Pediatric Case of Completely Thrombosed Giant Cavernous Carotid Aneurysm with Ipsilateral ICA Occlusion Mimicking an Intra-Axial Cystic Lesion: A Case Report and Review of the Literature

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Abstract

There have been a few cases where completely thrombosed cavernous carotid artery (CCA) aneurysms have resembled neoplasms based on neuroimaging data, but no reports have been documented in children. Here, we report an unusual pediatric case of a completely thrombosed giant CCA aneurysm with ipsilateral internal carotid artery (ICA) occlusion which imitates an intra-axial cystic lesion on magnetic resonance image (MRI). Therefore, we performed the surgery on suspicion of a cystic mass but it revealed a huge completely thrombosed CCA aneurysm with thrombosis of the ipsilateral ICA. The patient showed postoperatively no new neurological deficits and discharged a week later after admission.

Introduction

Aneurysms of the CCA account for between 3-5% of all intracranial aneurysms [1]. Because they are located in extradural space, the clinical manifestation is different from other intradural aneurysms. However, they can grow large in size, and can present with pressure symptoms due to mass effect on cranial nerves, such as ophthalmoplegia and facial pain [2]. Spontaneous intramural thrombosis of giant intracranial aneurysms occurs in between 13-20% of cases [3]. We describe a pediatric case of a huge cavernous sinus mass mimicking a cystic lesion with peripheral rim enhancement on MRI, where the surgery and subsequent histopathological investigation revealed that this mass was a completely thrombosed giant aneurysm of the CCA. We also searched the literature on thrombosed CCA aneurysms that mimic intracranial neoplasms.

Case Report

A 2-year-old boy with no previous medical history is admitted to us with progressive oculomotor palsy and seizure for 3 months. The symptoms began insidiously and were not associated with history suggestive of subarachnoid hemorrhage. Clinical examination revealed left ptosis, oculomotor paresis and left relative afferent pupillary defect. The patient was evaluated with the gadolinium-enhanced MRI that showed a huge axial cystic lesion adjacent to the left ICA with peripheral rim enhancement on T1-weighted and dark signal intensity on T2-weighted images that was suspicious for a giant ICA aneurysm (Figure 1). Differential diagnosis included intra-axial tumors such as glioma or ganglioglioma, dermoid cyst, epidermoid cyst, vestibular schwannoma and ependymoma. Then we performed MR Angiography, which showed giant left cavernous sinus measuring 2.7 × 2.9 × 4 cm and the occlusion of the left ICA, with no filling past the petrous segment (Figure 2). CTA also verified the findings of the MRA with, complete thrombosis of the aneurysm and lack of flow through the left ICA from the skull base to the suprarclinoid segment (Figure 3). Head CT scan also showed the direct erosion of cavernous sinus wall, and erosion into sellarurcica by the growing mass. His left middle cerebral artery (MCA) and anterior cerebral artery (ACA) were supplied through a patent Willis circuit via a left posterior communicating artery.
filling of the left MCA, ACA, and PCoA were seen. Based on the T1W MRI overlayed to the TOF-MRI a vascular (PCoA) and a patent anterior communicating artery. Reconstitution of flow at the supraclinoid segment with filling of the left MCA, ACA, and PCoA were seen. Based on the T1W MRI overlayed to the TOF-MRI a vascular

Figure 1: The gadolinium-enhanced MRI demonstrated a crescent-shaped hyperintensity of the lesion wall with no intraluminal filling of mass on T1-weighted frame and heterogenous signal intensity on T2-weighted images concerning for neoplastic process.

Figure 2: MR Angiogram shows a large thrombosed CCA aneurysm with absent flow in left ICA.

Figure 3: CT angiogram shows a lack of flow through the left ICA from the skull base to the supraclinoid segment.
aneurysm wall resection and reconstruction were performed (Figure 5).

Hystopathologic examination confirmed the diagnosis of thrombosed aneurysm, which revealed hemosiderin deposits, calcifications and inflammatory infiltrates within the remnants of the vessel wall. The origin of the lesion was considered more likely (Figure 4). Therefore, we performed subsequent craniotomy for resection of the lesion using a left pterional craniotomy the lesion exposed was found to be a large completely thrombosed aneurysm originating from the CCA. After cutting the wall of the aneurysm thrombectomy, aneurysm wall resection and reconstruction were performed (Figure 5).

Figure 4: T1W MRI images were registered to the TOF-MRI images by using 3D anatomical landmarks, and image fusion was done.

Figure 5: Intraoperative photographs displaying a huge thrombosed aneurysm. Meticulous dissection and cutting the aneurysm wall were performed.
patient showed postoperatively no new neurological deficits, postoperatively was put on aspirin for stroke prevention and discharged a week later after admission. At 1 month follow-up, he became doing well, his ophthalmoparesis and ptosis had improved.

Conclusion

Pediatric cerebral aneurysms occur at a frequency between 0.17% to 6.8% among all patients treated for intracranial aneurysms, where the percentage of pediatric cerebral aneurysms that are giant (diameter ≥ 25 mm) range from 14% to 54% [4]. Complete intraluminal thrombosis occurs especially in large and giant cerebral aneurysms in 13-20% of cases [5]. However, there are few reports where completely thrombosed CCA aneurysms have imitated neoplasms based on initial presentation and imaging [6,7], while no cases have been reported among the pediatric population. They also treated their patient with open surgery and removal of intramural clot via an aneurysmorrhaphy [8].

Most of the patients with large CCA aneurysms presented with symptoms of ophthalmoparesis, facial pain or hyposthesia due to the mass effect. In our patient, the medial extension of the mass caused the oculomotor paresis while the seizure activity is probably because of the lateral extension and compression of the insular cortex by an enlarging lesion.

CT angiography provided reliable and informative data regarding the thrombosed aneurysm, the extent of ICA thrombosis, and the distal blood flow provided by collateral circulation through the circle of Willis comparable to DSA with high sensitivity and specificity [9]. Preoperatively, a DSA is always helpful and must be included in the preoperative workup to visualize the parent vessel of the giant intracranial aneurysm and plan the operation. But in the case of a completely thrombosed aneurysm, DSA could be negative [10]. The accepted methods of treatment for giant cavernous aneurysms are occlusion of the ICA, with or without bypass, depending on the cross-flow, as well as results of balloon test occlusion [11]. Endovascular interventions for both the feeding vessel and the aneurysm itself are also done with good results and less morbidity than an extensive open procedure [12]. We also believe there is a role for conservative management in patients who are not acutely and severely symptomatic and fail balloon test occlusion [13]. Further clinical and imagiological follow-up is needed once recanalization of thrombosed lesions, although rare [14], cannot be anticipated [15]. Some authors advocate performing control angiographic studies at 3, 6, and 12 months and subsequently with yearly intervals until at least 3-year follow-up, due to the risk of recurrence and rehemorrhage [16].

Conflicts of Interest

There were no conflicts of interest during the writing or submission of this case report.

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Ethical Approval

Ethical approval was not sought for this case report as it was not required due to the nature of the case report.

Consent

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

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