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Endovascular embolization of intermittent massive epistaxis secondary to post-traumatic carotid artery pseudoaneurysms

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Abstract. *Endovascular embolization of intermittent massive epistaxis secondary to post-traumatic carotid artery pseudoaneurysm*. Epistaxis is a very common medical problem in otolaryngology practice and can usually be controlled with conservative interventions. Rarely, uncontrolled and life threatening epistaxis occurs. We present the case of a 29-year-old male who developed intractable intermittent epistaxis due to post-traumatic pseudoaneurysms arising from the cavernous segment of the left internal carotid artery. The patient was successfully treated with endovascular embolization.

Introduction

Epistaxis is a very common medical problem with variable manifestations.[1] Most patients with epistaxis present with anterior bleeding from the anterior nasal septum and are easily managed with minor interventions. However, in about 5% of patients the bleeding arises from the posterior portion of the nasal cavity and is often difficult to localize. Posterior epistaxis is typically massive.[1]

Life-threatening, intermittent, and intractable epistaxis associated with delayed craniocerebral trauma is rare.[2] In this article we present a 29-year-old male patient with intermittent, massive epistaxis associated with traumatic internal carotid artery pseudoaneurysms, managed with endovascular embolization.

Case presentation

A 29-year-old male patient suffered head trauma three weeks prior to presentation and was treated at an outside hospital. He presented to our emergency department complaining of repetitive episodes of epistaxis for one week, occasionally with severe bleeding. At evaluation in the emergency room, he did not have epistaxis but laboratory examination revealed a hemoglobin of 6.6 g/ dL and hematocrit of 19.2%. There was no history of coagulation disorder or anticoagulant treatment.

Computed tomography (CT) scan showed a fracture of the right lateral sphenoid sinus wall with blood in the sphenoid and ethmoid sinuses (Figure 1). After admission to the hospital, he was treated with bed rest, head elevation, and transfusion of three units of red blood cell suspensions. During the first transfusion massive epistaxis occurred. Nasal balloons were applied in each nasal cavity to control the bleeding. After nasal packing the patient was immediately admitted to the intensive care unit with monitoring of vital signs. After two to three hours, despite nasal packing, epistaxis occurred again but later stopped spontaneously. Maxillofacial magnetic resonance imaging (MRI) was performed and blood signal was detected in the sphenoid sinus (Figure 2).

The patient was referred on an emergency basis to the interventional radiology department for localization and hemostasis of the epistaxis. Bilateral external carotid artery (ECA) angiographies disclosed no bleeding. Bilateral selective internal carotid artery (ICA) angiographies revealed two pseudoaneurysms in the cavernous segment of left ICA (Figure 3). Both pseudoaneurysms were embolized with Guglielmi Detachable Coils (GDC, Boston Scientific, Fremont, CA, USA). While one of the pseudoaneurysms could be totally embolized, the other showed a small residual filling at the neck (Figure 4). There were no neurologic or clinical complications during or after the procedure and there was no relapse of epistaxis. The patient was discharged one week after the procedure. At six-month follow up, he complained only of minor headache,

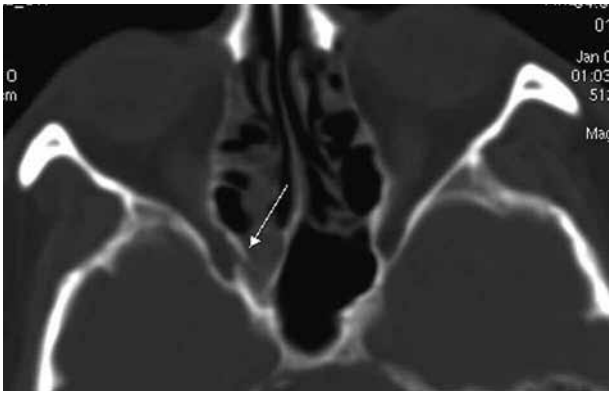

Figure 1
Transverse computed tomography with bone windows shows fracture of the left lateral wall of the sphenoid sinus (white arrow). Note blood density in the sphenoid and ethmoid sinuses.

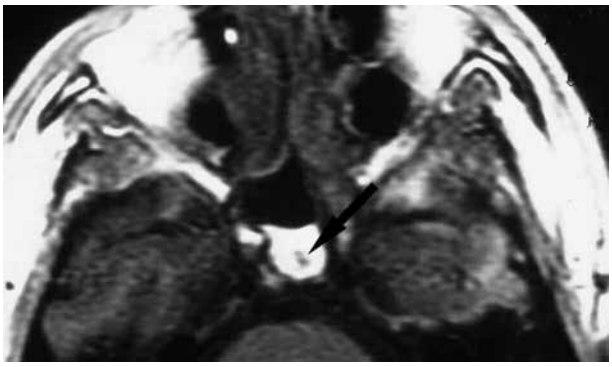

Figure 2
Transverse T1-weighted magnetic resonance imaging reveals hyperintensity in the sphenoid sinus with an air-fluid level, consistent with blood in the sphenoid sinus (black arrow).

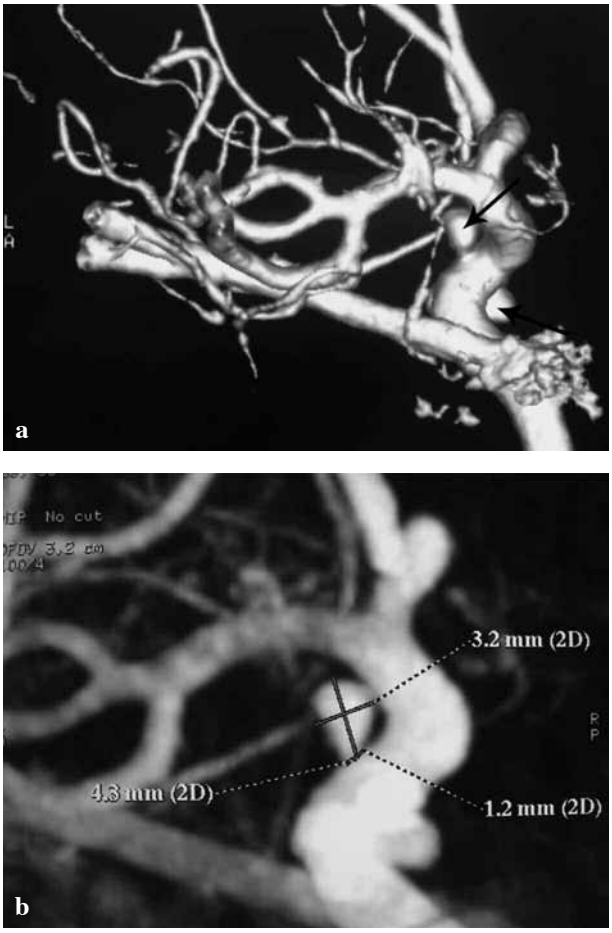

Figure 3
Pre-embolization 3D digital subtraction angiography with surface shaded (a) and maximum intensity projection (b) revealing two pseudoaneurysms at the cavernous segment of the left internal carotid artery.

which was managed with non-steroidal anti-inflammatory drugs. He reported no new episodes of epistaxis.

Discussion

Most nasal bleedings occur in the anterior part of the nasal septum and are caused by spontaneous rupture of blood vessels in the richly perfused nasal mucosa, at the level of the so-called locus Kiesselbacchi.[3] Predisposing factors are age, hypertension, anticoagulant medication, local inflammation, and coagulation disorders.[3] In most cases the bleeding stops spontaneously or ceases after firm compression for five minutes. Rarely nasal packing or application of vasoconstrictive sprays or coagulation-promoting mesh is necessary.[3] Superselective embolization of the internal maxillary arteries and the facial arteries with microparticles may be required in the case of uncontrollable bleeding. Pre-embolization angiography is performed to evaluate for the presence of any unsafe communication between the external carotid artery and the carotid or vertebral arteries.[4]

Posterior bleedings are usually more massive and therefore more difficult to localize, especially if bleeding is from both nostrils.[2] The bleeding arises from the ethmoidal or sphenopalatine arteries. Posterior epistaxis can be due to the abovementioned systemic causes, but frequently have a local underlying cause (Table 1). Initial therapy should be conservative with nasal packing or endoscopic cautery.[1] If these treatments are unsuccessful, radiologic investigations should be employed to search for an underlying local cause. CT and MRI can show frac-

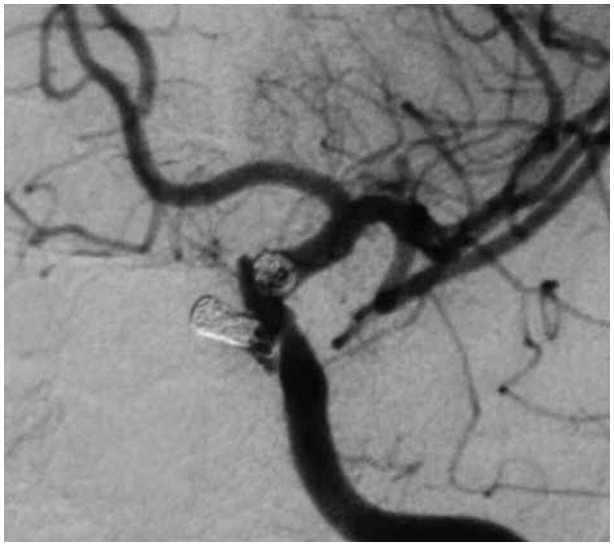

Figure 4
Post-embolization angiography shows coils in the pseudoaneurysms with total occlusion of the upper one and residual filling at the neck of the lower one.

Table 1
Differential diagnosis of intractable epistaxis

Post-traumatic ICA pseudoaneurysm
Arteriovenous malformation from the posterior nasal cavity
Hypervascular malignancies of nasal cavity and paranasal sinuses
ICA pseudoaneurysm secondary to invasive fungal sinusitis[6]
Aneurysmal bone cyst[12]
Radiation-induced pseudoaneurysm[13]
Moyamoya disease[14]
ICA trauma with transsphenoidal surgery[15]

tures, bone tumors, and hypervascular malignancies.[2] Vascular lesions can frequently be identified by these techniques, especially with the use of CT-angiography or MR-angiography. Selective catheter digital subtraction angiography (DSA) remains the gold standard.[1]

Aneurysms that involve the cavernous segment of the ICA are rare, representing only 3-5% of all intracranial aneurysms.[5] They rarely cause epistaxis unless they are large and erode the floor of the sphenoid sinus.

Pseudoaneurysms of the internal carotid artery can be caused by trauma, transsphenoidal surgery, or invasive fungal disease.[6] A pseudoaneurysm or false aneurysm is a communication between the arterial lumen and the overlying connective tissue. A blood filled cavity forms outside the vessel wall and seals the leak as it thromboses.[1]

The majority of traumatic pseudoaneurysms of the ICA result in massive bleeding with rupture into the sphenoid sinus, but in rare cases they present as recurrent epistaxis.[2] The mortality rate of bleeding from these pseudoaneurysms is reportedly up to 30%.[7] The clinical presentation of an ICA pseudoaneurysm may be delayed by months or years if the initial bleeding is not massive. This may result in delayed diagnosis and management. Delayed occurrence of the epistaxis may be explained by the time necessary for the traumatic or infectious event to weaken the vessel wall and for pulsatile pressure to erode through an intact bone along the sphenoid sinus.[8]

Neurosurgical therapy of aneurysms and pseudoaneurysms of the ICA is difficult and dangerous due to their location in the cavernous sinus. Therefore, endovascular therapy is currently the initial treatment of choice.[1] This method consists of the introduction of several detachable platinum coils into the lumen of the aneurysm until it is totally obliterated.[1] The use of a supporting balloon ("remodeling")[9] or a stent graft[10] may be necessary in case of a wide neck aneurysm or pseudoaneurysm. Some authors report the use of a covered stents in the lumen of the internal carotid artery with immediate repair of the blood vessel, but the procedure appears to be technically difficult.[11] In some cases of giant aneurysms, occlusion of the parent internal carotid artery with detachable balloons or coils is employed after careful balloon occlusion testing.[12]

Conclusion

Because of the high mortality and morbidity rate, the diagnosis and management of pseudoaneurysms of the ICA is a medical emergency. Awareness of the possibility of a post-traumatic pseudoaneurysm of the ICA in the differential diagnosis of intractable epistaxis is essential in order to appropriately treat this life-threatening condition. The case presented here highlights the fact that the diagnosis of pseudoaneurysm of the ICA must be considered in the differential diagnosis of intermittent massive epistaxis, especially if there is a history of head trauma.

References

1. Chaboki H, Patel AB, Freifeld S, Urken ML, Som PM. Cavernous carotid aneurysm presenting with epistaxis. *Head Neck*. 2004;26(8): 741-746.
2. 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):326-331.
3. Pope LE, Hobbs CG. Epistaxis: an update on current management. *Postgrad Med J*. 2005;81(955):309-314.
4. Sokoloff J, Wickbom I, McDonald D, Brahme F, Goergen TC, Goldberger LE. Therapeutic percutaneous embolization in intractable epistaxis. *Radiology*. 1974;111(2):285-287.
5. Kim JY, Farkas J, Putman CM, Varvares M. Paraclinoid internal carotid artery aneurysm presenting as massive epistaxis. *Ann Otol Rhinol Laryngol*. 2000;109(8 Pt 1):782-786.
6. Jao SY, Weng HH, Wong HF, Wang WH, Tsai YH. Successful endovascular treatment of intractable epistaxis due to ruptured internal carotid artery pseudoaneurysm secondary to invasive fungal sinusitis. *Head Neck*. 2011;33(3):437-440.
7. Chambers EF, Rosenbaum AE, Norman D, Newton TH. Traumatic aneurysms of cavernous internal carotid artery with secondary epistaxis. *AJNR Am J Neuroradiol*. 1981;2(5): 405-409.
8. Han MH, Sung MW, Chang KH, Min YG, Han DH, Han MC. Traumatic pseudoaneurysm of the intracavernous ICA presenting with massive epistaxis: imaging diagnosis and endovascular treatment. *Laryngoscope*. 1994; 104(3 Pt 1):370-307.
9. Shapiro M, Babb J, Becske T, Nelson PK. Safety and efficacy of adjunctive balloon remodeling during endovascular treatment of intracranial aneurysms: a literature review. *AJNR Am J Neuroradiol*. 2008;29(9):1777-1781.
10. Ko JK, Lee TH, Lee JI, Choi CH. Endovascular treatment using graft-stent for pseudoaneurysm of the cavernous internal carotid artery. *J Korean Neurosurg Soc*. 2011;50(1): 48-50.
11. Wang W, Li MH, Li YD, Gu BX, Wang J, Zhang PL, Li M. Treatment of traumatic internal carotid artery pseudoaneurysms with the Willis covered stent: a prospective study. *J Trauma*. 2011;70(4):816-822.
12. Składzieriń J, Olés K, Zagólski O, Moskała M, Sztuka M, Strek P, Wierzchowski W, Tomik J. A giant cranial aneurysmal bone cyst associated with fibrous dysplasia. *B-ENT*. 2008;4(1):29-33.
13. Buyukcam F, Sonmez FT, Aydin K. Successfully treated massive epistaxis in a patient with internal carotid artery pseudoaneurysm. *J Craniofac Surg*. 2010;21(4):1304-1305.
14. Hochmuth A, Ridder GJ, Gollner U, Boedeker CC, Klisch J. Moyamoya disease complicated by life-threatening epistaxis: first report of a case. *Acta Otolaryngol*. 2004;124(2):206-209.
15. Ghatge SB, Modi DB.Treatment of ruptured ICA during transsphenoidal surgery. Two different endovascular strategies in two cases. *Interv Neuroradiol*. 2010;16(1):31-37.

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