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A Case Report of an Intracranial Giant Aneurysm in a 10-Year-Old Female

Emmanuel E. Albano Jr, MD and Reynaldo Benedict V. Villamor Jr, MD, FAFN

Department of Neurosurgery, Vicente Sotto Memorial Medical Center, Cebu City

Pediatric intracranial aneurysms are rare and differ from aneurysms in adults in terms of location, etiology, natural history and management. This is a case report of giant aneurysm in a 10-year old patient presenting with symptoms of headache and vomiting. Cerebral catheter angiogram revealed a large aneurysm in the left middle cerebral artery, M1 segment. The patient underwent left pterional craniotomy, clip reconstruction of the patent artery, and aneurysmectomy. Post operatively the patient had an unremarkable course and was discharged improved after 1 week. Cerebral catheter angiogram was performed after 2 months and revealed no residual aneurysm.

Key words: pediatric, giant aneurysm, craniotomy, clip reconstruction, aneurysmectomy

Intracranial aneurysms in children are rare; 0.5% to 4.6% of intracranial aneurysms occur in patients aged 18 years or younger.[1] Aneurysms occurring in very young children and infants are exceedingly rare. Unlike their adult counterparts, pediatric aneurysms have been reported to exhibit features such as male predominance, a higher incidence in locations such as the internal carotid bifurcation and posterior circulation, and greater numbers of giant aneurysms.[2-5]

By far, pediatric aneurysms present more commonly as subarachnoid hemorrhage,[6] with peaks at ages 2-5 years and in adolescents older than 15 years.[7] However, the rate of subarachnoid hemorrhage in children is far lower than in adults.[8] This may be due to high incidence of giant aneurysms that present as space-occupying lesions rather than hemorrhage.[9]

In the Philippines, intracranial aneurysms are more common in adults rather that in children. Moreover, there has been no published data about pediatric intracranial aneurysm.

Presented here is a case of a 10 year-old female with left middle cerebral artery (MCA) giant aneurysm. This report discusses the epidemiology, signs and symptoms and management of the pediatric patient with a giant aneurysm.

The Case

This is a case of a 10-year-old female who came to the hospital with complaint of severe headache associated with vomiting. Emergency CT scan revealed a 5 cm x 4 cm heterogenous lesion in the left sylvian fissure (Figure 1). MRI revealed a 3.7 cm x 5.3 cm x 3.6 cm nodular lesion at the anterior temporal lobe characterized by prominence of cyst components with hemorrhagic contents as well as an enhancing mural nodule at its anterior margin (Figure 2). Differential diagnoses included pilocytic astrocytoma and supratentorial hemangioblastoma. However, the irregular appearance of the signal void corresponding to the left middle cerebral artery was suspicious for a vascular lesion. A cerebral catheter angiography revealed a wide neck aneurysm involving the entire length of the left middle cerebral artery. The neck measured approximately 12.28 mm with the visualized aneurysmal lumen measuring approximately 9.90 mm x 12.22 mm. A large anterior frontal artery was noted near the internal carotid artery terminal bifurcation. The lateral lenticulostriate vessels were noted proximal to the giant aneurysm. A giant left middle cerebral artery aneurysm, M1 segment was considered (Figure 3).

This patient was advised for surgical intervention. The procedure planned was a left pterional craniotomy, clip reconstruction of patent artery, and aneurysmectomy.

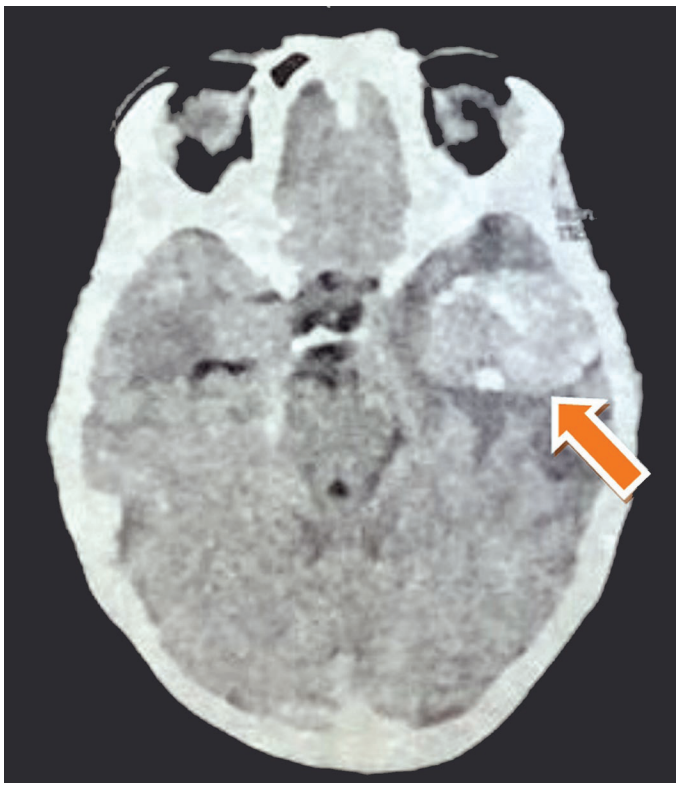

Figure 1. Computed tomography brain plain which noted a 5 cm x 4 cm heterogenous lesion (arrow) on the left sylvian fissure

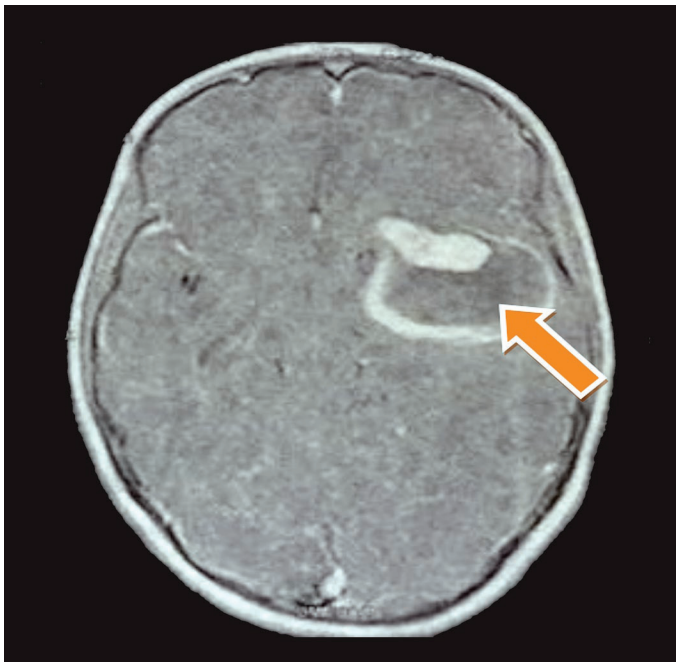

Figure 2. MRI brain (T1) with contrast noted 5.7cm x 5.3 cm x 3.6cm nodular mass lesion in the left anterior temporal lobe (arrow)

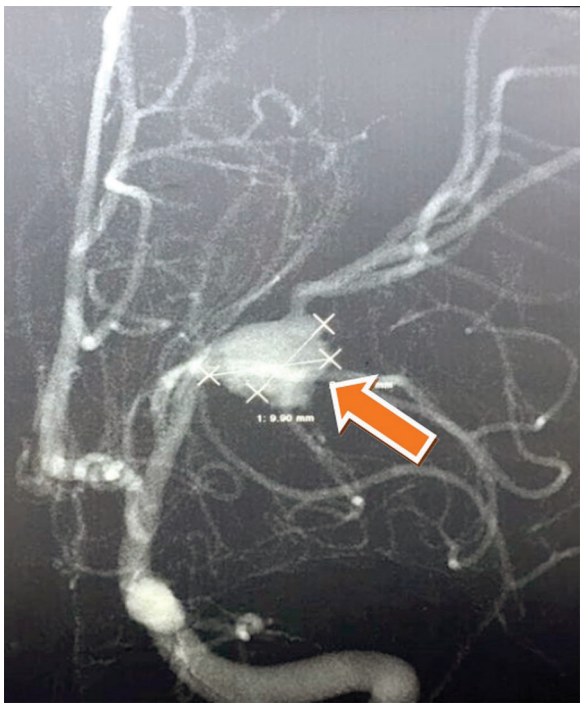

Figure 3. Cerebral Catheter Angiogram left ICA injection showing a left MCA aneurysm.

The patient was positioned supine with the head secured in three-point fixation, rotated 15°-20° away from the side of aneurysm. A curvilinear skin incision,was made 0.5 cm -1 cm anterior to the tragus and no more than 1.5 cm inferior to the zygoma. Soft tissue, myocutaneous flap and submuscular dissection followed. The pterion and lesser sphenoid wing were drilled medially toward the superior orbital fissure until a flat surface over the orbit connected the middle and cranial fossa. Multiple tacking sutures were placed on the dural flap and were pulled against pterion, leaving an unobstructed view into the carotid cistern. The sylvian fissure was dissected until the aneurysm was exposed and isolated (Figure 4).

Temporary clips were applied at the internal carotid artery and distal branch of MCA for proximal and distal control. The fundus of the unclippable aneurysm was transected. The aneurysm was opened and its neck was simplified. The neck was then reconstructed and contoured with fenestrated clips (Figure 5). The arterial patency and blood flow were assessed using intraoperative

doppler probes. The aneurysm wall (Figure 6) was sent for biopsy.

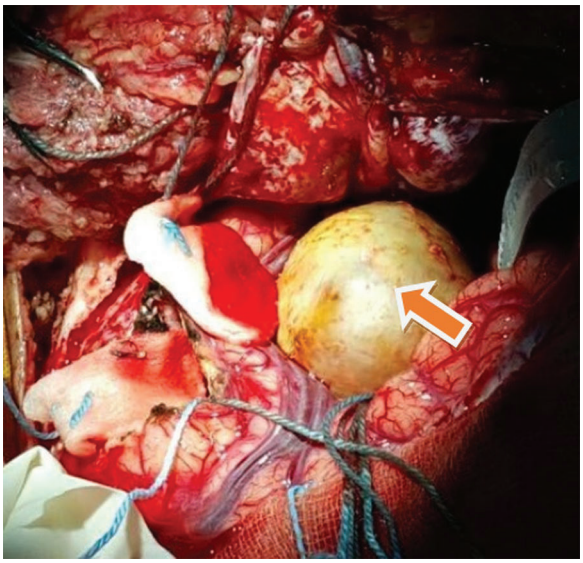

Figure 4. Left MCA aneurysm (arrow) measuring approximately 4.3 cm x 3.8 cm with a neck of 1.2 cm.

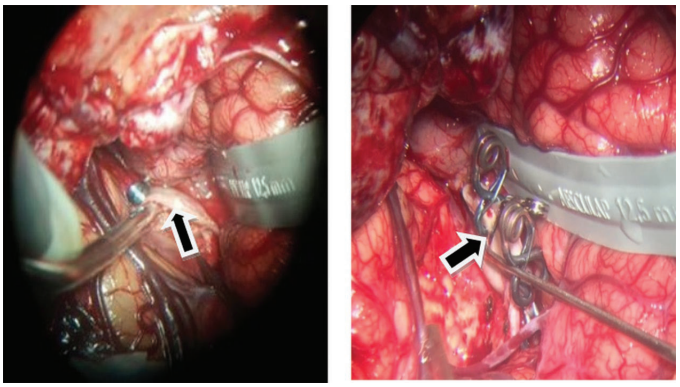

Figure 5. Clip reconstruction of left MCA aneurysm (arrow).

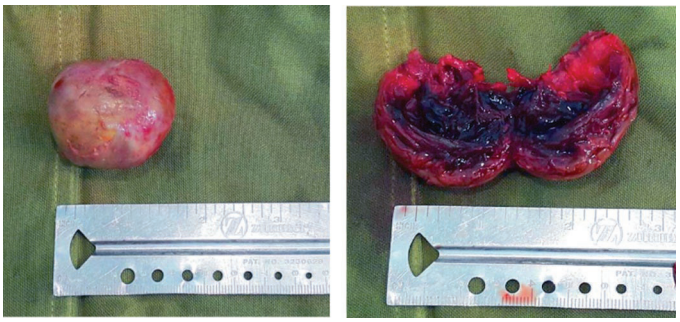

Figure 6. Left MCA aneurysm fundus.

Post operatively, the patient was extubated, and was transferred to neuro-critical care unit from post anesthesia care unit for observation. On the first post-operative day, the patient was awake and coherent. She could also follow commands. She was started on soft diet with strict aspiration precautions. On the succeeding post-operative days, no post-operative complication was noted. The patient was placed on a regular diet, was allowed to sit up on bed, and eventually was able to ambulate. On the 7th post-operative day, the patient was discharged.

On follow up, the patient was asymptomatic. A repeat cerebral catheter angiography was done 2 months later and revealed no residual aneurysm (Figure 7).

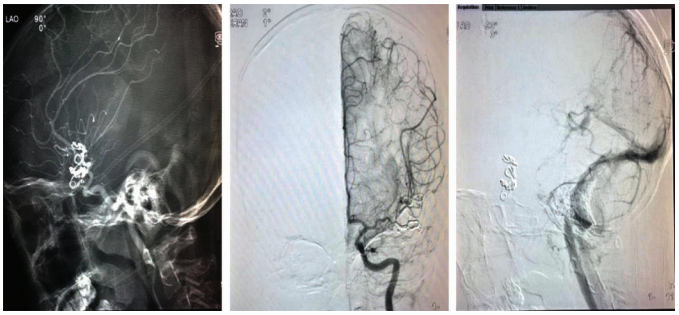

Figure 7. Repeat cerebral catheter angiography after clipping of left MCA aneurysm demonstrating no residual aneurysm.

Discussion

Pediatric intracranial aneurysms although rare, are potentially devastating. They differ from aneurysms of the adult population in location, etiology, natural history, and management.[10] Aneurysms in pediatric population also tend to be larger and have more complex shapes. Presented here is case of a 10-year-old female who had a giant intracranial aneurysm in the left middle cerebral artery (MCA). The patient presented with signs of increased intracranial pressure (headache and vomiting) due to mass effect from the aneurysm.

The age and distribution of pediatric intracranial aneurysms differ between sexes and has been reported to have a male to female ratio of 3:2,[11] though the ratio was 1:5 for children younger than 2 years. In a review of cases reported in literature by Sorteberg and Dahlberg,[12] the male:female ratio was 1.42:1. Boys show a gradual

increase in frequency with increasing age, whereas girls peak around menarche, with a female predominance at ages 14 and 15.[13] Accordingly, the patient already had her menstrual period, which probably predisposed her to the disease. In adults, aneurysm occurs twice as often in females than in males.[14]

Pediatric giant aneurysms seem to favor the vertebrobasilar arteries with reported incidences in that specific location ranging from 8-100%.[14] In the present case, the aneurysm was in the left MCA M1 segment, the second most common location, making up 16% of these lesions in the pediatric population.[12] In children aged 7-16 years old, up to 50% have aneurysms located at the first MCA branch.[12] The most common location of pediatric aneurysm is the ICA bifurcation at 33%.[14] It is five times more common in this location in children than in adults.[15]

Giant intracranial aneurysms by definition have a diameter of at least 25 mm. They are found more frequently in children than in adults.[14] The patient presented with a lesion that on MRI measured 3.7 cm x 5.3 cm x 3.6 cm. Intraoperatively, the aneurysm measured 4.3 cm x 3.8 cm and had a neck of 1.2 cm. The patient's presenting symptoms can be attributed to the humongous size of this lesion.

Subarachnoid hemorrhage (SAH) is the most common presenting symptom of intracranial aneurysms. However, the rate of SAH in children is far lower than in adult probably due to high incidence of giant aneurysms that present with mass effect. The symptoms of this mass effect depends on the location of aneurysm. In the anterior circulation, the mass effect can manifest as pain, acuity and visual field defects, and extraocular dysfunction. Dementia has also been described in anterior circulation aneurysms that are at least 3.5 cm.[16] In the posterior circulation, multiple cranial nerve dysfunctions may be present. If brainstem compression is significant, bulbar palsies and hemiparesis can also occur. Other presentations of pediatric giant aneurysms include headache, obstructive hydrocephalus, syncope, sinusitis, confusion, or ischemic stroke.[17] Seizures have been reported to be more than twice as often than in adults (36% versus 17%).[18] Likewise, acute hydrocephalus was seen more commonly in children (36%) than in adults (25%).[18]

Ruptured giant cerebral aneurysms if left untreated have a mortality rate up to 68% within 2 years and up to 85% within 5 years of diagnosis. Definitive treatment of ruptured aneurysms should be performed within 48 hours. However, a conservative or expectant approach should be considered only if there are mitigating factors such as patient's or family's refusal to treatment, poor medical grade or exceptionally high treatment risk. The overall annual rupture risk of an unruptured giant aneurysm, however, is only 0.7%.[19]

Treatment options for pediatric intracranial aneurysms depend on the capability of the neurosurgical centers and surgeons' experience. Simple aneurysms can be treated via surgical clipping or endovascular coiling. More complex aneurysms may require clip reconstruction and/or extracranial-intracranial bypass. The longer life expectancy of children is a challenge to the durability of treatment. In this respect, surgical treatment may be superior to endovascular approaches.[9]

Children treated endovascularly will need some form of additional treatment four times as often as those surgically managed.[20] It has also been observed that endovascularly treated aneurysms not only have a higher recurrence rate but also a higher rate of de novo aneurysm formation in children. The latter has been attributed to catheter manipulation causing small arterial wall defects as the potential source of aneurysm formation.[20] Endovascular technique is often unable to address the mass effect of a giant aneurysm.

In the case presented, surgical treatment was chosen as the definitive treatment. The availability of materials and an experienced vascular team allowed a smooth and successful operation.

Conclusion

Intracranial aneurysms in children differ from those in adults in location, morphology, etiology, natural history, and management. Giant aneurysms produce neurologic compromise related to mass effect. The most efficacious form of treatment involves complete elimination of this lesion from cerebral circulation. Regardless of whether these aneurysms are treated or observed, children with intracranial aneurysms require follow-up imaging and clinical surveillance, given their expected long life span

during which treated aneurysms could recur or additional aneurysms could arise.

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