Case Report

Role of Endovascular Approach as Diagnostic Technique and First-Line Therapy for the Patients with Micro-Arteriovenous Malformations: A Case Report and Literature Review

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Objective: We describe a rare case report of micro-arteriovenous malformation (micro-AVM) treated by the endovascular approach in addition with literature review.

Case Presentation: A 12-year-old boy presenting with a spontaneous intracerebral hematoma in the left occipital lobe underwent conventional diagnostic workups. The results of initial catheter angiography were considered to be equivocal as the AVM. Superselective angiography (SA) demonstrated a micro or small AVM (single feeder and single drainer type) with an aneurysmal dilatation. Immediate transarterial embolization (TAE) might fail to occlude the whole of nidus area completely, and subsequently, we switched to the surgical exploration of AVM lesion. Intraoperative findings demonstrated that the whole of AVM lesion had already been occluded completely, indicating the complete occlusion by TAE only. Pathological findings of the surgical specimen showed an aneurysmal dilatation was a venous aneurysm with vulnerable vascular wall structure, which was certainly the source of bleeding. Based on the above results, the retrospective revaluation of superselective angiogram permitted us to understand that the nidus of AVM was micro nidus type and TAE had resulted in the complete nidus occlusion.

Conclusion: SA is the most useful diagnostic modality to clarify the angioarchitecture of micro-AVM and AVM-related aneurysms. If SA is successfully performed and relatively safe TAE is expected to be possible, the subsequent attempt to do curative embolization as a first-line treatment may be worthy of consideration. However, the surgical procedure should be fully reserved for the possible incomplete obliteration and hemorrhagic complications.

Introduction

Cerebral micro-arteriovenous malformations (micro-AVMs) were originally defined by Yasargil[1] as brain AVMs with a

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nidus with <1 cm in diameter. They are thought to account for about 7% of all cerebral AVMs and 21% of AVMs presenting hemorrhage onset.[2] It can be challenging to diagnose and treat micro-AVMs during the management of acute spontaneous intracranial hemorrhage, because we often have difficulty in visualizing the whole angioarchitecture of micro-AVMs by various conventional diagnostic modalities.[3–8] Previous reports predominantly recommended the surgical treatment.[3,7–10] However, most recently, the major progresses in the field of endovascular devices and therapeutic techniques for AVMs have increased the role of endovascular approach in the diagnosis and treatment of micro-AVMs.[2,4–6] It was reported that there were not rare cases that small AVMs consisting of single or a few feeders could be completely occluded by only the endovascular treatment,[11] suggesting that micro-AVMs typically consisting of

single feeder and single drainer may be cured with endovascular treatment only. In the present case, we underwent the superselective diagnostic angiography and transarterial embolization (TAE) for a micro-AVM as a first-line therapy. After TAE, we confirmed the complete occlusion of a micro-AVM by direct surgery. We described a rare case report of micro-AVM in addition with literature review.

Case Presentation

A 12-year-old boy with no past medical history complained of a mild headache, nausea, and the visual disturbance in the right eye. Next day, he kept feeling these symptoms, and then his family took him to our hospital. In the emergency outpatient department, neurological examination appeared to be almost normal except for the slight visual disturbance in the right visual field. An emergency CT showed a 30 × 28 × 28 mm-sized intracerebral hematoma in the left occipital lobe (**Fig. 1A**). He underwent emergency 3D-CTA, which showed no definitive vascular pathology (**Fig. 1B** and **1C**). As a subsequent examination, conventional catheter angiography was performed. Vertebral angiography showed an abnormal vessel with the tangled vascular structure in the distal portion of a posterior cerebral artery in the late arterial phase (**Fig. 2A** and **2B**). This abnormal vessel showed the prolonged arterial filling (**Fig. 2C**). In addition, the sagittal source images of 3D rotational angiography demonstrated that an abnormal vessel was running just beneath the hematoma (**Fig. 2D**). Meanwhile, other angiographic findings such as the nidus and drainers were not definitely confirmed. A follow-up angiography including superselective angiography (SA) was planned after a week, because an underlying micro-AVM was strongly suspected. The follow-up angiography was performed under general anesthesia. Conventional angiography showed no interval change. For the superselective exploration, a 4-Fr introducer sheath in the right femoral artery was exchanged to a 5-Fr ASAHI FUBUKI Dilator Kit (Asahi Intecc, Aichi, Japan), which was guided into the left vertebral artery. A Guidepost (Tokai Medical, Aichi, Japan) was placed in the second portion of a posterior cerebral artery (P2) with the same axis as an intermediate catheter. A DeFrictor Nano Microcatheter (Medico's Hirata, Osaka, Japan) with a microguidewire (ASAHI CHIKAI X 010; Asahi Intecc) was navigated up to the portion just proximal to the beginning of an abnormal artery. SA showed that the abnormal artery (a definite feeder) ran superiorly, and then, after turning

back, it ran inferiorly toward the superficial area, draining into the superficial cerebral vein (**Fig. 3A–3C**). Additionally, on the way of descending to the superficial, an aneurysmal dilatation was revealed, which might be the source of bleeding. SA revealed a micro or small AVM consisting of a single feeder and a single drainer with an aneurysmal dilatation. Regarding the nidus area, the vessel's caliber change was observed in the vessel's U-turn part. The descending vessel after an aneurysmal dilatation was thought to be a drainer side because of the obvious vessel thickness and tortuousness. Therefore, we speculated that the nidus area existed somewhere between vessel's U-turn point and aneurysmal dilatation at least (**Fig. 3B** and **3C**). Following the SA, we decided to perform TAE. Because of the small-sized and tortuous feeder, we had difficulty in the microcatheterization, which resulted in the vessel perforation at the origin of the feeder. In order to embolize the perforated feeder, we pulled a microcatheter toward the proximal site of vessel perforation and injected 25% *n*-butyl-2-cyanoacrylate (NBCA) with the further expectation of glue penetration to the nidus, if possible. The vessel perforation was occluded. The injected glue penetrated to the vessel's U-turn part beyond the caliber change point and additionally penetrated to another newly emerged silent feeder too (**Fig. 3D**). The possible silent feeder ran not toward the vessel's U-turn part (the caliber change point) but toward the aneurysmal dilatation. Vertebral angiography demonstrated that the extravasation disappeared and abnormal vessels related to AVM became invisible (**Fig. 3E** and **3F**). After the embolization, a head CT showed that the hemorrhagic complication was minor (**Fig. 3G**). However, we subsequently decided to perform the surgical exploration of AVM, because we couldn't reach the conviction for the glue penetration to the whole of nidus area.

The patient was placed in the prone position and underwent a left occipital craniotomy. After opening the dura mater, we evacuated the intracerebral hematoma and explored the cavity wall with reference to NBCA casts showing by the fluoroscopic image of C-arm. Using the NBCA-filled feeders as a radiographic marker, we easily found a small, tangled vessel with a berry aneurysmal dilatation (**Fig. 4A**). These vascular structures had already collapsed, and Doppler flowmetry revealed no blood flow signal. Any other vascular components suspected of the nidus were not observed near the aneurysmal dilatation. The parent vessel of an aneurysmal dilatation showed a vein-like appearance. Pathological findings of the surgical
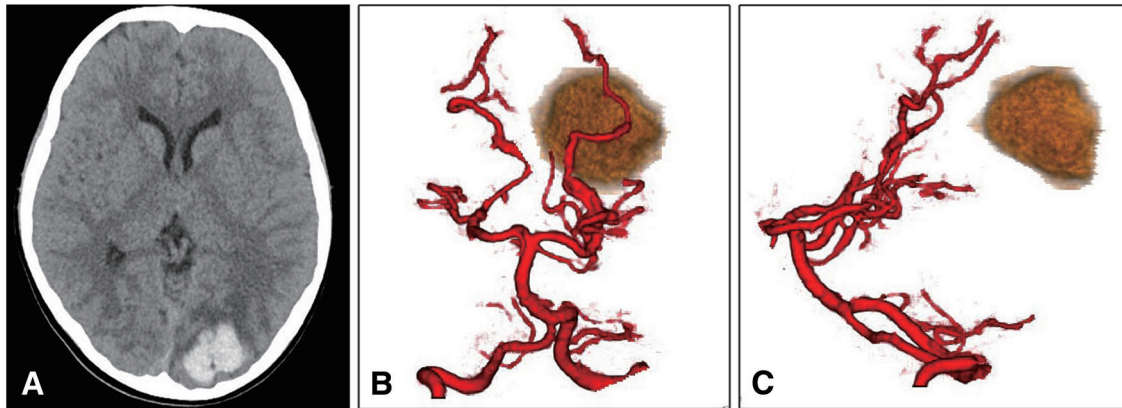
Fig. 1 A head CT on admission (**A**) showed a subcortical hematoma in the left occipital lobe. 3D-CTA in the anterior–posterior view (**B**) and the lateral view (**C**) did not show any abnormal vessels around the hematoma.
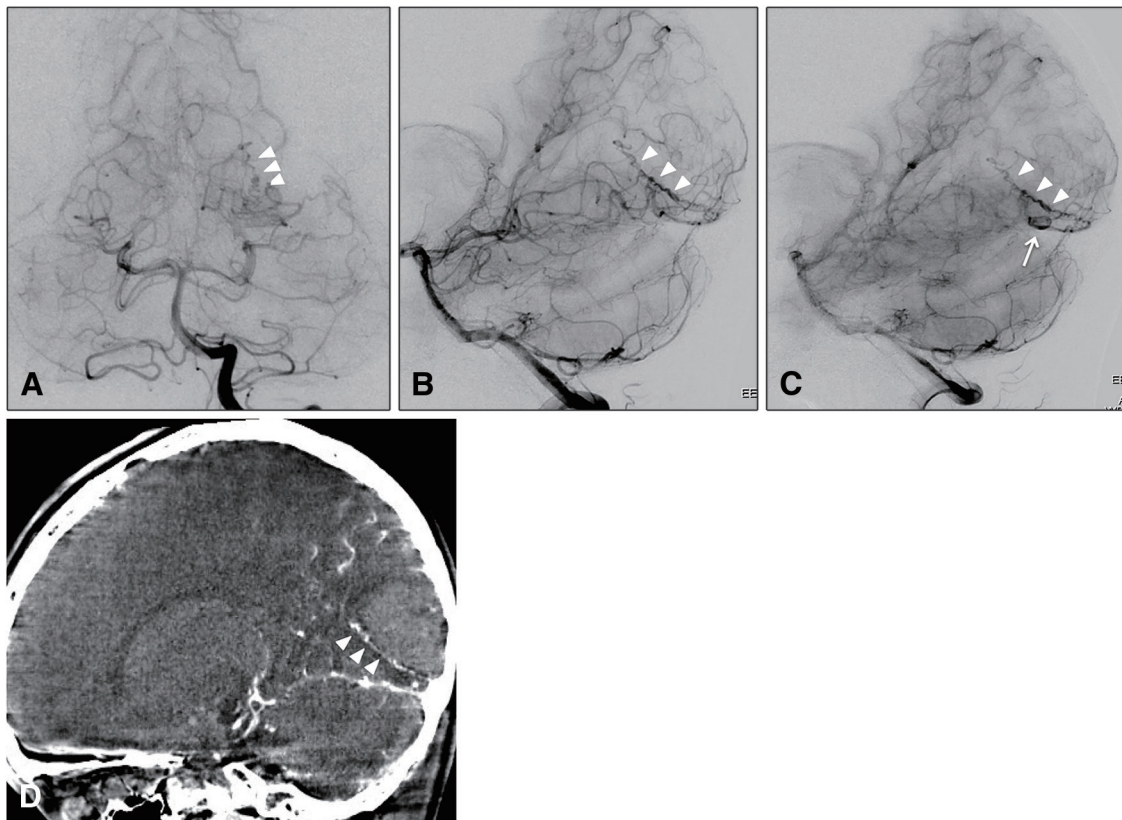

Fig. 2 Conventional catheter angiography was performed on admission. Anterior–posterior view (**A**) and lateral view (**B**) in the left vertebral angiography showed an abnormal vessel with a tangled vascular structure (white arrowheads in **A**–**C**) in the late arterial phase. (**C**) An abnormal vessel showed the prolonged filling of contrast medium until the capillary phase (**C**: lateral view in the capillary phase). An abnormal vessel eventually drained into a superficial cerebral vein (white arrow). Although an underlying AVM lesion was suspected, the anatomical relationship among feeder, nidus, and drainer was not definitely identified. The result of initial angiography was judged to be equivocal as an arteriovenous malformation. (**D**) The sagittal source image of 3D rotational angiography showed an abnormal vessel running (white arrowheads) just beneath the hematoma, suggesting that the abnormal vessel was related to the source of bleeding. AVM: arteriovenous malformation

specimen demonstrated that the aneurysmal wall was composed of the vein-like structure without the elastic fiber and connective tissue with the inflammatory change, indicating a venous aneurysm with the vulnerable vascular wall

(**Fig. 4B** and **4C**). The nidus could not be identified pathologically due to a small amount of specimen for a pathological examination. Postoperative angiography 2 weeks after the operation did not show any abnormal angioarchitecture.
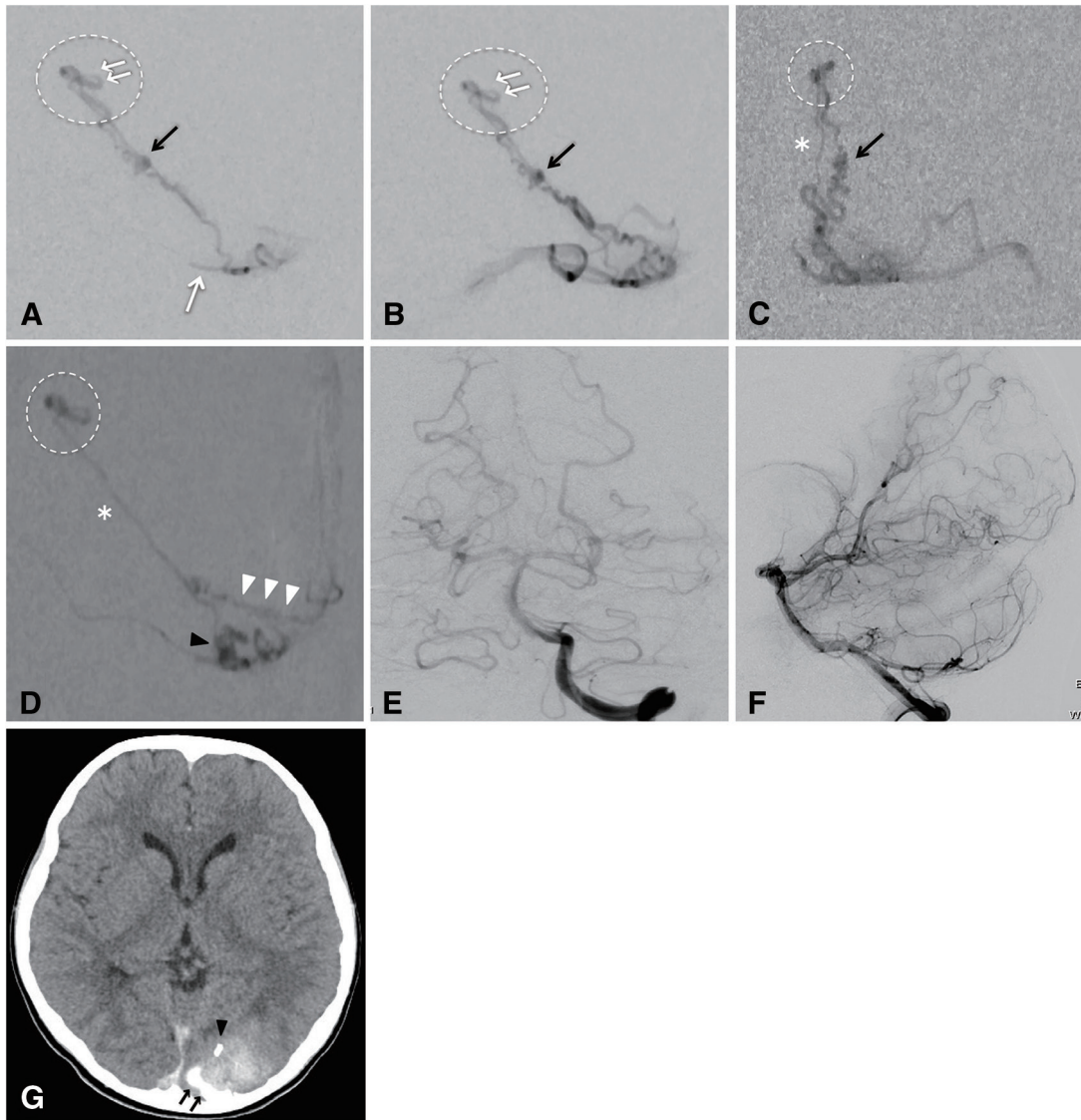
Fig. 3 At the repeated angiography 1 week after the onset, SA was performed. The lateral view of SA in the late arterial phase (**A**) and in the capillary phase (**B**). The anterior–posterior view of SA (**C**). Single white arrow (in **A**) indicates the position of a microcatheter tip. Double white arrows (in **A** and **B**) indicate the vessel's caliber change point. Asterisks (in **C** and **D**) indicate an ascending definite feeder. A feeder ran superiorly, and then, after turning back, it ran inferiorly toward the superficial area, draining into the superficial cerebral vein (dotted circle in **A**–**C**: vessel's U-turn part). On the way of descending to the superficial part, an aneurysmal dilatation (black arrows in **A**–**C**) was revealed. The vessel's caliber change point (double white arrows) was observed in the vessel's U-turn part (dotted circles). The descending vessel after the aneurysmal dilatation was thought to be a drainer side because of the obvious vessel thickness and tortuousness. Therefore, the nidus area was speculated to exist somewhere between the vessel's U-turn point and aneurysmal dilatation. The fluoroscopic image after TAE by 25% NBCA (**D**). The injected glue penetrated to whole of the vessel's U-turn part (dotted circle in **D**) beyond the caliber change point and additionally penetrated to another newly emerged possible silent feeder (white arrowheads in **D**) too. At the vessel perforation site, NBCA casts were massively aggregated to the extravascular space (black arrowhead in **D**). Anterior–posterior view (**E**) and lateral view (**F**) of left vertebral angiography after TAE, showing that the extravasation disappeared and abnormal vessels related to the AVM became invisible. A head CT immediately after the endovascular treatment (**G**), showing a piece of NBCA cast (black arrowhead) and localized extravasation of contrast medium (black arrows). AVM: arteriovenous malformation; NBCA: n-butyl-2-cyanoacrylate; SA: superselective angiography, TAE: transarterial embolization

The patient showed good recovery with no new neurological deficits and was discharged home.

Written informed consent was obtained from the patient for the publication of this case report and accompanying images.

Discussion

As limited case series of micro-AVMs have ever been reported,[3,6,12,13)] there are still a lot of unknown characteristics of micro-AVMs. Most of micro-AVMs were reported to
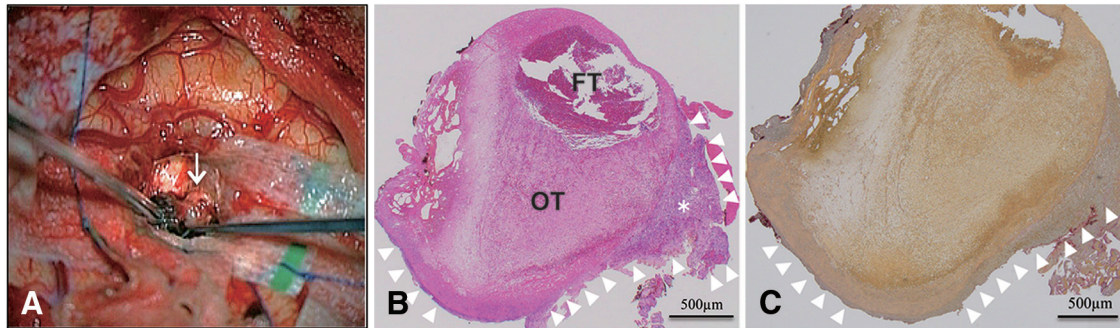
Fig. 4 (**A**) Intraoperative photograph, showing a berry aneurysmal dilatation (white arrow) with the blood clot. Resected tissue of the aneurysmal dilatation was pathologically examined by H&E staining (**B**, scale bar: 500 μm) and EVG staining (**C**, scale bar: 500 μm). The section of H&E staining demonstrated the massive component of OT and the component of FT inside the blood vessel-like structure and connective tissue (white arrowheads in **B** and **C**) with macrophage infiltration (asterisk in **B**). The component of fresh thrombus was speculated to arise secondarily due to nidus occlusion by TAE. The section of EVG staining demonstrated that the elastic fiber was not almost observed as the arterial wall structure (white arrowheads in **B** and **C**), indicating the vessel wall structure was much closer to a vein than an artery. These pathological findings indicated that an aneurysmal dilatation was compatible with a venous aneurysm with the vulnerable vascular wall structure, demonstrating the source of bleeding. EVG: elastica van Gieson; FT: fresh thrombus; H&E: hematoxylin and eosin; OT: organized thrombus; TAE: transarterial embolization

Table 1 Previous reports on the angiographic characteristics of micro-AVMs

Authors	No. of micro-AVMs	Mean age (y/o)	Initial angiographic results			The rate of SA at the repeated angiography*	Mean delay between onset and diagnosis (days)
			No. of positive results (%)	No. of equivocal results	No. of negative results		
Cellerini et al. 2002[4]	10	48.8	5 (50%)	3	2	4/5	129.8
Perrini et al. 2004[7]	14	44.6	8 (57.1%)	3	3	3/6	50.4
Andreou et al. 2008[2]	25	29.7	18 (72%)	5	2	6/7	Unknown
Alén et al. 2013[3]	28	37.1	22 (78.6%)	0	6	4/6	Unknown

*The rate of patients who underwent SA, when the initial angiographic results were equivocal or negative. micro-AVMs: micro-arteriovenous malformations; SA: superselective angiography; y/o: years old

occur in between children and young people under 50 years of age.[2–8,10,14] First of all, the diagnosis of micro-AVM requires high index of clinical suspicion in young patients with relatively large-sized and supratentorial lobar hemorrhage. Several papers demonstrated tiny vascular lesions in the walls of hematoma cavities to be pathologically compatible with micro-AVM in patients with atypical intracranial hematomas.[15,16] According to previous reports,[3,6,10] micro-AVMs are mostly located in the superficial areas and sometimes located in the deep-seated areas. Neither CT nor MRI findings are usually diagnostic. While conventional angiogram is thought to be a gold standard modality for diagnosis of AVMs, not all micro-AVMs can be detected on conventional angiogram. Early angiography in the setting of large intracranial hemorrhage may obscure identification of the micro-AVMs. Most investigators have speculated that low blood flow through the lesion, thrombosis, post-hemorrhagic vascular spasm, or compression of the vessels by the hematoma may account for the lack of

visualization of the micro-AVMs in the acute or subacute phase.[3,4,6,7] Angiographically demonstrated micro-AVMs are reported to be composed of a small nidus, which are slightly visualized by filling in the late arterial phase due to the low blood volume and slow blood flow.[2,4] The feeding artery is usually not dilated, and there is no transdural supply. Venous drainage is usually a single draining vein (occasionally two draining veins) of normal or slightly enlarged caliber. Some patients with micro-AVMs show angiographically equivocal results such as capillary blush, prolonged distal arterial filling, venous pooling, and early venous filling in the absence of a nidus. Even negative angiographic findings are not uncommon. Therefore, even if the initial angiograms show the equivocal or negative results, patients suspected of micro-AVMs should undergo repeated angiography including SA after the resolution of hematomas. Several articles have demonstrated that SA with placement of microcatheters into the feeding arteries was helpful in the diagnosis of micro-AVM (**Table 1**).[2,3,4,7]

Additionally, several papers reported that SA was helpful in detecting underlying aneurysms that might be the source of bleeding and the indication for emergency embolization.[2,17] Regarding the treatment of micro-AVMs, eight publications of micro-AVMs treated by endovascular treatment were found in the literature between 1992 and 2021 (**Table 2**).[2–8,14] As shown in **Table 2**, recent case series showed that the complete obliteration rate by endovascular treatment had an upward tendency.[2–8,14] If the micro-AVM nidus is angiographically accessible and the lesion is judged to be curable by embolization, endovascular treatment as a first-line treatment may be worthy of consideration. However, microcatheter and microguidewire in the distal and small vascular lesions must be cautiously manipulated. Andreou et al.[2] reported vessel perforation and glued microctheter as the complications of SA and TAE. In our review of 8 previous studies reporting the case series of endovascular treatments (**Table 2**), although the complication rate related to the endovascular procedure ranged from 0% to 22%, the permanent morbidity and mortality rate related to the endovascular procedure was relatively low (ranging from 0% to 11%).[2–8,14]

In the present case, while the results of initial examinations were negative findings on CTA and equivocal findings on conventional DSA, SA demonstrated a micro or small AVM with an aneurysmal dilatation. The angioarchitecture of AVM consisted of a single feeder and a single drainer, and the nidus area was speculated to exist somewhere between vessel's U-turn part (the vessel's caliber change point) and an aneurysmal dilatation. The aneurysmal dilatation was suspected as the bleeding source. The NBCA glue penetration to an aneurysmal dilatation by TAE was expected to result in the complete occlusion of AVM. Therefore, we decided to perform the endovascular treatment following SA. Under the unfavorable condition due to the vessel perforation, we successfully embolized not only the perforation site but also the vessel's U-turn part (beyond the caliber change point). Although the AVM lesion disappeared angiographically, the following direct surgery was considered to be necessary in order to explore the unembolized AVM lesion including an aneurysmal dilatation. Intraoperative findings suggested that the complete occlusion of AVM lesion including an aneurysmal dilatation was obtained by the prior TAE only. According to pathological findings of the surgical specimen, a berry aneurysmal dilatation was more compatible with a venous aneurysm than an arterial aneurysm. Regarding the postnidal venous aneurysm,

Table 2 The outcome of endovascular treatment for patients with micro-AVMs in the literature

| Authors | No. of micro-AVMs | Mean age (y/o) | No. of Endo treatment | | CO rate | Embolic agent | Endo complication | M&M related to Endo procedures |
			As first line	As salvage line (postsurgery)	By Endo treatment			
Willinsky et al. 1992[14]	5	38.2	3	0	0% (0/3)	All NBCA (34%–40%)	None	None
Stiver et al. 2000[8]	12	35.7	2	0	0% (0/2)	All NBCA	None	None
Cellerini et al. 2002[4]	10	48.8	6	1	71.4% (5/7)	All NBCA	None	None
Perrini et al. 2004[7]	14	44.6	8	1	77.8% (7/9)	All NBCA	2 (SAH, IVH) → 22%	1 hemianopsia (11%)
Andreou et al. 2008[2]	26	29.7	26	0	76.9% (20/26)	25 NBCA (20%–25%) 1 ONYX	1 perforation 1 ICH (postsurgery) 1 glued catheter → 12%	1 transient hemiparesis (3.8%)
Alén et al. 2013[3]	28	37.1	2	0	100% (2/2)	All NBCA	None	None
De Andrade et al. 2020[5]	3	40.6	3	0	100% (3/3)	All SQUID18 (EVOH-based agent)	None	None
Ferracci et al. 2021[6]	20	47.3	10	1	81.8% (9/11)	9 NBCA 2 ONYX	18.2%	None

micro-AVMs: micro-arteriovenous malformations; CO: complete occlusion; Endo: endovascular; ICH: intracerebral hemorrhage; IVH: intraventricular hemorrhage; M&M: morbidity and mortality; NBCA: *n*-butyl-2-cyano-acrylate; SAH: subarachnoid hemorrhage

some papers reported that the wall of postnidal varix (enlarged venous aneurysm) with the thrombosed component could be composed of vulnerable connective tissue with the inflammatory change such as the infiltration of macrophages compared to the one without the thrombosed component.[18,19] In addition, D'Aliberti et al. reported that the postnidal venous aneurysm close to the nidus tended to resemble a berry aneurysm and it had high bleeding potential.[20] These reports were compatible with the pathological findings of the present case, and these results supported that an aneurysmal dilatation of this case was a venous aneurysm with a significantly high bleeding risk. Based on the intraoperative and pathological findings, the retrospective revaluation of superselective angiogram before TAE allowed us to recognize that the descending abnormal vessel after the caliber change point of vessel's U-turn part was a drainer. As the reason, the following points were considered. First, it seemed to be mildly dilated (that is to say, caliber change) and more tortuous compared with an ascending definite feeder. Second, as the intraoperative findings, the nidus component was not observed near an aneurysmal dilatation and the parent vessel of an aneurysmal dilatation showed the vein-like appearance. Third, the pathological analysis also demonstrated that an aneurysmal dilatation was compatible with the postnidal venous aneurysm. Therefore, we concluded that the nidus area was located between the caliber change point and the beginning of a descending vessel (drainer) in the vessel's U-turn part. From the above results, the AVM of this case was classified to a micro-AVM. Injected NBCA penetrated to a drainer side via the micro nidus during TAE (**Fig. 3D**). It follows that the complete occlusion of a micro-AVM was obtained by the endovascular treatment only.

Our experience in the present case may suggest that the endovascular approach including SA plays a promising role in exploring the angioarchitecture of micro-AVMs and doing the subsequent curative embolization as a first-line therapy. In the present case, the direct surgery after TAE was consequently unnecessary. However, the following surgical intervention was practically helpful for the subsequent therapeutic management because it played a complementary role not only in demonstrating the complete occlusion of a micro-AVM but also in obtaining the additional information about the angioarchitecture of a micro-AVM. In addition, the evidence of TAE for the patients with micro-AVMs is still less well-established than the direct surgery. Therefore, following direct surgery

immediately after TAE should be prepared as a complementary role. Finally, as the limitations of endovascular embolization as a first-line therapy, we should state that the incomplete occlusion by TAE has the possibility of changing the angioarchitecture of AVM into a more complicated one.

Conclusion

It is often difficult to detect the micro-AVMs only by conventional diagnostic workups. When the initial catheter angiography is equivocal or negative, repeated angiography including SA may be favorable for the purpose of making clear the angioarchitecture of micro-AVMs. If SA is successfully performed and relatively safe TAE is expected to be possible, the subsequent curative embolization may be worthy of consideration. However, the surgical procedure should be fully reserved for the incomplete obliteration and hemorrhagic complications.

Acknowledgments

Disclosure Statement

All authors have declared that they have no conflicts of interest associated with this manuscript.

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