Spontaneous Healing and Complete Disappearance of a Giant Basilar Tip Aneurysm in a Child

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Summary

We report a rare case of spontaneous total thrombosis of a giant basilar tip aneurysm resulting in compression of the brainstem, diagnosed in a two-year-old child who presented with neurological deficits and third cranial nerve impairment. After conservative treatment, the giant aneurysm was completely thrombosed and the clinical symptoms were remarkably improved. MRI demonstrated dramatic shrinkage and ultimately complete disappearance of the giant aneurysm at seven month follow-up.

known about the nature history of thrombosis in GAs. Some degree of spontaneous thrombosis is identified in 9% to 13% of intracranial aneurysms⁴. The percentage of thrombosis formation in the GAs increases with size and has been found to vary between 52.4% and 83%⁵, while the spontaneous healing and complete disappearance of an aneurysms is exceptional⁶⁻⁸. We report an unusual case of spontaneous thrombosis of a distal basilar GA and complete disappearance on the follow-up magnetic resonance image (MRI).

Case Report

A two-year-old boy was in good health until 12 days prior to admission when he experienced a sudden onset of right-sided hemiplegia and aphasia together with left-sided third cranial nerve palsy. Computed tomography (CT) depicted a large high-density mass lesion in the suprasellar and interpeduncular cisterns extending to the left cerebral peduncle (figure 1A). MRI revealed a similar lesion of iso-intense signal on T1-weighted images and hypointense signal on T2-weighted images, with compression of the brain stem associated with edematous change (figures 1B, 1C, 1D). There was no evidence of subarachnoid hemorrhage

Introduction

The incidence of giant aneurysms (GAs) among all aneurysms is low, and the figures for both autopsy and clinical material are around 5%¹. The most frequent specific sites are on the cavernous and intradural segments of the internal carotid artery, follow by the vertebrobasilar system, the middle cerebral and the anterior communicating arteries. The frequency of GAs is significantly higher in children, occurring four times more commonly than in adults. The reported incidence of GAs in pediatric studies varies from 20-45%, and of these 40-45% are on the vertebrobasilar system^{2,3}. Very little is

on CT or MRI. Four-vessel angiography showed presence of a GA with partial thrombosis at the basilar tip associated with spasm of the distal basilar and left superior cerebellar arteries, there were several cerebellar arteries in the vicinity of the aneurysm neck (figures 1E, 1F). Bilateral posterior communicating arteries were demonstrated with opacification of the posterior cerebral arteries on bilateral carotid artery angiography. The CT and MRI were in

favor of thrombosis as a cause for the acute onset of clinical presentation, it seemed that the existence of spasm may have resulted from aneurysmal rupture. After angiography, the patient gradually recovered from his deficits to a degree that he could mobilize again, he also recovered his third cranial nerve palsy to the degree that he could open his eyelids. He also regained his speech and started to communicate with his family. However, he had a slurred

Figure 1 A) Non-contrast CT scan depicts a high-density mass lesion in the suprasellar and interpeduncular cisterns with compression of the brain stem. B, C, D) T1-weighted axial (B) and T2-weighted axial (C), sagittal (D) images demonstrate a partial thrombotic giant aneurysm in the distal basilar artery associated with compression and deformity of the brain stem as well as high signal edematous change on the T2-weighted images. E) Vertebrobasilar angiograms of anteroposterior view demonstrates thrombosis of the majority of the giant aneurysm in the basilar tip with small residual lumen. Vascular occlusion of the distal basilar and left superior cerebellar arteries are evident. F, G) Follow-up MRI 7 months later, axial (F) and sagittal (G) T1-weighted images reveal total spontaneous thrombosis of the giant aneurysm with remarkable shrinkage and complete disappearance of the aneurysm, there is neither compression nor mass effect of the brain stem currently.

speech pattern. Considering the extent of the thrombosis and the clinical tolerance of the lesion, the child was put on the low doses of aspirin to limit thrombotic phenomena and emboli migration to the intracranial branches. Gradual improvement in the neurological deficits was observed in the first three months, and all symptoms resolved within six months. MRI at seven months later revealed dramatic shrinkage with total disappearance of the GA

(figures 1G, 1H). There was neither mass effect nor edema in the brain stem. The patient is currently complete free of neurological deficit.

Discussion

GAs may clinically simulate brain tumors or vascular thromboembolic disease. The clinical manifestation is also variable, since about 47% of patients with GAs may present with sub-

arachnoid hemorrhage, the remaining patients present with neurological symptoms and signs that are either non-specific or related to intracranial mass effect⁶. Although the promotion of intra-aneurysmal thrombosis is the aim of various conservative therapies for cerebral aneurysms, the exact mechanisms and factors responsible for spontaneous thrombosis of the intracranial aneurysm have not been fully explained. Different authors have highlighted various factors, such as ratio of the aneurysm volume to orifice size, the age of the aneurysm, the haemodynamics within the parent artery and direct distortion of the parent artery by the aneurysmal sac, decreased blood flow inside the aneurysm, angiographic procedure, vasospasm, hypotension, dehydration, turbulence, antifibrinolytic agents, the high intracranial pressure, aneurysmal size and caliber of the parent vessel^{7,9-12}. According to Schubiger, a congenital defect in the tunica media of the vessel results in a change in the haemodynamic and turbulent flow cause clotting and deposition of onion skin thrombotic layers¹³; These intraluminal clots may reduce or in extreme cases completely obliterate the lumen of the aneurysm. In our case, we did not know the exact factor causing the thrombosis of the GA, the thrombosis may be triggered by the vasospasm of vertebrobasilar arteries shown at angiography, which could be a source of haemodynamic change in the parent artery and the aneurysm. Furthermore, the angiographic procedure itself might initiate the thrombosis of the aneurysm.

GAs of the vertebrobasilar arteries are one of the most difficult cases to manage because most GAs have a poorly defined neck and the perforator arteries feeding the brain stem may arise from the aneurysmal wall. In treating GAs, it is important to preserve the blood flow of the parent artery. GAs can be treated by surgical clipping or ligation of both vertebral arteries to reduce the direct impact of the blood flow to the aneurysm. Endovascular treatment with detachable balloons may be used to occlude the vertebral or basilar artery as an alternative to surgical ligation and clipping. Other authors reported using GDC to occlude GAs

or the parent artery. There is a high morbidity connected with these procedures¹⁴, this is due to GA involvement of functionally important branches in the wall of the aneurysms or trauma to these vessels during an attempt to isolate the aneurysmal sac, or involvement of the main trunk of the vessel in the aneurysmal wall or lumen. Intracranial aneurysms in children differ significantly in both clinical and radiological features from those that occur in adults^{2,15}, they usually have a better prognosis than adult patients because of the low incidence of systemic arterial hypertension and a decreased incidence of vasospasm after bleeding. Therefore conservative and medical treatment play a major role in therapeutic management of pediatric aneurysms¹⁵.

Our observations demonstrate that spontaneous thrombosis of a large part of a giant aneurysm could occur in less than one month, complete thrombosis and disappearance could develop in less than seven months. The neurological symptoms and signs were related to the mass effect and progressive thrombosis of the GA. The remarkable feature of this case is the clinical improvement accompanied with progressive shrinkage of a thrombosed GA.

Finally, this case raises the question: is it necessary to use invasive techniques to treat all intracranial GAs? Intracranial aneurysms indeed undergo a process of spontaneous thrombosis in certain cases. Although the development of a complete spontaneous intra-aneurysmal thrombosis is a favorable but rare occurrence, partial thrombosis of the aneurysm with intra-aneurysmal clot does not protect against subarachnoid hemorrhage⁵.

There is a poor prognosis of an aneurysm rupture and therefore aggressive treatment using surgical and endovascular approach should be conducted whenever it is possible. However, in cases of GAs with high morbidity and mortality for surgical and endovascular treatment, where the patient is asymptomatic or has a favorable clinical prognosis, as well as series imaging showing the spontaneous progressive thrombosis of the GA, conservative treatment with close observation is an alternative management.

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