Supraclinoid Internal Carotid Arterial Aneurysm Presenting as a Suprasellar Mass-like Lesion in a Child

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Summary

We present the case of a two-year and seven-month-old boy with a partially-thrombosed giant lobulated aneurysm in the supraclinoid portion of the internal carotid artery. He presented with several months of symptoms of progressive frontal headache and visual loss. CT revealed a large lobulated suprasellar mass lesion mimicking a craniopharyngioma. After the aneurysm was successfully obliterated by an endovascular procedure, regression of the giant aneurysm was confirmed on followed-up MRI. The differential diagnosis, possible etiologies, and the endovascular technique for pediatric patient will be discussed.

Introduction

Pediatric intracranial aneurysms represent less than 5% of the total number of intracranial aneurysms in the general population¹⁻³. The most frequent clinical sign in the pediatric age group is subarachnoid hemorrhage². Symptoms related to mass effect occur in about 20% of all children as the initial presenting symptom of an intracranial aneurysm³⁻⁵. Large and giant aneurysms leading to neurological deficits or seizure are more common in children than in adults and even the most frequent presenting feature in children^{6,7}.

The diagnostic work-up and therapeutic decision may be clinically challenging in pediatric patients because characteristics of pediatric aneurysms include diversity of type, predilection for the internal carotid artery and the posterior fossa, their peripheral location, and their large size^{2,5,7}. We report the case of a child with a giant multi-lobulated aneurysm mimicking craniopharyngioma, who was finally treated by endovascular exclusion of the aneurysm.

Case Report

A two-year and seven-month-old boy was referred to our hospital due to progressive frontal headache during the previous several months.

He underwent craniotomy for a large lobulated suprasellar mass detected on CT under the assumption of having a craniopharyngioma (figure 1A). Surgical resection was not possible at that time because the mass was found to be a huge aneurysm. MRI performed following surgery showed the aneurysm to have the same features and to be of the same size and shape as was detected on the preoperative to previous CT scans.

At the time on referral, the child weighed 13 kg. He spoke and walked normally without any neurological deficits. Serologic examinations for autoimmune disease and infection, includ-

Figure 1 A two-year and seven-month-old child presenting with frontal headache and progressive visual loss. A) Enhanced CT scan shows a large lobulated enhancing mass in the suprasellar area projecting left laterally. There is a non-enhancing thrombotic portion medially. B) Anteroposterior (C) lateral views of the left internal carotid arteriogram reveal a giant lobulating aneurysm in the supraclinoid portion. There is also a localized dilatation in cavernous portion of the internal carotid artery. D) Anterior and (E) posterior views of the 3D angiogram clearly show the origin of the neck of the aneurysm above the ophthalmic artery. The broad neck of the aneurysm encircling almost all the luminal wall of the internal carotid artery, is well-demonstrated on the posterior view.

ing fungus, herpes virus, and HIV, were all negative. There was no history of trauma. Ophthalmologic examination was normal except that his left vision was decreased.

Cerebral angiogram revealed a lobulated giant aneurysm in the supraclinoid portion of the left internal carotid artery (figure 1B-E). The aneurysm had three lobulations projecting anterolaterally at 5 mm above the origin of the ophthalmic artery. The non-enhancing portion caused by partial thrombosis of the aneurysm was noted in the largest and medially located aneurysmal sac on CT (figure 1A). The thera-

peutic decision was made to sacrifice the internal carotid artery, including the neck of the aneurysm, with a balloon because the neck was broad and unidentifiable even on rotational angiograms (figure 1D,E). Embolization was performed as a second session. 4F sheaths were introduced through both femoral arteries. Using a modified technique⁸, a No 16 gold-valve balloon was preloaded within a 4F guiding catheter after control angiography with a 4F diagnostic catheter and introduced into the left internal carotid artery. Contralateral injection during temporary occlusion of the ipsilateral

internal carotid artery, showed symmetric venous drainage in the left side of the brain. After detachment of the balloon at the proper position, coils were used for occlusion of the proximal internal carotid artery including the origin of the ophthalmic artery (figure 1F). Post-procedural angiogram showed no further filling of the aneurysm (figure 1G). The child's headache disappeared after procedure and his vision improved slowly and progressively. Follow-up MRI revealed the markedly decreased size of the aneurysm (figure 1H).

Discussion

Although the frequency of incidence of giant aneurysm is variable, according to the literature, the presenting feature of mass effect is one of the important manifestations of aneurysm in pediatric patient⁹. In this report we presented a child with giant supraclinoid aneurysm of the internal carotid artery, who showed a typical mode of presentation mimicking a suprasellar mass lesion, such as craniopharyngioma. Correctly diagnosing this condition from a suprasellar mass, especially a craniopharyngioma, is very important in this age group.

It is difficult to differentiate aneurysm from other suprasellar lesions because of the usual presenting symptomatology of headache and loss of vision. Although craniopharyngiomas are known to exhibit three basic characteristics

Figure 1 F) Successful embolization of the internal carotid artery as well as the neck of the aneurysm, was performed with balloon and coils. G) Post-embolization angiogram shows complete occlusion of the left internal carotid artery without filling of the aneurysmal sac. H) Follow-up MRI reveals the much decreased size of the aneurysm.

i.e. calcification, cyst(s), and contrast enhancement on CT¹⁰, such findings can also be seen in aneurysms¹¹. MR is superior to CT for delineating distortions of the optic chiasm and other suprasellar structures, for demonstrating the status of the carotid artery, and for differentiating them from aneurysms by flow void and heterogeneous increased signal intensity in areas of slower turbulent flow¹².

Traumatic origin is common in this age group although there was no history of trauma in our patient². Traumatic aneurysms typically arise at the skull base or from distal anterior, middle or posterior cerebral arteries resulting from direct mural injury or acceleration-induced shear. Reported traumatic aneurysms account for 14%-39% of all pediatric aneurysms.

Pediatric traumatic cerebral aneurysms may present early or late after trauma¹³. Most present early with intracranial hemorrhage. Late presentation occurs infrequently, and typically as an aneurysmal mass. Spontaneous thrombosis of a traumatic aneurysm is uncommon although the tendency in children toward thrombosis of a non-traumatic aneurysm is more common than in adults^{6,14}.

Mycotic aneurysms caused by subacute or chronic infection or even in the absence of bacterial endocarditis, can cause cranial nerve compression from an enlarging aneurysm although the most common presentation of mycotic aneurysm is subarachnoid hemorrhage or intracranial hemorrhage due to rupture of the aneurysm¹⁵. Mycotic aneurysms tend to be located peripherally in the cerebral vessels and most often involve the middle cerebral artery. Antibiotic therapy is curative in most cases. There is also a demonstrated association between intracranial aneurysms and immunodeficiency with HIV infection or familial mucocutaneous candidiasis¹⁶. There are multifocal fusiform arterial dilatations involving the major vessels of the circle of Willis, involving mainly the supraclinoid portion of the internal carotid artery and the basilar artery.

Primary intrinsic abnormalities of the arterial wall rather than a hemodynamic factor may contribute to the pathogenesis of a giant aneurysm in the pediatric population⁶. Patients

with polycystic kidney disease show an 8% incidence of aneurysm and there is an 18% incidence in families with one member who has already been diagnosed with an aneurysm. Other systemic disorders, including Ehlers-Danlos syndrome, Klippel Trenaunay syndrome, tuberous sclerosis, moyamoya disease, co-actation of the aorta, and fibromuscular hyperplasia, have been documented to be associated with aneurysms in children. In our patient, segmental dysplastic ectasia may have been the etiology because there was no evidence of infection, trauma or associated familial disorders¹⁷.

Although surgical resection can decompress a giant thrombosed aneurysm with mass effect¹⁸, complete occlusion of the parent artery including the aneurysmal neck results in shrinkage of the thrombosed aneurysm and repairs the diseased arterial wall as in our patient. Proximal ligation and incomplete clipping may lead to fatal hemorrhage of the aneurysm because there is a risk of rupture as long as the aneurysmal lumen remains after incomplete obliteration of the aneurysm¹⁹.

Balloon embolization of the aneurysm in this small child was a technical challenge. As Burrows et al described a technique⁷, we used a self-sealed gold valve balloon preloaded into a 4F guiding catheter after being loaded on a microcatheter. Selection of the left carotid artery is sometimes difficult because a 4F guiding catheter must be advanced without a guide-wire.

Miniaturizations of the devices and improvements in the embolic materials used have made it possible to perform endovascular therapy safely on neonates, infants, and children^{6,20}. Furthermore, cerebral plasticity and tolerance of spasm in children are fundamental features predisposing favorable outcomes in pediatric patient².

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