Endovascular Treatment of Traumatic Intracranial Aneurysm in an Infant

A Case Report

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Summary

Traumatic intracranial aneurysms in children are rare and mostly related to skull fracture or rapid decelerating closed head injury. We report the case of an infant who developed intracranial aneurysm after minor head trauma and managed by endovascular treatment.

A seven-month-old infant presented with delayed intracranial hemorrhage following minor head trauma. Cerebral angiography disclosed a multilobulated fusiform aneurysm involving the right anterior cerebral artery (ACA) distal to the anterior communicating artery. Endovascular treatment of the aneurysm was performed and the infant made an excellent recovery during six months clinical and radiological follow-up.

Delayed presentation of intracranial hemorrhage with acute deterioration in the infant after head trauma warrants angiography for proper diagnosis and management of the traumatic aneurysm, which has a high mortality rate after rupture and rebleeding. Endovascular treatment of traumatic aneurysm is feasible in infants, and occlusion of distal intracranial arterial aneurysms can be safely and precisely achieved using current coil technology.

Introduction

Pediatric intracranial aneurysms are rare, representing less than 5% of all intracranial

aneurysms¹⁻⁵. Although 10 to 39% of pediatric aneurysms are post-traumatic in origin, most of them are in the older age group^{4,6-8}. Traumatic intracranial aneurysms in childhood are largely caused by skull fracture or rapid decelerating closed head injury. Clinical outcome in a pediatric patient with traumatic intracranial aneurysm depends on severity of the injury, with a potentially high mortality from rupture or rebleeding of the aneurysm⁷. Management has mostly been confined to surgical clipping, and few reports concern the endovascular treatment of traumatic aneurysm in infants.

We describe an infant who presented with delayed intracranial hemorrhage from ruptured traumatic aneurysm involving the ACA, managed by endovascular treatment.

Case report

A seven-month-old boy was admitted with diagnosis of intracranial hemorrhage. This previously healthy baby had a relatively minor trauma to the head when he struck his head against a door. At this time, there was no loss of consciousness and he remained well. One day later, he developed irritability and had an acute deterioration in his level of consciousness associated with some nausea and vomiting. He was taken to an emergency room after two episodes of seizure. He then became hypertensive and

Figure 1 A-B Brain CT showed a massive intraventricular hemorrhage with a parenchymal hemorrhage in the right subfrontal region (A) as well as some subarachnoid hemorrhage in the interhemispheric region (A and B).

Figure 2 A-B Surface shaded display of 3D reconstruction images (A and B) of right internal carotid angiography shows multilobulated dissecting aneurysm involving the proximal portion of A2 segment of right ACA including the origin of frontopolar artery.

unresponsive, and required intubation. Brain CT showed a large intraventricular hemorrhage with a parenchymal hemorrhage in the right frontal lobe as well as some subarachnoid hemorrhage in the interhemispheric region (figure 1). There was no history of intoxication, fever, or previous seizures.

The baby was then transferred to a Pediatric Center. On arrival, his Glasgow Coma Score was 6/15 (M4 / E1 / V1). The pupils were 3 mm with sluggish reaction to light. He withdrew to painful stimuli. Because of acute hydrocephalus with clinical symptoms, bilateral ventricular drains were inserted. Cerebral angio-

Figure 3 A Selective angiography of right ACA using microcatheter shows close proximity of the origin of the artery of Heubner, and encroached origin of the frontopolar artery by the aneurysm. A) Control microcatheter angiography after placement of four fibred GDC coils shows total occlusion of the aneurysm with sacrificed ACA distal to the aneurysm. There is a patent artery of Heubner proximal to the embolized aneurysm, and the anterior communicating artery is also patent with contrast opacification into the left ACA.

Figure 40 Right internal carotid angiogram shows nonvisualized right ACA and multiple segmental luminal narrowings at the right internal carotid artery, likely suggesting induced vasospasm (A). But prominent leptomeningeal collateral flow to the right frontal region is demonstrated through the right middle cerebral artery on delayed arterial phase (B).

graphy on the day of admission showed a large multilobulated aneurysm involving the right ACA complex (figure 2). A multidisciplinary team recommended selective angiography and endovascular treatment in order to manage the risk of rebleeding of this post-traumatic dissecting aneurysm.

On day five after admission, selective angiography followed by embolization was performed under general anesthesia. After right femoral arterial puncture, a 5 Fr guiding catheter was

Figure 5 Repeat right internal carotid angiogram at 6 months after embolization shows obliteration of the aneurysm as well as proximal right ACA (A). Prominent leptomeningeal collateral flow to the right frontal region is again demonstrated through the right middle cerebral artery on delayed arterial phase (B).

placed in the right internal carotid artery. Selective right anterior cerebral arteriography with the microcatheter showed a multilobulated fusiform aneurysm segmentally involving the proximal A2 segment including the origin of the frontopolar artery (figure 3a). The lesion proximally spared the origin of the artery of Heubner. After placement of a microcatheter within the aneurysm, four Vortex-18 Fibered GDCs were deposited; two 2x6 mm, one 2x5 mm and 2x4 mm in order. Because this fusiform aneurysm involved the entire circular wall of the segmental A2 and proximal frontopolar artery, sacrificing the origin of the distal right ACA was inevitable during trapping of the aneurysm. Selective right anterior cerebral angiogram showed complete occlusion of the aneurysm as well as distal ACA, sparing the artery of Heubner (figure 3b). On control right internal carotid angiography, the proximal right ACA was not opacified in antegrade fashion. It was thought to be in spasm related to the embolization procedure. The Right ACA territory, however, had prominent leptomeningeal collateral flow via the middle cerebral artery (figure 4).

Fifteen days after admission, the infant was alert and awake, responding to visual, tactile and sensory stimuli, without any limb weakness. Nineteen days after admission, a right

ventriculoperitoneal shunt was inserted after removal of the ventricular drain. When he was discharged 29 days after admission, there was mild hypotonia. His visual and auditory responses, personal-social skills, fine motor and language skills were age appropriate on follow-up evaluation after four months.

Repeat right internal carotid angiogram six months after embolization showed obliteration of the aneurysm. Although the right ACA was occluded, prominent leptomeningeal collateral flow from the right middle cerebral artery to the right frontal region was again demonstrated on the delayed arterial phase.

Discussion

Pediatric intracranial aneurysms are rare, representing less than 5% of all intracranial aneurysms¹⁻⁵. Traumatic origin of aneurysm in the general population is low, accounting for less than 1% of all aneurysms⁹. However, approximately 25 to 75% of traumatic aneurysms occur in pediatric patients^{7,9}, and 10 to 39 % of pediatric aneurysms are post-traumatic in origin^{4,6-8}.

Traumatic intracranial aneurysms can be due to penetrating or nonpenetrating trauma. The origin of nonpenetrating trauma includes injury to the carotid or vertebral artery by skull

base fracture¹⁰⁻¹², injury to intracranial vessels from the cerebral falx or sphenoidal ridge^{6,9,13,14}, or the distal cortical arteries with skull fracture^{15,16}. Injury by cerebral falx or sphenoidal ridge is relatively common in children, and usually results from rapid deceleration closed head trauma. Sudden movement of brain and artery against the stationary edge of the falx or sphenoidal ridge may be the cause of vessel wall injury¹⁷. This was also the most probable mechanism in our patient in view of his trauma history, delayed presentation of hemorrhage and location of the aneurysm. Although this injury usually comes from rapid deceleration head injury, aneurysms have also been reported to occur with minor injury or even with minimal history of trauma as in our case¹⁸. In a review of traumatic aneurysms in childhood, Buckingham et al⁷ found 23 cases of traumatic aneurysm involving the ACA, and 20 of them presented delayed intracranial hemorrhage. The time interval between trauma and hemorrhage varies from several hours to nine weeks after injury. Relative proximal location of aneurysm in our case, compared to usual pericallosal location of this type injury, may be from the injury of the vessel against sphenoidal ridge.

The clinical presentation of rare aneurysmal rupture in infants may be difficult to detect, and often starts with irritability only as in our case [13]. About half of the children with traumatic intracranial hemorrhage present with delayed intracranial hemorrhage, and remaining presentations are related to location of the aneurysm including recurrent epistaxis, progressive cranial nerve palsy, and bulging or enlarging skull fracture^{4,5,7}. Children with ruptured traumatic aneurysm, however, may show acute deterioration even after subtle presentation, and have an overall mortality of 31%. Traumatic aneurysms involving the ACA also showed 17% (4/23 patients) of mortality from aneurysmal rupture⁷.

In addition to trauma history, the morphology and location of the aneurysm supports traumatic dissection in the nature of the aneurysm in this infant. The aneurysm was multilobulated, and involved the entire circular wall of the ACA including the origin of the frontopolar artery. Although different in location and age from our case, the incidence of early recurrent subarachnoid hemorrhage in patients with rup-

tured intradural vertebral artery dissection varies from 24 to 70%, and the mortality from a second subarachnoid hemorrhage is up to 45% [19;20]. So early treatment is essential for such aneurysms with hemorrhagic presentation. While intracranial dissecting aneurysms presenting with ischemic stroke symptoms are managed with anticoagulant therapy, those presenting with hemorrhage should be managed by proximal sacrifice of the parent vessel or by trapping of the involved segment of the affected vessel if possible²¹.

Traumatic aneurysms have been treated by surgery or endovascular treatment with favorable results in the older pediatric age group, but those in infants are not well known^{4-7,22}. During the last few decades, miniaturization of the devices and improvements in embolic materials have made it possible to perform endovascular therapy safely in infants as well as children^{4,5,21,23}.

As previously reported, the modality of treatment of pediatric intracranial aneurysms at our institution is determined by a multidisciplinary team and depends on the aneurysm location, size and shape, coexistence of intracerebral hemorrhage and overall patient condition⁴. We favored endovascular treatment to manage the risk of rebleeding in this case. Proximal occlusion of this aneurysm seemed to be inadequate, because there was a short segment between the origin of the artery of Heubner and the aneurysm. Thus we decided to occlude the aneurysm itself. Sacrificing the parent lumen of the right ACA at the site of aneurysmal involvement was inevitable. In the management of giant intracranial aneurysm, balloon test occlusions may be performed prior to definitive occlusion of the parent vessel by surgical or endovascular measures. However, the placement of a test occlusion balloon beyond the circle of Willis in an infant represents an additional risk besides the fact that testing an infant under general anesthesia is cumbersome. Detachable coils can be placed precisely in the targeted segment. This was important especially in our case in which a significant proximal perforator needed to be spared. We used fibered GDCs to precipitate thrombosis.

Control angiography after embolization showed occlusion of the proximal ACA territory as well as complete obliteration of the aneurysm itself. But markedly prominent leptomeningeal

collateral flow throughout the right ACA territory via the right middle cerebral artery was demonstrated similar to the angiogram immediately after embolization.

While infancy would not always guarantee collateral flow, we believe that the prominence of collateral flow into the occluded vascular territory immediately after sacrifice favors good clinical outcome on follow-up. The infant in our case also recovered well, and showed normal age appropriate neurological responses, personal-social skills, fine motor and language skills until clinical follow-up at four months after treatment.

Conclusion

Traumatic intracranial aneurysm in an infant may have delayed presentation of intracranial hemorrhage, with rather acute deterioration. Suspicion of aneurysm in an infant with previous head injury presenting with hemorrhage warrants angiography for proper management of a possible traumatic aneurysm, which has high mortality rate after rupture. Endovascular treatment of traumatic aneurysm can be performed safely even in an infant. Precise occlusion of the targeted segment of the abnormal vessel can be achieved using detachable coil technology.

References

- Locksley HB: Natural history of subarachnoid hemorrhage, intracranial aneurysms and arteriovenous malformations. Based on 6368 cases in the cooperative study. *J Neurosurg* 25: 219-239, 1966.
- Patel AN, Richardson AE: Ruptured intracranial aneurysms in the first two decades of life. A study of 58 patients. *J Neurosurg* 35: 571-576, 1971.
- Heiskanen O: Ruptured intracranial arterial aneurysms of children and adolescents. Surgical and total management results. *Childs Nerv Syst* 5: 66-70, 1989.
- Laughlin S, TerBrugge KG et Al: Endovascular management of paediatric intracranial aneurysms. *Interventional Neuroradiology* 3: 205-214, 1997.
- Lasjaunias PL, Campi A et Al: Aneurysmal disease in children: review of 20 cases with intracranial arterial localisations. *Interventional Neuroradiology* 3: 215-229, 1997.
- Yazbak PA, McComb JG et Al: Pediatric traumatic intracranial aneurysms. *Pediatr Neurosurg* 22: 15-19, 1995.
- Buckingham MJ, Crone KR et Al: Traumatic intracranial aneurysms in childhood: two cases and a review of the literature. *Neurosurgery* 22: 398-408, 1998.
- Ventureyra EC, Higgins MJ: Traumatic intracranial aneurysms in childhood and adolescence. Case reports and review of the literature. *Childs Nerv Syst* 10: 361-379, 1994.
- Nakstad P, Nornes H et Al: Traumatic aneurysms of the pericallosal arteries. *Neuroradiology* 28: 335-338, 1986.
- Pozzati E, Gaist G et Al: Traumatic aneurysms of the supraclinoid internal carotid artery. *J Neurosurg* 57: 418-422, 1982.
- Yonas H, Dujovny M: "True" traumatic aneurysm of the intracranial internal carotid artery: case report. *Neurosurgery* 7: 499-502, 1980.
- Paul GA, Shaw CM et Al: True traumatic aneurysm of the vertebral artery: case report. *J Neurosurg* 53: 101-105, 1980.
- Tan TC, Chan CM, Chiu HM: Traumatic intracranial aneurysm in infancy. *Br J Neurosurg* 15: 137-139, 2001.
- Lam CH, Montes J et Al: Traumatic aneurysm from shaken baby syndrome: case report. *Neurosurgery* 39: 1252-1255, 1996.
- Holmes B, Harbaugh RE: Traumatic intracranial aneurysms: a contemporary review. *J Trauma* 35: 855-860, 1993.
- Rumbaugh CL, Bergeron RT et Al: Traumatic aneurysms of the cortical cerebral arteries. Radiographic aspects. *Radiology* 96: 49-54, 1970.
- Laurent JP, Cheek WR et Al: Traumatic intracranial aneurysm in an infant: case report and review of the literature. *Neurosurgery* 9: 303-306, 1981.
- Senegor M: Traumatic pericallosal aneurysm in a patient with no major trauma. Case report. *J Neurosurg* 75: 475-477, 1991.
- Mizutani T, Aruga T et Al: Recurrent subarachnoid hemorrhage from untreated ruptured vertebrobasilar dissecting aneurysms. *Neurosurgery* 36: 905-911, 1995.
- Yamaura A, Watanabe Y et Al: Dissecting aneurysms of the intracranial vertebral artery. *J Neurosurg* 72: 183-188, 1990.
- TerBrugge KG: Neurointerventional procedures in the pediatric age group. *Childs Nerv Syst* 15: 751-754, 1999.
- Fleischer AS, Patton JM et Al: Cerebral aneurysms of traumatic origin. *Surg Neurol* 4: 233-239, 1975.
- Suh DC, Alvarez H et Al: Intracranial haemorrhage within the first two years of life. *Acta Neurochir* 143: 997-1004, 2001.
- Matsuyama T, Matsumoto M et Al: Why are infant gerbils more resistant than adults to cerebral infarction after carotid ligation? *J Cereb Blood Flow Metab* 3: 381-385, 1983.

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