Radiological Changes in Infantile Dissecting Anterior Communicating Artery Aneurysm Treated Endovascularly A Case Report and Five-Year Follow-Up

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Summary

Intracranial aneurysms are extremely rare in infants, and to our knowledge only seven infants treated for ruptured spontaneous dissecting aneurysms have been reported. Good outcomes have been achieved with endovascular treatment of infantile aneurysm. We the endovascular treatment of a one-month-old girl for ruptured dissecting aneurysm located in the anterior communicating artery, and the unique radiological changes that were observed during the perioperative and follow-up periods. These changes suggest that blood coagulation and fibrinolytic response play a part in the repair and healing processes of dissecting aneurysms. Careful neuroradiological surveys are needed for pediatric dissecting aneurysms treated endovascularly.

Introduction

Intracranial aneurysms are uncommon in children, accounting for only 0.17–6.8% of all intracranial aneurysm cases¹⁻⁷. Less than 2% of all childhood aneurysms occur during infancy⁸, and the incidence of ruptured aneurysms in infants is extremely low. The location and morphology as well as the clinical and radiological features of pediatric cases differ from those of adult cases^{1,9}.

Posterior circulation and internal carotid artery bifurcation aneurysms are more common.

However, one study revealed that many aneurysms in children are found in the anterior circulation (76%), especially in the middle cerebral artery (MCA)¹⁰. Moreover, the ratio of dissecting aneurysms is high (16–45%) in children^{1,11-13}, and Lasjaunias et al. demonstrated the tendency for the rate of dissecting aneurysms to increase with younger age¹.

Endovascular treatment of intracranial aneurysms leads to a better clinical outcome than surgical clipping, especially when rupture has occurred¹⁴, and is currently being applied to pediatric cases and achieving good outcomes^{13,15,16}. However, long-term efficacy in children remains unclear because no studies with extended post-treatment follow-up periods have been performed thus far. To our knowledge, only seven cases of endovascular treatment of ruptured spontaneous dissecting aneurysms in infants have been reported, but without information on long-term outcomes^{1,12,13,17}. We are also unaware of any case of an infant being treated endovascularly for spontaneous dissecting aneurysm located in the anterior communicating artery (ACoA).

We describe the case of an infant who has been followed for five years since endovascular treatment of a ruptured dissecting ACoA aneurysm, and the unique radiological changes during the perioperative and follow-up periods that may serve as important clues to understanding the repair and healing processes of infantile dissecting aneurysms.

Figure 1 A) Initial brain CT showing SAH around the isodense mass lesion in the interhemispheric cistern. B,C) MRI (T2-weighted sequences) showing an area of low intensity on the ventral side of the mass lesion that was strongly enhanced with gadolinium contrast. D) MRA showing no aneurysms. The left ACA is not visible. ACA, anterior cerebral artery; CT, computed tomography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging; SAH, subarachnoid hemorrhage.

Case Report

A one-month-old girl who had been delivered at 40 weeks' gestation and had an unremarkable perinatal period presented to her local hospital with a generalized seizure. There, computed tomography (CT) of the brain revealed subarachnoid hemorrhage (SAH) around an isodense mass lesion in the interhemispheric cistern (Figure 1A). The infant was immediately transferred to the neonatal unit at our hospital, where the seizure subsided after a short time and neurological examination was unremarkable. The anterior fontanel was soft and sunken and her head circumference was within normal limits. Her platelet count, prothrombin time, and activated partial thromboplastin time were all within normal ranges, but anemia was noted with a hemoglobin of 6.9 g/dL. She had no findings suggestive of an infectious etiology. No signs of trauma were identified, and no retinal hemorrhage was noted on fundoscopic examination; therefore, child abuse was ruled out. She had no congenital factors or family history that could have caused an intracranial aneurysm. Magnetic resonance imaging (MRI) confirmed these results, with T2-weighted sequences showing an area of low intensity in the ventral side of the mass lesion (Figure 1B) that was strongly enhanced with gadolinium contrast (Figure 1C). Magnetic resonance angiography (MRA) was also performed and no aneurysms were seen, but the left anterior cerebral artery (ACA) was not visible (Figure 1D). SAH was present, but findings suggested that its origin was either an aneurysm or a tumor. Her general condition did not require emergency treatment at that time, so we decided to observe her with minimum treatment and do close follow-up with neuroradiological studies.

A follow-up brain CT was performed five days later, and the mass lesion now had high density (Figure 2A). Changes were also seen on follow-up MRI and MRA, with T2-weighted sequences showing large areas of low intensity suggestive of flow void (Figure 2B) and MRA showing an aneurysm-like vascular signal in the midline (Figure 2C). Based on these radiological changes, we were confident that the mass was a vascular lesion. Therefore, cerebral angiography was performed via the right femoral artery under general anesthesia and confirmed the ACoA aneurysm (at the left A1-A2 junction; Figure 3A,B) with a change in luminal caliber at the distal part of the left A1 (Figure 3C). A 4-French catheter was inserted into the left internal carotid artery under fluoroscopic guidance, and an attempt was made to advance the microcatheter along a microguidewire toward the distal side of the diseased vessel to perform endovascular trapping with aneurysmal occlusion. However, the microcatheter was not able to traverse this vessel, which was narrowed by dissection. During this attempt extravasation occurred, and we thus decided to occlude the parent artery from this point proximally. When we began inserting the coils, they were accidentally placed into the aneurysmal sac through the pseudolumen rather than into the vessel. Ultimately parent artery occlusion was achieved when the last coil was placed at the dissected distal A1 and filling of the aneurysm with contrast was no longer observed (Figure 3D). The left pericallosal artery was supplied with retrograde circulation from the splenic artery after surgery, but its flow was quite sluggish (Figure 3E).

Post-coiling, the patient was kept intubated and under sedation for a few days because of thickened SAH caused by intraoperative ex-

Figure 2 A) Day 5 brain CT showing a high-density mass lesion in the interhemispheric cistern. B) MRI showing a significant low-intensity mass lesion located in the same area as on CT. C) MRA showing an aneurysm-like vascular signal in the mid-line. CT, computed tomography; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

Figure 3 A, B) DSA left common carotid artery injection anterior and lateral views showing the aneurysm at the left A1-A2 junction. C) DSA intraoperative working-angle view showing luminal caliber change at the distal A1 (black arrow). D) Postoperative angiogram showing no contrast filling of the aneurysm, and parent artery occlusion is performed by placing a coil at the distal A1 (white arrow). The pericallosal artery is not imaged. E) DSA left vertebral artery injection postoperative view showing retrograde contrast filling of the left pericallosal artery in the capillary phase (white arrowheads). DSA, digital subtraction angiogram.

Figure 4 MRA and source images 2 weeks (A-C), 3 months (D-F) and 1 year (G-I) after endovascular treatment. A) A significant vascular-like signal is shown in the midline. B,C) MRA source images demonstrating that inflow of blood to the aneurysm appears to come from the right A1. D-F) MRA and source images showing complete occlusion of the aneurysm and a faint appearance of the left A2. G-I) MRA and source images showing complete occlusion of the aneurysm. The left A1 is also observed and appears to be connected to the left A2. MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.

travasation, following which she recovered with a normal level of activity and appetite. No neurological deficits were evident, and follow-up brain CT showed washout of the SAH.

The patient was scheduled for two-week follow-up MRA, which showed radiological changes that looked like recanalization of the aneurysm (Figure 4A). The source images of the MRA showed that the left ACA had been

occluded and that blood flow to the aneurysm seemed to come from the right ACA (Figure 4B,C), but we could not deny the possibility that the high-intensity signal indicated a thrombus. However, we hesitated to perform additional invasive studies on the infant because she had no signs or symptoms of rebleeding. We therefore discharged her to home and scheduled her to undergo repeat MRI and

MRA three months later. At the next follow-up, the MRA and its source images confirmed complete occlusion of the aneurysm and the appearance of the left A2 (Figure 4D-F). On one-year follow-up MRA, the aneurysm was not seen but the left ACA had become more visible than on previous imaging (Figure 4G-I). These findings made us consider the occurrence of recanalization.

We continue to follow the patient with annual neuroradiological studies. No aneurysmal recanalization has been found on these follow-up studies thus far. We performed an angiogram when the patient was two years old that showed occlusion of the aneurysm and recanalization of the left ACA but a tangled left A1 course (Figure 5). The most recent angiogram was performed at five-year follow-up and showed findings similar to those seen on the previous one (data not shown). The left ACA was still patent. At the time of this writing, the patient is five and a half years old and is growing up with no apparent neurological deficits or developmental disorders.

Discussion

Pediatric intracranial aneurysms account for 0.17–6.8% of the total incidence of intracranial aneurysms in the general population¹⁻⁷ and are particularly infrequent in infants. Cases of aneurysmal rupture account for 57% to more than 70% in children^{11,13,18} and 73% in infants¹⁰. There are also some differences in aneurysm location between pediatric and adult patients. In children, aneurysms are more commonly found in the posterior circulation and internal carotid artery bifurcation¹¹⁻¹³, and ACoA aneurysms account for 3–16.2%^{11,19,20}. Moreover, the distribution of aneurysms differs slightly between children and infants, in whom many aneurysms are located in the anterior circulation (76%) and in whom aneurysms of the MCA are nearly three times more prevalent than in any other vessel¹⁰. ACoA aneurysms, however, account for only 7% of aneurysms in infants¹⁰.

Dissecting aneurysms can generally be detected by computed tomographic angiography (CTA), MRA, and cerebral angiography. Findings that suggest dissection are irregular stenosis, segmental stenosis, and aneurysmal formation (pearl-and-string sign), irregular fusiform or aneurysmal dilation, double lumen, and occlusion²¹. The etiology of dissecting aneurysm

in pediatric patients differs from that in adults, in whom dissecting aneurysms are often caused by disruption of an internal elastic lamina weakened by longstanding hemodynamic stress such as hypertension²¹. Trauma, infection, or congenital disease can cause dissecting aneurysms from disruption or defect of the vessel lamina not only in adults, but also in children^{11,22}, in whom trauma often causes dissecting aneurysm²³.

Lasjaunias et al. demonstrated the tendency for the rate of dissecting aneurysm to increase as patient age decreases, and in their study, dissecting aneurysms were seen in eight of 11 patients less than two years old¹. Other studies have suggested the possibility that some neonatal spontaneous dissecting aneurysms are caused by traumatic stress during the birth process^{8,19,24}. These aneurysms often occur around the tentorial incisura, and this may be one reason most dissecting aneurysms are seen in the posterior circulation in very young children²⁵.

Dissecting aneurysms cause ischemia, SAH, or both. In SAH cases, rebleeding usually occurs within 24 hours of the initial bleed²⁶. These tendencies are also seen in pediatric ruptured dissecting aneurysms^{1,27}, and early procedures confer good outcomes¹². Mural hematoma as a common pathway is the most important factor in the promotion of thrombosis of dissecting intracranial aneurysms². Therefore, procedures such as parent-artery occlusion that promote mural thrombosis are often required for the treatment of dissecting aneurysms. However, dissecting aneurysms occasionally disappear because of progressive thrombosis of the aneurysmal sac or parent-artery lumen^{2,21}. In adults, 18.3% of intracranial arterial dissections showed complete normalization between 15 days and two months following presentation with symptoms on neuroimaging follow-up, but these are non-ruptured cases²¹. Spontaneous thrombosis of a pediatric dissecting aneurysm is not uncommon; 13.6% of dissecting aneurysms undergo spontaneous complete thrombosis and require no further treatment¹. Certainly intracranial arterial dissection frequently causes severe problems, but most dissecting lesions will be thrombosed sometime during their repair and healing process regardless of whether they were treated or not²¹. Early aggressive procedures are suggested for pediatric ruptured dissecting aneurysms¹². However, when they must be treated conserva-

Figure 5 Two-year follow-up DSA right (A,B) and left (C,D) ICA injection anterior and lateral views showing complete occlusion of the aneurysm and left ACA recanalization. E) DSA left ICA injection oblique view of the left A1 confirming its tangled course. ACA, anterior cerebral artery; DSA, digital subtraction angiogram; ICA, internal carotid artery.

tively because of unavoidable circumstances, it is important to know in which period spontaneous thrombosis can occur and what will happen afterwards. In cases of rupture, a rapid thrombotic process often begins within a few days after rupture², but thrombosis sometimes requires more time; for example, as long as seven months^{1,2}. However, one report demonstrated recanalization six months after spontaneous thrombosis of an infantile ruptured aneurysm²⁸, and another reported recanalization three weeks after spontaneous thrombosis of a ruptured ACoA aneurysm in a five-month-old infant⁹. Our case also showed the repetition of recanalization and thrombosis in the dissecting aneurysm before and after endovascular treatment. The variety of aneurysmal substance characteristics brought unique radiological changes during the perioperative and follow-up periods that can be explained by blood coagulation and fibrinolytic response. That is, the blood repeats the coagulation and fibrinolysis processes in the pseudolumen or dissecting aneurysmal sac. This response is considered to represent part of the repair and healing processes of the dissecting vessel walls or aneurysms²⁹. If the aneurysm has thrombosed once, recanalization can occur because of transiently intensified fibrinolysis. Therefore, close follow-up is advised because early recanalization is not uncommon⁹.

The imaging modalities used in pediatric cases require special consideration because of the negative effects of too much radiation and the necessity for sedatives. In the acute phase, the condition of a dissecting aneurysm changes daily, so it is important to characterize it accurately with neuroradiological imaging. Following this, children with intracranial aneurysms require a long period of follow-up because of their long remaining life. Therefore, an easy

and noninvasive imaging modality is suitable for children. For surveillance and treatment of pediatric aneurysms, CTA or cerebral angiography are often performed during the acute phase. This is often followed by MRA, which is one of the best imaging modalities for pediatric aneurysms because it provides high-resolution images and is less invasive^{11,12,30-32}. However, MRA constructed with the time-of-flight method is strongly affected by speed of blood flow. Turbulent flow or slow flow creates a low-intensity signal and can result in a false-negative MRA. In addition, subacute hematoma or fat appears as a high-intensity signal on T1-weighted images as well as on MRA (false positive). When an image is suspicious for a false negative or false positive, additional sequences or contrast-enhanced MRA are useful for determining the aneurysmal condition³³. Van Raay et al. reported the usefulness of transfontanel cranial ultrasonography, especially in neonatal cases, and claimed that it should be the first-line investigation in this population²². Doppler technique is also useful for investigating the condition of the aneurysm and surrounding vessels²². In infant cases, ultrasonographic modalities can be very useful in determining treatment. There is no consensus on how often or for how long pediatric aneurysm patients should be followed after acute-phase treatment. Recurrence of aneurysm and the development of de novo aneurysms after the initial procedure have been reported in pediatric cases, and these phenomena are highly likely to be related to dissection or fusiform morphology^{32,34}. The incidence of recurrent and de novo aneurysms was reported by Kakarla et al. to be 2.6% and 7.8% over a mean follow-up duration of 53 months³², and Hetts et al. reported that 8.4% developed de novo or enlarging aneurysms an average of 4.2

years after initial presentation³⁴. Moreover, Sannai et al. reported that aneurysm recurrence or the development of de novo aneurysms was frequent in children treated endovascularly during a mean follow-up period of 5.7 years³¹. Therefore, some reports have proposed that cerebral angiography be performed six months after the endovascular treatment and if there is no evidence of recanalization, then annual MRA can be done and its frequency eventually decreased to once every five years^{11,30}. In our case, kaleidoscopic neuroradiological changes were observed within the first six months, and we agree with the follow-up guidelines proposed by the above authors.

Careful neuroradiological surveys are needed for pediatric dissecting aneurysms treated endovascularly compared with adult or surgical clipping cases.

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Conclusions

The incidence of ruptured aneurysm in infants is extremely low. We have presented the long-term outcome of a case of infantile dissecting ACoA aneurysm treated endovascularly. Early aggressive treatment with procedures such as parent-artery occlusion must be performed to promote mural thrombosis for ruptured dissecting aneurysms. In our case, the recurrence of thrombosis and recanalization before and after endovascular treatment were detected because of radiological changes. Blood coagulation and fibrinolytic response are considered to represent part of the repair and healing processes of the dissecting vessel walls or aneurysms. Careful neuroradiological surveys are needed for pediatric dissecting aneurysms treated endovascularly.

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