Traumatic Internal Carotid Artery Pseudoaneurysm Leading to Epistaxis and Anemia: Case Report

Epistaksis ve Anemiye Yol Açan Travmatik İnternal Karotid Arter Anevrizması

Gülen DEMİRPOLAT, MD,[a]
İsmail ORAN, MD[b]

[a]Department of Radiology, Kahramanmaraş Sütçü İmam University Faculty of Medicine, Kahramanmaraş
[b]Department of Radiology, Ege University Faculty of Medicine, İzmir

Geliş Tarihi/*Received:* 10.10.2008
Kabul Tarihi/*Accepted:* 06.11.2008

Yazışma Adresi/*Correspondence:*
Gülen DEMİRPOLAT, MD
Kahramanmaraş Sütçü İmam University Faculty of Medicine, Department of Radiology, Kahramanmaraş, TÜRKİYE/TURKEY
gulendemirpolat@hotmail.com

ABSTRACT In this study we are presenting a 16 year-old girl with a giant pseudoaneurysm originated from intracavernous segment of internal carotid artery. The patient was being investigated due to massive epistaxis and anemia. Her history was normal except severe trauma 3 months ago. Her otolaryngologic examination was normal. Paranasal sinus computed tomography showed multiple sphenoid fractures and a centrally enhancing mass obliterating sphenoid and ethmoid sinuses. A giant aneurysm developing from the intracavernous segment of internal carotid artery was detected in magnetic resonance imaging and magnetic resonance angiography. Embolisation was planned and right internal carotid artery was occluded with balloon. No complication was occurred during and after the procedure. Control magnetic resonance angiography showed totally occluded aneurysm. The patient stated that epistaxis did not reoccur. Bearing in mind that pseudoaneurysm may develop after serious trauma and being aware of its signs and symptoms could prevent its high mortality and morbidity.

Key Words: Epistaxis; carotid artery injuries; anemia

ÖZET Biz bu çalışmamızda travma sonrasında internal karotid arter intrakavernöz segmentte dev psödoanevrizma gelişen 16 yaşındaki hastayı sunuyoruz. Yeni başlayan masif burun kanaması ve anemi nedeniyle araştırılan hastanın öyküsünde 3 ay önce geçirilen ciddi travma dışında özellik yoktu. Hastanın otolaringolojik muayenesi normaldi. Paranazal sinüslerin bilgisayarlı tomografisinde sfenoid kemikte multipl fraktürler ve sfenoid sinüs ile etmoid sinüsleri dolduran, santral boyanma gösteren kitle saptandı. Manyetik rezonans inceleme ve manyetik rezonans anjiyografide sağ internal karotid arterin intrakavernöz segmentinden gelişen dev anevrizma izlendi. Olguya embolizasyon planlandı ve sağ internal karotid arter balon ile kapatılarak embolizasyon gerçekleştirildi. İşlem sırasında ve işlem sonrasında herhangi bir komplikasyon gelişmedi. Kontrol amaçlı çekilen manyetik rezonans anjiyografide anevrizmanın tamamen tıkanmış olduğu görüldü. Takiplerinde hasta burun kanamasının bir daha tekrarlamadığını bildirdi. Ciddi travmalardan sonra psödoanevrizma gelişebileceğinin akılda tutulması ve eşlik eden bulgu ve belirtilerin bilinmesiyle psödoanevrizmaya bağlı oluşan yüksek mortalite ve morbidite oranları önlenebilir.

Anahtar Kelimeler: Burun kanaması; karotid arter zedelenmesi; anemi

Turkiye Klinikleri J Cardiovasc Sci 2009;21(1):109-13

Traumatic vascular lesions of the head can arise after either blunt or penetrating forces. Carotid artery blunt trauma incidence is reported at 0.1% and as high as 0.45% in centers that routinely employ carotid and vertebral angiography.[1] Vascular injury can be minimal like intimal tear or can be very serious with dissection, laceration, pseudoaneurysm or fistula formation and high morbidity and mortality rates. Traumatic and ia-

trogenic carotid artery aneurysms are pseudoaneurysms and comprise less than 1-2% of all intracranial aneurysms.[2] Pseudoaneurysm intracavernous segment of internal carotid artery (ICA) s usually accompanied by fractures of the lateral wall of the sphenoid sinus. Delayed subarachnoid hemorrhage, severe headache of sudden onset, meningeal irritation, nausea, vomiting, unexplained neurological deterioration, epistaxis, cranial nerve palsy and unexplained cortical bleeding can be the presenting symptoms. The most common and almost invariable symptom of intracavernous segment aneurysm is massive epistaxis.[3] Epistaxis can begin days or years after the trauma.[4] In this report, we present a case with posttraumatic carotid artery pseudoaneurysm to discuss its pathophysiology, symptoms and management.

CASE REPORT

A 16-year old girl, presented with recent episodes of massive epistaxis and anemia. She was examined for upper gastrointestinal bleeding however all examinations, including gastrointestinal system endoscopies, were normal. There was no serious illness in history except a major trauma 3 months ago. After the accident she was hospitalized with cranial base fracture, spleen injury and left leg fracture after a fall down from the balcony. Neurological examination was normal and she was followed conservatively after treating the leg fracture and discharged home without any complication.

Recently done otolaryngologic examination did not reveal any abnormalities. Paranasal sinus computerized tomography scan was obtained and showed a soft tissue mass occupying the sphenoid and ethmoid sinuses. Multiple distortions due to fractures in the sphenoid sinus walls, resorption and expansion of the ethmoid sinus walls were seen. Central opacification was detected in the soft tissue mass, after contrast injection (Figure 1 A, B). For further investigation, she was evaluated with cranial magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA). Giant ICA pseudoaneurysm with jet flow into the sphenoid sinus was detected (Figure 2). DSA confirmed right ICA cavernous segment aneurysm with a wi-

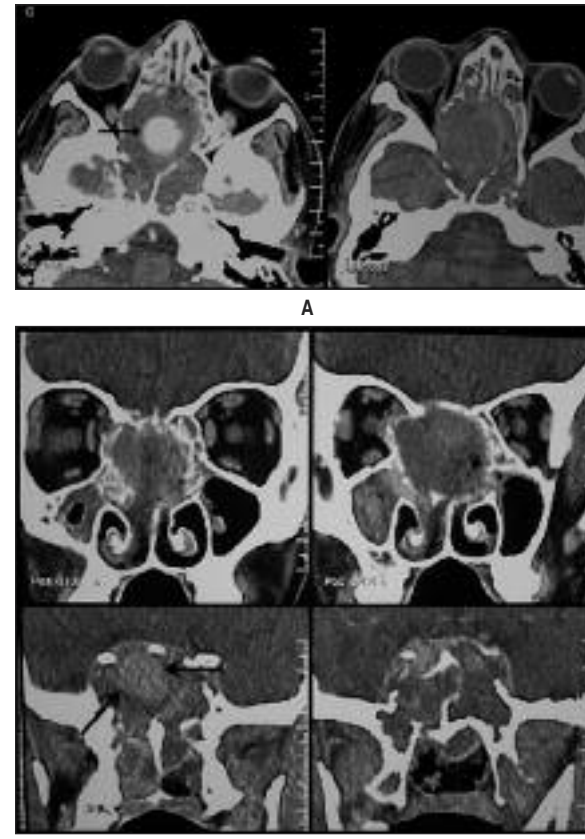

FIGURE 1: Precontrast and postcontrast axial (A) and postcontrast coronal (B) CT scans of the paranasal sinuses show a soft tissue mass with bony erosion and lateral expansion of the sphenoid and ethmoid sinuses. The mass shows central enhancement (arrow).

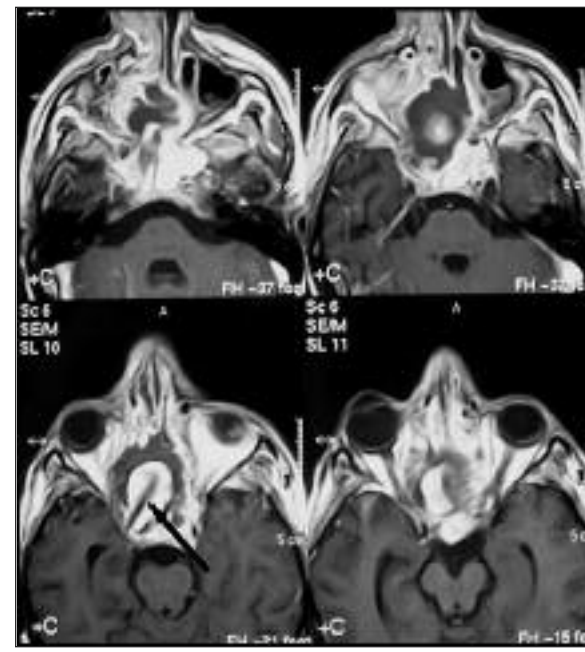

FIGURE 2: Contrast enhanced axial T1 weighted MRI shows hypointense peripheral thrombi and jet flow (arrow) within the enhancing aneurysm.

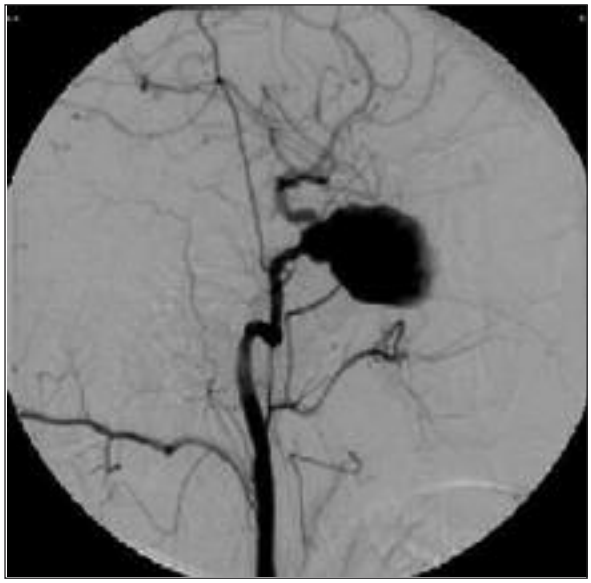

FIGURE 3: Right ICA angiography shows giant aneurysm of the intracavernous segment.

de neck (Figure 3). Endovascular therapy with balloon occlusion was planned. After informed consent of the patient and her parents, four vessels cerebral angiography and balloon occlusion test of the right ICA was performed. Temporary occlusion of the right ICA for 30 minutes was well tolerated without development of any neurological deficit. This test demonstrated enough collateral circulation from the contra lateral ICA. Consequently right ICA was occluded proximal to the aneurysm with two Goldvalve GVB 17 detachable balloons (Minvasys, Gennevilliers, France) (Figure 4). Control angiography from left ICA injection showed right anterior cerebral artery filling and occlusion in the cavernous segment aneurysm. No neurological complications developed during and after the treatment. Control MRA 1 month later showed total occlusion of the aneurysm.

DISCUSSION

Traumatic pseudoaneurysm occurs with haematoma formation after complete wall transection (adventitia, media and intima) compromising partial circumference of the vessel. The unclotted portion of haematoma located around the injured wall is filled by circulating blood in continuity with the arterial lumen. Haematoma may liquefy in around one week and rebleeding may occur or it may cause fibrous wall occurrence. However arterial pulsations can cause weakening in the wall and life-threatening rupture after latent period.[5,6]

Posttraumatic ICA aneurysm is a rare but life threatening complication with up to 50% mortality rate.[4,5] It usually develops in young healthy males in the second to fourth decades of life. ICA's anatomical features such as relatively fixed entrance into, and exit from, the cavernous sinus may make it susceptible to injury.[7] A pseudoaneurysm develops after a vessel wall injury and may expand over time, giving rise to a giant aneurysm. It may affect the adjacent vascular, nervous, and bony elements with eroding and extending into the neighboring structures. Renn and Rhoton have shown that ICA bulges into the sphenoid sinus in 71% of cadaveric specimens; the bony layer of the sinus was less than 1mm in 66 % of cases and absent in 4%, with only the dura covering the artery.[8] Delayed epistaxis presents after the aneurysm erodes the sphenoid sinus lateral wall. Because of the close anatomical relationship of the intracavernous segment to the oculomotor, optic, abducens, trochlear, and trigeminal nerves, these structures may also be damaged during the development of the aneurysm and lead to retroorbital pain, blindness, facial numbness, and/or oculomotor palsy.[4]

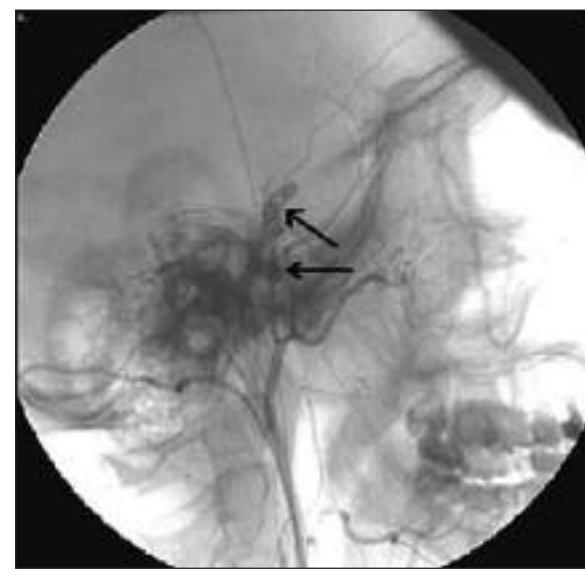

FIGURE 4: Control angiography. Balloons (arrows) in the cavernous segment and occlusion of the ICA are seen.

Latent period between the trauma and the symptoms makes it difficult to diagnose traumatic intracranial aneurysms. Maurer et al stated that triad of unilateral blindness, cranial base fractures, and recurrent severe epistaxes should prompt the diagnosis of cavernous segment pseudoaneurysm.[9] It is recommended that angiography should be performed as quickly as possible. If the first angiography is negative and intermittent epistaxis is present, control angiography should be done. In patients without epistaxis, angiography should be performed 2-3 weeks after the trauma.[3,4]

Mortality increases up to 3 times in patients with ruptured aneurysms. These aneurysms can be treated with emergent surgical or endovascular methods. Open surgery, with clipping of the aneurysm neck or proximal ligation of the ICA, was the standard treatment before the improvement of the endovascular techniques. Direct exposure of the cavernous segment is difficult and has high risks. These pseudoaneurysms are adjacent to the cranial base, which makes the entire artery wall and neighboring nerves susceptible to injury during surgical exploration.[3]

Endovascular procedures have some advantages over surgical therapy. The patient is conscious during the endovascular therapy and neurological examination is possible. Coil embolization is not proper for huge aneurysms as in the present patient. It is accepted that, parent artery occlusion is the first option in the treatment of pseudoaneurysms, if adequate collateral circulation is present.[10] Detachable balloons or platinum coils can be used for occlusion. If collateral circulation is not enough, extracranial-intracranial by-pass should be performed before the endovascular therapy.

In the English literature treatment of petrous and cavernous segment aneurysms with Jostent coronary stent graft has been published. Closuring of the side branches coming from the parent artery is a very important complication of this technique. Besides, these stents are not produced for cerebral vasculature and dissection or vasospasm of the artery can occur during the insertion.[11] This stent may be suitable only in selected cases.

Surgical therapy has a mortality rate reaching 20-22%.[12] Endovascular procedures are associated with negligible mortality rates.[11] Temporary (10%) or permanent (5%) ischemia is the major complication of the endovascular treatment.

CONCLUSION

Posttraumatic aneurysms of the intracavernous ICA can be associated with delayed, recurrent and sometimes lethal massive epistaxis. This vascular lesion should be considered in patients with traumatic cranial base fractures. Symptoms due to subarachnoid hemorrhage or cranial nerve involvement may also be seen. Remote history of trauma makes the diagnosis difficult. Knowledge of the risk factors, clinical symptoms and its urgency can minimize the high mortality risk. Prompt angiographic imaging is mandatory in these patients. In asymptomatic patients control DSA should be performed 2-3 weeks after trauma.

REFERENCES

1. Diaz-Daza O, Arraiza FJ, Barkley JM, Whigham CJ. Endovascular therapy of traumatic vascular lesions of the head and neck. Cardiovasc Intervent Radiol 2003;26(3):213-21.
2. Handa J, Handa H. Severe epistaxis caused by traumatic aneurysm of cavernous carotid artery. Surg Neurol 1976;5(4):241-3.
3. Uzan M, Cantasdemir M, Seckin MS, Hanci M, Kocer N, Sarioglu AC, et al. Traumatic intracranial carotid tree aneurysms. Neurosurgery 1998;43(6):1314-20.
4. Chen D, Concus AP, Halbach VV, Cheung SW. Epistaxis originating from traumatic pseudoaneurysm of the internal carotid artery: diagnosis and endovascular therapy. Laryngoscope 1998;108(3):326-31.
5. Zanini MA, Tahara A, Santos GS, Freitas CC, Jory M, Caldas JG, et al. Pseudoaneurysm of the internal carotid artery presenting with massive (recurrent) epistaxes: a life-threatening complication of craniofacial trauma. Arq Neuropsiquiatr 2008;66(2A):268-71.
6. Yavaş S, Sarıtaş A, Kervan Ü, Bardakçı H, İşcan HZ, Akgül A, et al. [False aneurysm of the internal carotid artery after a penetrating injury: case report.] Turkiye Klinikleri J Cardiovasc Sci 2007;19(3):193-6.
7. Haffajee MR, Naidoo S. Giant intracavernous internal carotid artery aneurysm with fatal epistaxis. Clin Anat 2003;16(3):277-81.
8. Renn WH, Rhoton AL Jr. Microsurgical anatomy of the sellar region. J Neurosurg 1975;43(3):288-98.

9. Maurer JJ, Mills M, German WJ. Triad of unilateral blindness, orbital fractures and massive epistaxis after head injury. J Neurosurg 1961;18:837-40.
10. Saatci I, Cekirge HS, Ozturk MH, Arat A, Ergungor F, Sekerci Z, et al. Treatment of internal carotid artery aneurysms with a covered stent: experience in 24 patients with mid-term follow-up results. AJNR Am J Neuroradiol 2004;25(10):1742-9.
11. Higashida RT, Halbach VV, Dowd C, Barnwell SL, Dormandy B, Bell J, et al. Endovascular detachable balloon embolization therapy of cavernous carotid artery aneurysms: results in 87 cases. J Neurosurg 1990;72(6): 857-63.
12. Parkinson D, West M. Traumatic intracranial aneurysms. J Neurosurg 1980;52(1):11-20.