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Case Report Open Access

Spontaneous Thrombosis of a Ruptured Pial Arteriovenous Malformation and an Associated Large Intranidal Aneurysm with Perianeurysmal Edema in an Infant: A Case Report

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Abstract

The authors reported an extremely rare case of complete spontaneous thrombosis of a pial arteriovenous malformation associated with a large intranidal aneurysm in an infant. A 10-month-old infant experienced nocturnal seizure witnessed by her mother. Computed tomography scan and magnetic resonance imaging (MRI) of the brain disclosed a ruptured intranidal aneurysm resulting in acute hemorrhage in left anterior interhemispheric subdural space with extension along posterior interhemispheric space, tentorium, bilateral tentorial cerebelli, and hemispheric convexities. MRI and magnetic resonance angiography (MRA) showed a pial arteriovenous malformation (AVM), at medial side of the left superior frontal gyrus, associated with a large partially thrombosed aneurysm with perianeurysmal edema. Cerebral angiography confirmed a small pial AVM arising from left middle internal frontal branches of the left anterior cerebral artery (ACA) with early venous drainage into medial frontal veins and forward to the superior sagittal sinus. Subseletive injection of the left ACA injection clearly demonstrated multiple indirect small feeders, unsuitable for endovascular treatment. Sequential MRI of the brain revealed the evolution of a large partially thrombosed intranidal aneurysm with surrounding parenchymal edema. Follow-up MRI and contrasted MRA, obtained at the age of 7 years, demonstrated complete obliteration of the pial AVM and large thrombosed intranidal aneurysm. At the age of 11 years, control angiography confirmed total disappearance of the pial arteriovenous malformation and a large thrombosed intranidal aneurysm. The factors associated with spontaneous regression of a ruptured pial AVM and an associated intranidal aneurysm in the present case was the presence of intracranial hemorrhage and small nidus.

Keywords: Spontaneous thrombosis; Regression; Infant brain arteriovenous malformation; Partially thrombosed aneurysm; Perianeurysmal edema

Introduction

Pial arteriovenous malformations (AVMs) are uncommon in children aged less than 2 years. The physiology, type, and architecture of pial AVMs are closer to those in adults after 5 to 7 years of age [1]. In general population, including mainly adults and some children, the prevalence of spontaneous regression of brain AVMs was 0.3%-0.8% [2-4]. Spontaneous thrombosis of pediatric AVMs is extremely rare, especially in infant [3,5]. Spontaneous regression of infant AVMs has been previously reported in only one case [6]. We describe another infant with spontaneous thrombosis of a ruptured pial AVM associated with a large intranidal aneurysm. Long-term sequential images are also clearly demonstrated in our case. We also review literature of spontaneous regression of pediatric AVM in patients aged less than 5 years, and discuss about the possible mechanism of these rare events.

Case Report

A 10-month-old female infant was admitted to community hospital near her hometown following a seizure during sleep at night. Her mother witnessed the seizure, starting by screaming, eyes rolling up, and then generalized seizures lasting for 2 minutes. There was no fever and history of trauma. She was born at term, weighting 3.080 gm. Computed tomography (CT) scan of the brain showed an enhancing hyperdense round mass, approximately 15 mm in size, with surrounding edema at medial aspect of the left superior frontal gyrus. There was acute thin subdural hematoma (SDH) located at left side of the anterior interhemispheric fissure adjacent to the medial part of the mass (Figure 1). Despite administration of antiepileptic drug, she still had multiple episodes of seizure. She was intubated and transferred to intensive care unit. The provisional diagnosis was a ruptured aneurysm caused acute SDH with a differential diagnosis of hemorrhagic tumor. Magnetic resonance imaging (MRI) of the brain, obtained one week after symptom onset, clearly revealed thin SDH extending posteriorly along posterior interhemispheric fissure, posterior falx, bilateral tentorial cerebelli, and enveloping bilateral hemispheric convexities, more visible on the left side. There was a tangle of abnormal flow voids, probably representing a pial AVM, at medial side of the left superior frontal region. The left anterior interhemispheric SDH adjoined medial side of the large round mass, probably indicating the source of hemorrhage from ruptured aneurysm. T1-weighted gadolinium-enhanced MRI demonstrated peripheral rim enhancing round mass with an eccentrically located

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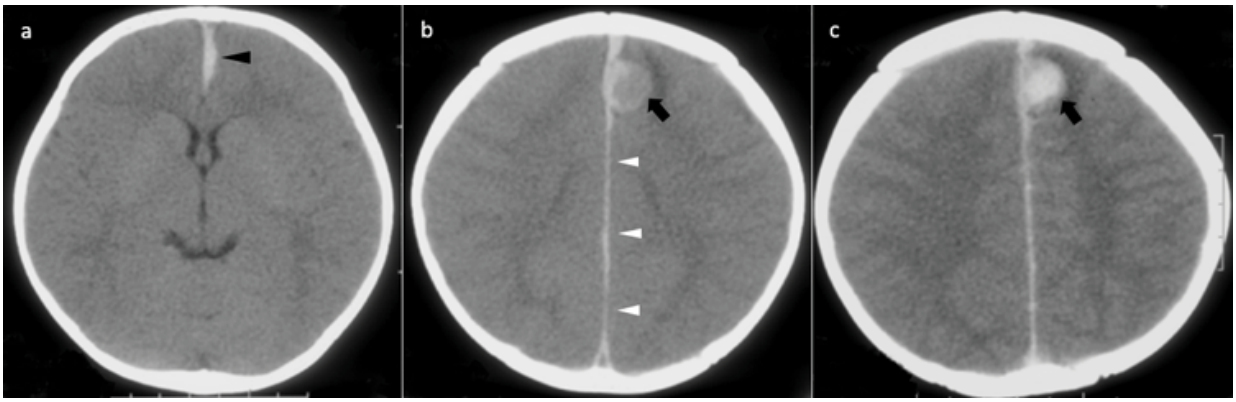

Figure 1: (a,b) Pre-contrast and (c) post-contrast axial computed tomography scans of the brain showed an enhancing hyperdense round mass (arrows), approximately 15 mm in size, with surrounding edema at medial aspect of the left superior frontal gyrus. There was small hyperdense lesion (black arrowhead), located at left side of the anterior interhemispheric fissure adjacent to the medial part of the mass, extending posteriorly along interhemispheric fissure (white arrowheads), probably representing acute interhemispheric subdural hemorrhage.

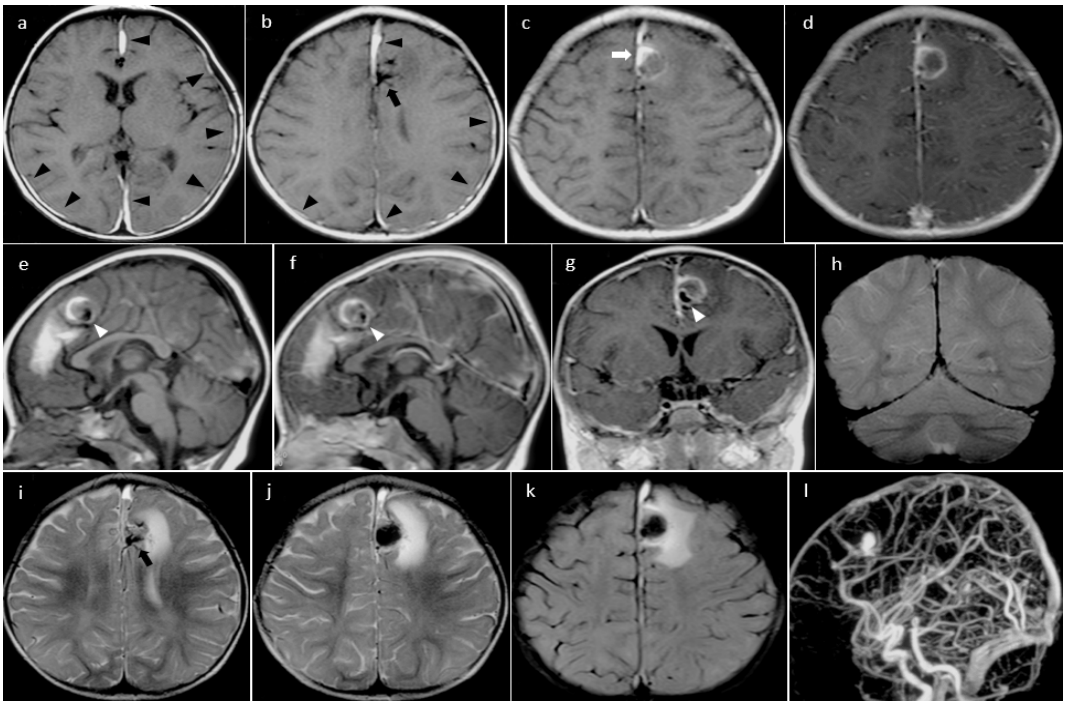

Figure 2: One week after symptom onset. (a,b,c) Axial and (e) sagittal T1-weighted magnetic resonance imaging (MRI) of the brain showed thin subdural hematoma (SDH) (black arrowheads) along left anterior, posterior interhemispheric space, and bilateral hemispheric convexities, more visible on the left side. There was a tangle of abnormal flow voids (black arrow), probably representing pial arteriovenous malformation (AVM), at medial side of the left superior frontal region. The left anterior interhemispheric SDH adjoined medial side of the large round mass (white arrow), probably representing the source of hemorrhage from ruptured aneurysm. (d) Axial, (f) sagittal, and (g) coronal T1-weighted gadolinium-enhanced MRI revealed peripheral rim enhancing round mass with an eccentrically located signal void (white arrowheads), probably indicating of a large partially thrombosed aneurysm. (h) Coronal gradient-echo (GRE) MRI also showed diffuse thin SDH along falx cerebri and bilateral tentorial cerebelli. (i,j) Axial T2-weighted and (k) fluid-attenuated inversion recovery (FLAIR) MRI also demonstrated a small pial AVM (black arrow) associated with a large aneurysm with progression of perianeurysmal edema at the left superior frontal gyrus. (l) MR angiography of the brain confirmed the pial AVM with a large intranidal aneurysm.

signal void, compactible with a large partially thrombosed aneurysm. Gradient-echo (GRE) MRI also revealed diffuse thin SDH along falx cerebri and bilateral tentorial cerebelli. T2-weighted and fluid-attenuated inversion recovery (FLAIR) MRI demonstrated progression of perianeurysmal edema at the left superior frontal gyrus. Magnetic resonance angiography (MRA) of the brain confirmed the pial AVM with a large intranidal aneurysm (Figure 2). The infant was admitted for 3 weeks until clinically stable, and then referred to Prasat Neurological Institute for endovascular treatment. The physical examination showed mild developmental delay, closure of the anterior fontanel, and normal vital signs. Her head circumference was 44 cm. Her weight and length were 7.5 kg and 70 cm, respectively. On neurological examination, there were no signs of meningeal irritation, papilledema, or other neurological abnormality. Follow-up MRI obtained one month after symptom

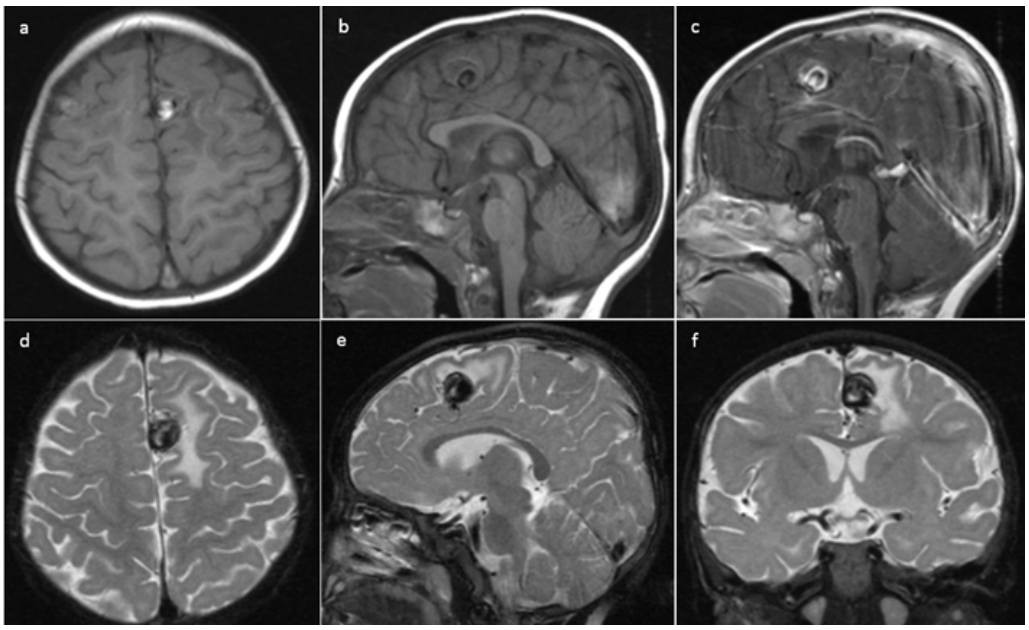

Figure 3: One month after symptom onset. (a) Axial T1-weighted magnetic resonance imaging (MRI) showed abnormal hyperintensity in an aneurysm, probably representing intraluminal thrombus. (b) Sagittal nonenhancing, and (c) enhancing T1-weighted MRI revealed heterogeneous signal intensity with eccentrically rim enhancing lesion, confirming a laminated appearance of partially thrombosed aneurysm. (d) Axial, (e) sagittal, and (f) coronal T2-weighted MRI also exhibited heterogeneous signal in a large aneurysm surrounding edema of the left superior frontal gyrus.

onset, revealed complete resolution of SDH, and heterogeneous signal intensity on T1 and T2-weighted with eccentrically rim enhancing lesion, corresponding to a laminated appearance of partially thrombosed aneurysm. Perianeurysmal edema of the left superior frontal gyrus had still remained (Figure 3). Cerebral angiography also confirmed a small pial AVM associated with large intranidal aneurysm arising from left middle internal frontal branches of the left anterior cerebral artery with early venous drainage into medial frontal veins and forward to the superior sagittal sinus. According to the Spetzler-Martin grading system, this pial AVM was assessed as grade I. There was a saccular aneurysm, approximately 10 mm in size, located within the small nidus. Subseletive injection of the left ACA clearly demonstrated a small nidus, multiple indirect small feeders, the large intranidal aneurysm, and early draining veins (Figure 4). Due to small indirect feeders, the microcatheter could not navigate to the nidus or intranidal aneurysm. Therefore, the microsurgical resection was informed for this patient, but her parents refused consent to this option. Follow-up MRI obtained 5 months later disclosed significant reduction in size of a large partially thrombosed intranidal aneurysm and complete resolution of perianeurysmal edema at the left superior frontal gyrus (Figure 5). An electroencephalogram (EEG) was interpreted as focal epileptic basis over the left parieto-occipital regions. Annual EEG has been performed and showed similar patterns. Therefore, antiepileptic drug was used continuously in this patient. Follow-up MRI and contrasted MRA, obtained at the age of 7 years or 6 years after symptom onset, confirmed complete obliteration of the pial AVM and large intranidal aneurysm (Figure 6). During annual follow-up, the patient had normal development and no recurrent seizures. She has learned and achieved average level of her classroom. Follow-up cerebral angiography, obtained at the age of 11 years, still confirmed disappearance of the pial arteriovenous malformation and large thrombosed intranidal aneurysm (Figure 7).

Discussion

The most common presentation of pediatric cerebral AVMs, often more than 60%, was hemorrhage. From literature review, most pediatric patients diagnosed with ruptured brain AVMs are in their adolescence, rarely found in patients under the age of 8 years and scarcely in infancy [7,8]. According to aged-related classification of pediatric cerebral vascular malformations, infant pial AVMs are more often fistulous than glomerular [9]. The annual hemorrhage rate of pediatric ruptured brain AVM is within a range of 2%-3%. Any deep venous drainage and associated aneurysm increase the risk for future bleeding [10].

Spontaneous interhemispheric and/or tentorial SDH without subarachnoid hemorrhage caused by ruptured aneurysms of ACA or internal carotid-posterior communicating artery has been previously reported [11-15]. Being forced to extend over the convexity surface in the narrow interhemispheric space, interhemispheric SDH from ruptured distal ACA is usually continuous with a convexity SDH [15]. In addition, continuity between interhemispheric and tentorial SDH may indicate a ruptured internal carotid-posterior community aneurysm [12]. There are two possibilities for explaining the occurrence of interhemispheric or tentorial SDH secondary to a rupture aneurysm. Firstly, it is proposed that a prior sentinel bleed causes inflammation and adhesion between aneurysm and arachnoid membrane with subsequent bleeding through the local tear of membrane into the subdural space. Secondly, a ruptured aneurysm may bleed directly under high pressure into the subdural space through ruptured pia-arachnoid mater. In the present study, the images demonstrated left interhemispheric SDH extending posteriorly along the course of falx cerebri to posterior interhemispheric space, inferiorly spreading over bilateral tentorium cerebelli, and then laterally enveloping bilateral convexities. Undoubtedly, the large intranidal aneurysm was the likely source of the hemorrhage in our case. The nidus was supplied by branches of the left ACA. However, this type of hemorrhagic manifestation is uncommon. As previously

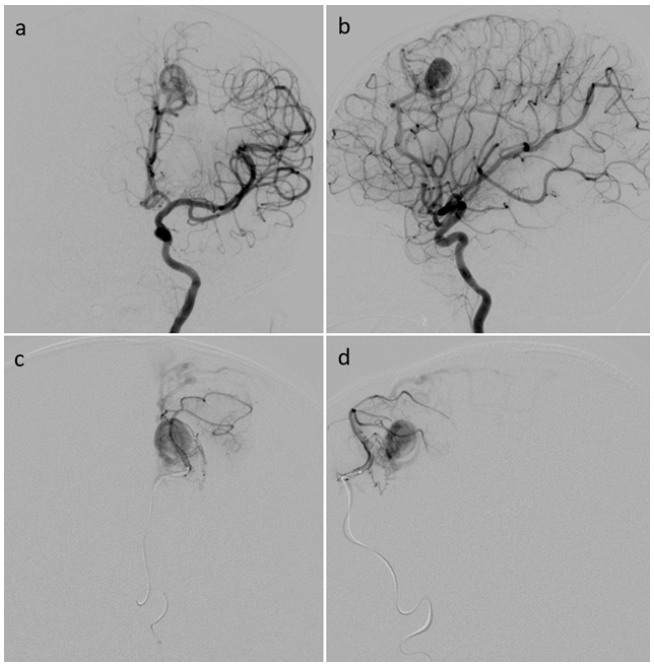

Figure 4: (a) Anteroposterior and (b) lateral views of the left internal carotid artery injection revealed the small pial arteriovenous malformation, supplied by left middle internal frontal branches of the left anterior cerebral artery with early venous drainage into medial frontal veins and forward to the superior sagittal sinus. A saccular aneurysm, approximately 10 mm in size, was located within the small nidus. (c) Anteroposterior and (d) lateral views of the left ACA injection with microcatheter clearly demonstrated a small nidus, multiple indirect small feeders, the large intranidal aneurysm, and early draining veins.

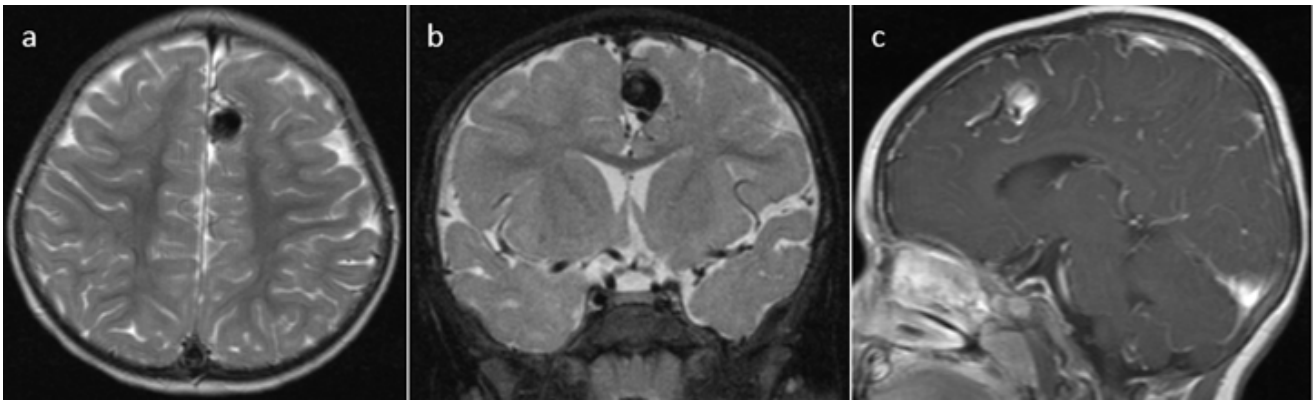

Figure 5: (a) Axial and (b) coronal T2-weighted magnetic resonance imaging (MRI) of the brain, obtained 7 months after symptom onset, showed reduction in aneurysm size and resolution of perianeurysmal edema at the left superior frontal gyrus. (c) Sagittal T1-weighted gadolinium-enhanced MRI also revealed significant reduction in size of a large partially thrombosed aneurysm.

mentioned, a large intranidal aneurysm may adjoin medial side of an arachnoid membrane and bleed directly into subdural space, resulting from increased vascular pressure.

The principal aim of the treatment in pediatric patients with ruptured brain AVMs are complete obliteration of the AVMs, preserving or restoring neurological function, and preventing the risk of re-hemorrhage. Treatment options of pediatric ruptured brain AVM are surgery, embolization, radiosurgery, or by a combination of these modalities. However, microsurgical AVM resection alone remains the treatment of choice for the management of pediatric cerebral AVMs, especially in low-grade (Spetzler-Martin I–II), with excellent outcomes. Endovascular treatment and radiosurgery should be considers as an adjunctive treatment [7,8,16]. However, from the study of long-term hemorrhagic risk of cerebral AVMs in children and adults by Fullerton et al. [17], they found that children were at lower risk of subsequent bleeding compared with adults. Therefore, they suggested that pediatric brain AVMs should be treated less aggressive than those in adults.

AVMs associated with aneurysm have higher risk of rebleeding than isolated AVMs. AVM-associated aneurysms can occur in 29% of pediatric AVMs. Their locations are subdivided into arterial, intranidal, or venous site. The presence of an aneurysm in an arterial location is strongly related to hemorrhage. AVMs associated aneurysm should not be treated in delayed fashion [7]. In our patient, the pial AVM associated with a large intranidal aneurysm was found to be unsuitable

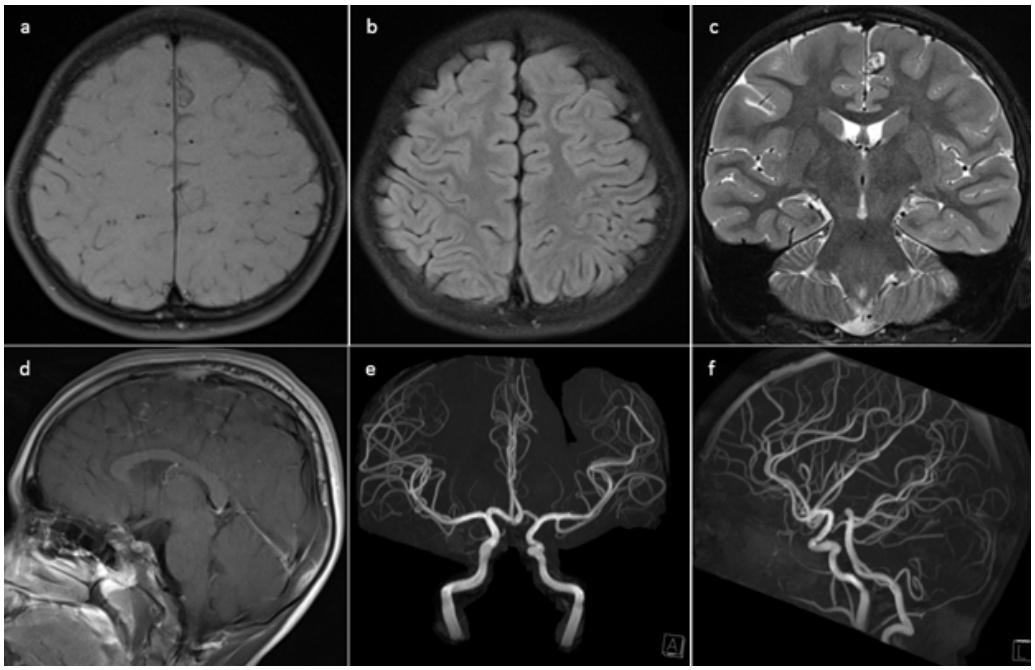

Figure 6: At the age of 7 years, (a) Axial T1-weighted, (b) fluid-attenuated inversion recovery (FLAIR), (c) Coronal T2-weighted, and (d) Sagittal T1- weighted gadolinium-enhanced magnetic resonance imaging (MRI) of the brain confirmed complete thrombosis of the pial arteriovenous malformation (AVM) and large aneurysm with disappearance of perianeurysmal edema. (e) Anteroposterior and (f) lateral views of time-of-flight (TOF) MR angiography also demonstrated complete obliteration of the pial AVM and large intranidal aneurysm.

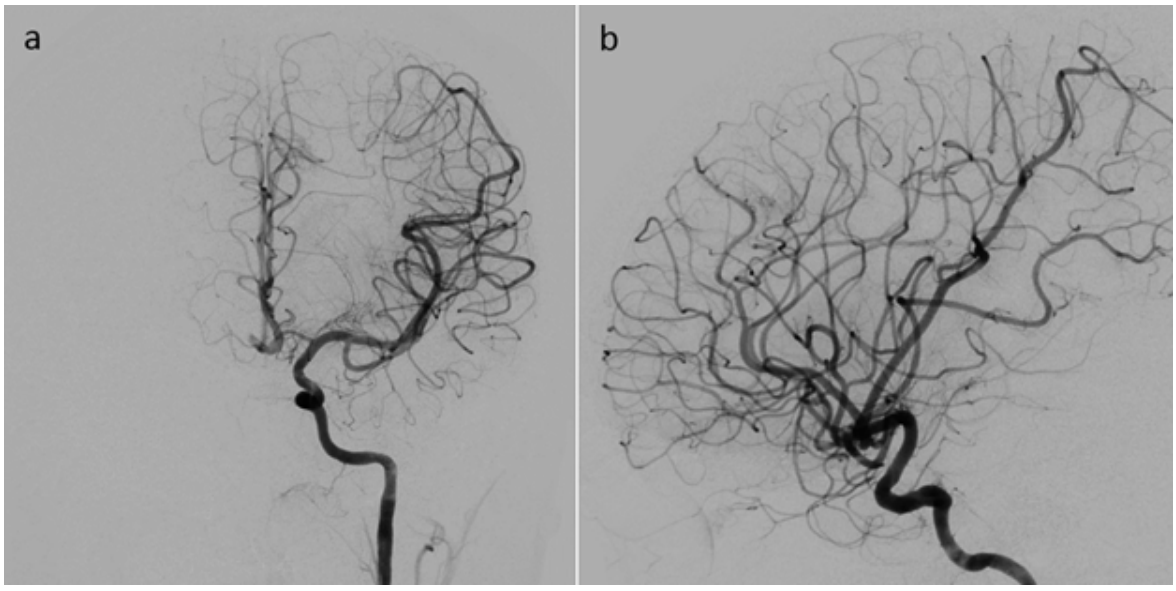

Figure 7: At the age of 11 years. (a) Anteroposterior and (b) lateral views of the right internal carotid artery injection confirmed disappearance of the pial arteriovenous malformation and a large thrombosed intranidal aneurysm.

for endovascular treatment. The infant was left untreated with surgical resection due to the decision making of her parents. However, long-term follow-up of this patient has been continued annually. Sequential images were obtained until total thrombosis of the AVM and large intranidal aneurysm occurred.

Spontaneous regression of pediatric AVMs, especially less than 5 years, is extremely rare [3,5]. Since 1977, Dyck [18] reported spontaneous thrombosis of a brain AVM in 4-year-old girl, manifested with seizure. Operative findings showed the left parietal AVM supplied by distal branches of ACA with multiple distended and thrombosed venous channels without the evidence of hemorrhage. Extensive intravascular thrombosis was confirmed by histopathological examination. It was proposed that propagation of the thrombus resulting from elongation and tortuosity of the lesion due to intravascular turbulence. Many years later, Leung et al. [19] reported spontaneous regression of cerebellar AVMs in 4-year-old girl with Hereditary Hemorrhagic Telangiectasia

(HHT). The angioarchitecture of this AVM was small nidus, single draining vein and no presence of hemorrhage.

To the best of our knowledge, there was only one previous report of spontaneous disappearance of infant cerebral AVM by Mabe and Furuse [6]. During a 7-month period after first angiography, they reported spontaneous regression of a large cerebellar AVM in a 4-month-old infant, manifested with impaired development and increased head circumference from hydrocephalus. Postoperative subdural hematoma following ventriculoperitoneal shunt may be the only predisposing factor.

Based on literature reviews, mainly in adult, predisposing factors of spontaneous regression of the brain AVMs are small nidus, hemorrhagic presentation, and single draining vein. The most common factor associated with spontaneous thrombosis of AVMs is the presence of a single drain vein. The hematoma and/or edema probably compressed the draining vein, resulting in partial or complete occlusion of the vein, leading to venous stasis and thrombus propagation into the nidus of AVM. Venous thrombosis of the main draining veins is probably the imperative factor for spontaneous regression of brain AVMs [2-5]. In the present study, the factors associated with spontaneous thrombosis of this brain AVM were the presence of intracranial hemorrhage and small nidus. Interestingly, sequential MRI in our case clearly demonstrated thrombus formation beginning within a large intranidal aneurysm until complete thrombosis occurred.

Perianeurysmal edema, vasogenic edema in the white matter surrounding an intracranial aneurysm, is increasingly reported as an early sign in the course of aneurysmal rupture. It can occur spontaneous or following endovascular treatment in small to giant aneurysm. Inflammatory process in the remodeling aneurysmal wall, mass effect, and hemodynamic stress may play an imperative role in the edema formation. The edema might be associated with a regrowth of the aneurysm [20-23]. The presence of partial thrombosis and the volume of intracranial aneurysm may be significant risk factors for the formation of perilesional edema. Metabolic factors, i.e. thrombin and degradation products of hemoglobin, secreted from thrombosed parts of aneurysmal wall may diffuse into surrounding brain parenchyma and subsequently induce perianeurysmal edema [24]. In our case, it was unclear that perianeurysmal edema occurred whether preceding or following the rupture of the large intranidal aneurysm. However, in our case, brain edema around partially thrombosed large aneurysm had completely regressed spontaneously in the period of 7 months.

Recanalization of previously thrombosed AVMs has been previously reported in few adult patients [25,26]. Nukui et al. [25] reported partial recanalization of the occipital AVM demonstrated 16 months following spontaneous thrombosis. Mizutani et al. [26] also reported total recanalization of the frontal AVM in an adult illustrated by follow-up angiography 31 months after complete spontaneous thrombosis. To confirm complete cure of the AVMs, they suggested that follow-up angiography should be performed for at least 3 years. Neovascularization within a thrombosed AVM may lead to lesion recanalization, proven by immunohistochemical analysis of a thrombosed AVM [4]. Due to the possibility of recanalization of totally thrombosed AVMs, the serial imaging follow-up, i.e. MRA or angiography, is mandatory [2,3,26]. Deep venous drainage and young age less than 20 years are imperative factors increasing the risk of brain AVM recurrence after resection [27]. Therefore, a long-term imaging follow-up is recommended in pediatric patient. However, to assess the stability of complete obliteration of cerebral AVM in children, control angiography should be the modality of choice instead of MRI [1]. In the present case, complete cure of pial AVM and large thrombosed intranidal aneurysm was confirmed by control angiography at the age of 11 years, 4 years after demonstrating by MRI and contrasted MRA.

Conclusion

We reported an extremely rare case of spontaneous regression of a pial AVM associated with large intranidal aneurysm. With long-term follow-up, sequential images demonstrated the evolution of a large partially thrombosed aneurysm, and confirmed total thrombosis of a large aneurysm and small pial AVM. The predisposing factors of this phenomenon in our case were hemorrhagic presentation and small nidus. This present study also revealed complete regression of perianeurysmal edema. However, spontaneous thrombosis of ruptured cerebral AVMs remains fortuitous and unpredictable occurrence; it should be avoided using this experience as an option in the management of brain AVM.

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