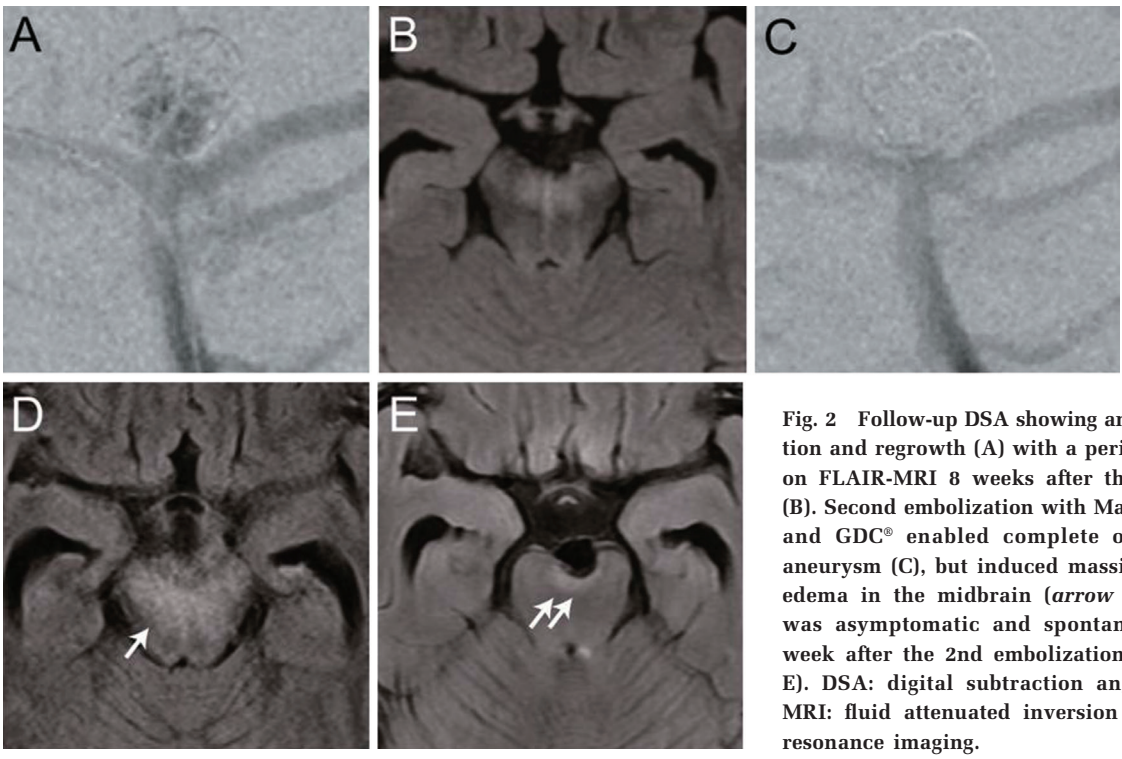

Fig. 2 Follow-up DSA showing aneurysm recanalization and regrowth (A) with a perianeurysmal edema on FLAIR-MRI 8 weeks after the initial treatment (B). Second embolization with Matrix®, Micrus Coil®, and GDC® enabled complete obliteration of the aneurysm (C), but induced massive perianeurysmal edema in the midbrain (*arrow* in D). The edema was asymptomatic and spontaneously resolved a week after the 2nd embolization (*double arrow* in E). DSA: digital subtraction angiography, FLAIR-MRI: fluid attenuated inversion recovery-magnetic resonance imaging.

controlled trial, the international subarachnoid aneurysm trial, found that patients who underwent endovascular coiling of ruptured intracranial aneurysm had a 6.9% absolute risk reduction in dependency or death at 1 year, compared with those who underwent surgical clipping.[15] However, the long-term efficacy and durability of endovascular coiling remains in controversial. Edema formation in the perianeurysmal region has been reported to be one of

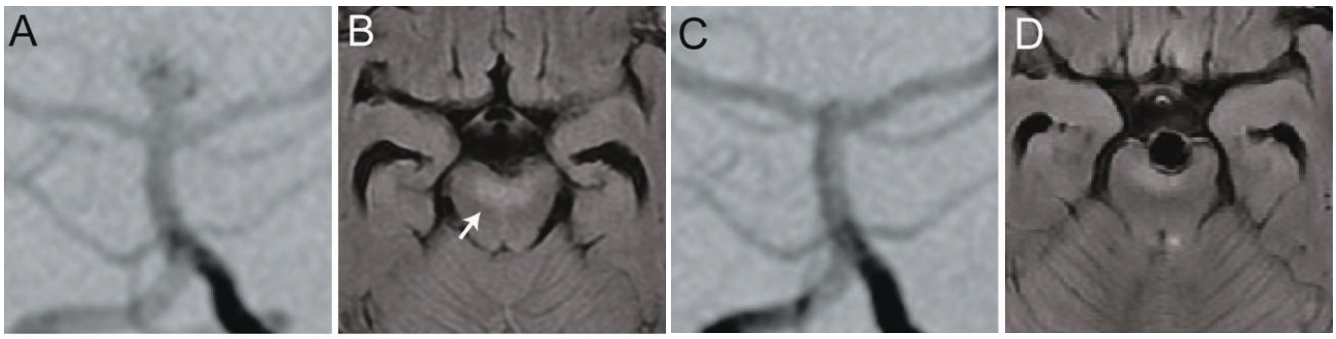

Fig. 3 Follow-up DSA showing aneurysm recanalization and regrowth again (A) with a perianeurysmal edema on FLAIR-MRI 16 weeks after the initial treatment (*arrow* in B). Third embolization with GDC® enabled complete obliteration of the aneurysm (C), which achieved with a slight improvement of the edema (D). DSA: digital subtraction angiography, FLAIR-MRI: fluid attenuated inversion recovery-magnetic resonance imaging.

Table 1 Reported cases showing perianeurysmal edema after endovascular coil embolization

Author, year	Age, sex	Location	Aneurysm size (mm)	Coil type in the initial Tx	Onset (day)	Presumed cause	Treatment	Outcome
Meyers et al., 2004[5]	46, F	PCA	20	GDC, Matrix, HC	21	C	Corticosteroid	Improved
Horie et al., 2007[3]	72, M	ICA paraclinoid	15	GDC,	180	R	Re-embolization	Improved
	72, F	BA-SCA	15	GDC	90	R	Corticosteroid, Re-embolization	Improved
	73, F	ICA terminalis	16	GDC	90	R	Re-embolization	Improved
Pickett et al., 2007[7]	55, F	ICA paraclinoid	20	HC, BPC	21	C	Corticosteroid	No change
	56, M	ICA paraclinoid	25	HC, BPC	30	C	Corticosteroid	Deteriorated
Turner et al., 2008[8]	69, F	ICA paraclinoid	22	HC, BPC	14	C	Corticosteroid	NA
Marden and Putman, 2008[4]	27, F	ICA terminalis	8	GDC, Matrix	7	C	None	Improved
Fanning et al., 2008[2]	47, F	ICA paraclinoid	14	BPC, HC	129	R, T	None	No change
	51, F	ICA paraclinoid	7.3	BPC, HC	4	C	None	Improved
	72, F	ICA terminalis	10.5	BPC, HC	249	C	NA	NA
	59, M	ICA terminalis	10.2	BPC, HC	36	C	None	NA
	59, M	ICA terminalis	12.9	BPC, HC	39	C	NA	NA
White, 2008[11]	73, M	VA	17	Orbit, Micrus, MicroPlex	7	C	Corticosteroid	Deteriorated
	55, M	BA	13	MicroPlex	6	C	NA	NA
Craven et al., 2009[1]	51, F	MCA	7	BPC	90	C	None	Improved
Vu Dang et al., 2009[9]	46, F	ICA Pcom	12	Axium, GDC, MicroPlex	30	C	Corticosteroid	Improved
Misaki et al., 2010[6]	69, F	ICA	19	GDC, Orbit	6	C	None	Improved
Present case	48, F	BA	6	GDC, Matrix, Micrus	14, 63, 112	T, C, R	Re-embolization	Improved

BA-SCA: basal artery-superior cerebellar artery, BPC: bare platinum coil, C: chemical inflammatory reactions by the coils, HC: hydro coil, ICA: internal carotid artery, MCA: middle cerebral artery, NA: not available, PCA: posteriro cerebral artery, Pcom: posterior communicating artery, R: recanalization, T: thrombosis, Tx: treatment, VA: vertebral artery.

the serious complications after the endovascular treatment.

Reported cases showing perianeurysmal edema after endovascular treatment are summarized in Table 1.[1–12] There were 19 patients with a mean age of 58 years. Among them, only 3 patients had ruptured aneurysm suggesting subarachnoid hemorrhage itself did not affect perianeurysmal edema formation. It is noteworthy that most cases had large aneurysm which buried in the brain parenchyma, and our case also had an enlarged aneurysm which buried in the midbrain. In terms of the coil materials, even bare platinum coils can induce perianeurysmal edema and several kinds of coils were used all together in most cases. Treatment options for perianeurysmal edema were corticosteroids and re-embolization. Medical therapy with corticosteroids was considered if the symptoms were progressive in parallel with imaging findings.[4] The edema finally improved spontaneously or with treatment in 10 patients (52.6%).

In order to evaluate the etiology of de novo perianeurysmal edema, we divided the presumed causes of the edema into three types: acute thrombosis, chemical inflammatory reactions by the coils, recanalization of the aneurysm with mass effect.[1–11] In most cases, chemical inflammatory reaction was considered as a main cause of the edema formation regardless of the coil types.[1,2,4–11] Interestingly, perianeurysmal edema possibly due to chemical inflammation has a wide distribution of the edema onset (day 4 to day 249, median: day 21 after the initial treatment) indicating that most of the edema are asymptomatic and incidentally detected with postoperative imaging in most cases.

In the present case, repeated perianeurysmal edema following coil embolization could be classified into all the three types. In the 1st perianeurysmal edema, acute thrombosis formation in the aneurysm or chemical inflammation due to bare platinum coils could contribute to the edema since this is spontaneously resolved. Thrombus formation within the aneurysm is reported to induce cytokine release from activated platelets.[16] On the other hand, aneurysm recanalization and regrowth could contribute to the 2nd and 3rd edema formation since the edema size was parallel with the degree of recanalization and regrowth. The pulsatile blood flow when striking the coils may result in a regrowth of the aneurysm and may also be transmitted to the aneurysm wall via the coils, thus leading to the perianeurysmal edema.[3] In this situation, the edema improved after re-embolization of the aneurysm. Finally, chemical inflammatory reactions from second generation coils and bare platinum coils could contribute to the massive edema after 2nd re-embolization, which resolved spontaneously. The second generation coils are developed to promote healing across the aneurysm neck by inciting an intense fibrocellular response.[4] In an experimental aneurysm model, bioabsorbable polymeric material (BPM) treated aneurysms demonstrated persistent macrophage enrichment compared to GDC,[17] and therefore second generation coils could enhance inflammatory reactions after the embolization.

We describe an extremely rare case of a ruptured aneurysm showing repeated perianeurysmal edema after endovascular coil embolization with different etiologies over time. We have to be aware of this potential complication in the long term after endovascular coil embolization for the intracranial aneurysm, especially with large size or buried into the brain parenchyma.

Conflicts of Interest Disclosure

None.

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