Case Report

Spontaneous Thrombosis and Stabilization of a Dissecting PCA Aneurysm in a Child

Vikas Bhatia, Arushi Gahlot Saini1, Rajeev Chauhan2

Department of Radiodiagnosis and Imaging, 1Pediatrics, 2Anaesthesia and Intensive care, Postgraduate Institute of Medical Education and Research, Chandigarh, India

ABSTRACT

Spontaneously dissecting posterior cerebral artery (PCA) aneurysm in the pediatric age group is a rare entity. We discuss a child with a resolution of the aneurysm due to spontaneous thrombosis in the PCA.

KEYWORDS: Dissecting aneurysm, PCA, thrombosis

INTRODUCTION

Pediatric intracranial aneurysms are rare and constitute 2–6% of all aneurysms. These are characterized by a male preponderance, predilection for the carotid artery bifurcation and posterior circulation, a higher incidence of large aneurysms, and a higher incidence of spontaneous thrombosis as compared with adults. Most pediatric aneurysms have predisposing factors such as infection, tumor, dissection, or trauma. The PCA aneurysms are uncommon and are seen in 0.8–1.4% of all aneurysms. Only one-tenth of these aneurysms are seen in the pediatric population. Arterial dissection is the most frequent cause of stroke in the posterior circulation in children. An intracranial dissection is seen in nearly 4% cases and is commonly related to preceding head trauma. PCA aneurysm with spontaneous intracranial dissection is anecdotally reported in children. We discuss a case with spontaneous thrombosis of such an aneurysm and its probable mechanism.

CASE

A seven-year-old girl presented with an acute, severe headache for the past four days, which was associated with intermittent, projectile vomiting. There was no alteration in sensorium, visual disturbance, seizures, fever, neck stiffness, behavioral changes, photophobia, focal motor deficit, cranial nerve palsy, or diplopia. Past and family history was unremarkable. Her general and detailed neurological examination was normal. A clinical diagnosis of an acute, severe secondary headache secondary to an underlying arteriovenous malformation or aneurysm was considered. A noncontrast computed tomography scan showed a small bleed in the right perimesencephalic cistern. Diagnostic cerebral angiography showed a contrast-filled outpouching [Figure 1A] with irregular walls [Figure 1B] and proximal small segmental stenosis, at the right PCA-P3 segment. This confirmed the diagnosis of a dissecting aneurysm in the PCA. The patient was taken up for endovascular PCA occlusion after 72 h, and selective right vertebral artery injection was administered. There was nonfilling of the aneurysm and the right PCA distal to the neck of the aneurysm [Figure 2]. A probable spontaneous thrombosis of the aneurysm and PCA was considered, and the patient was managed conservatively. She remained well in follow-up till one year.

DISCUSSION

Intracranial arterial dissection is difficult to diagnose on routine imaging. The angiographic appearance of an aneurysm with the parent vessel stenosis, and stagnation of the contrast is the hallmark of a dissecting aneurysm. Head trauma is the leading cause of intracranial dissecting

Address for correspondence: Dr. Vikas Bhatia, Department of Radiology and Imaging, Postgraduate Institute of Medical Education and Research, Chandigarh, India. E-mail: drvikasbhatia@gmail.com

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aneurysms in children, and a spontaneous dissection is unusual.[7,8] The natural history of intracranial dissections is not exactly clear. These may remain stable and progressively decrease in size, thrombose, or catastrophically lead to massive bleeds and death. Spontaneous thrombosis is reported in 1–2% of pediatric aneurysms.[4] It is seen in upto 38% cases of large or giant aneurysms and is usually seen with associated parent vessel thrombosis.[9] The proposed mechanisms include a high aneurysm sac volume to neck size ratio, presence of proximal parent artery stenosis, progression of dissection leading to proximal artery occlusion, or enlarging aneurysm compression over parent artery.[7,10] The role of contrast media used for diagnostic angiography has also been proposed, possibly due to activation of coagulation or the thrombosis mechanism.[11,12] In our case, possibly the high sac to neck ratio and the presence of low intraneurysmal flow due to proximal stenosis or progression of dissection may, in isolation or together, have caused spontaneous thrombosis of the aneurysm and the parent artery distal to the aneurysm. Thus, endovascular management in the form of PCA occlusion was not carried out and spontaneous occlusion of the PCA did not result in any neurological deficit. This was most likely due to gradual thrombosis leading to the development of collateral flow to the involved PCA territory. A review of prior large studies showing spontaneous thrombosis of pediatric dissecting aneurysms is shown in Table 1. Neurological deficit ranging from 0% to 17% has been previously reported for PCA occlusion.[13]

Table 1: Studies with pediatric cases showing spontaneous thrombosis of dissecting aneurysms.[14–18]

| Study                  | Age/ sex | Presentation          | Location        | size         | Follow up  | Outcome |
|------------------------|----------|-----------------------|-----------------|--------------|------------|---------|
| Yi-Sen Zhang et al., 2016 | 13/ M    | Asymptomatic          | PICA            | 8 × 3 mm     | 4 years    | Good    |
|                        | 18/ F    | Headache              | VA/ BA junction | 28 × 12 mm   | 2 years    | Good    |
| Päivi Koroknay-Páli, 2013 | 3 patients | Not mentioned          | Not mentioned   | Not mentioned | 30 years  | Good    |
| Jiantao Liang, 2009    | 9 years/ F | Asymptomatic          | RICA bifurcation| Large (10–25 mm) | 1–2 years | Good    |
| Dittapong Songsaeng, 2009 | 11 years/ M | Subarachnoid bleed   | Acom            | <10 mm       | 1–2 years  | Good    |
|                        | 8 years/ F | Headache              | Right MCA       | 25 × 18 mm   | 1 year     | Good    |
|                        | 1.5 years/ F | Hemiplegia            | Left MCA        | 10 mm        | 4 years    | Stable  |
|                        | 10 months/M | Subarachnoid bleed   | Left MCA        | 17 × 17 mm   | 4 weeks    | Good    |
|                        | 2 years/ F | Headache              | Left MCA        | 19 × 8 mm    | 2 months   | Good    |
|                        | 1 year/ M | Vomiting              | Right P2 PCA    | 19 × 14 mm   | 4 days     | Good    |
|                        | 2 years/ M | Subarachnoid bleed   | Basilar artery  | 25 × 15 mm   | 6 months   | Good    |
|                        | 6 years/ M | Headache              | Basilar artery  | 20 × 14 mm   | 3 months   | Good    |
|                        | 12 years/ M | Focal deficit         | Basilar artery  | 13 × 1 2 mm  | 3 months   | Good    |
| Lasjuanis, 2005        | 2 years/ M | Right hemiparesis     | Basilar tip     | N/A          | N/A       | Good    |
|                        | 1 year/ F | Headache              | ICA             | N/A          | N/A       | Good    |
|                        | 5 months  | SAH                   | ICA             | N/A          | N/A       | Good    |
|                        | 5 years/ F | Headache              | Basilar         | N/A          | N/A       | Good    |
|                        | 8 years/ F | Headache              | MCA             | N/A          | 1 year     | Good    |
**Conclusion**

Spontaneous arterial dissecting aneurysms in posterior circulation are uncommon. These have a dynamic natural history with variable and unpredictable outcomes. Spontaneous thrombosis of aneurysms has been seen; however, this outcome is highly unpredictable and, thus, requires a close interval follow-up and early management.

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**Conflicts of interest**

There are no conflicts of interest.

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