# PSEUDOANEURYSM OF THE INTERNAL CAROTID ARTERY PRESENTING WITH MASSIVE (RECURRENT) EPISTAXES

## A life-threatening complication of craniofacial trauma

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Vascular lesions can be serious complications resulting of blunt or penetrating trauma[1,2]. Internal carotid artery lesion is one of most serious and relatively frequent in all mechanisms of craniofacial trauma. Several clinical manifestations can occur as central neurologic and cranial nerves deficits as well as several degrees of bleeding (from mild symptomatic to fatal). Recurrent and massive epistaxis can occur after trauma due to pseudoaneurysms of the external and internal carotid artery (ICA)[3,4]. Considering its life-threatening course, the assisting physician has a relatively narrow time to detect and treat these lesions.

We present two cases of recurrent and massive epistaxis secondary to ICA pseudoaneurysm following blunt and perforating trauma. Evolution was fatal in the first case with delayed treatment and uneventfully in the second which was treated by occlusion of the pseudoaneurysm and ICA via endovascular intervention.

### CASES

#### Case 1

A 22-year-old girl suffered a motor vehicle accident with facial and cranial blunt trauma six months earlier. On admission at another institution, she presented nasal and oral bleeding and normal neurological examination (Glasgow score 15). Admission computed tomography (CT) showed basal orbitofrontal and maxillary fracture and no abnormality in the paranasal sinus (Fig 1). In subsequent days, she developed progressive right eye amaurosis, facial hypoesthesia, and epistaxis. Orbital decom-
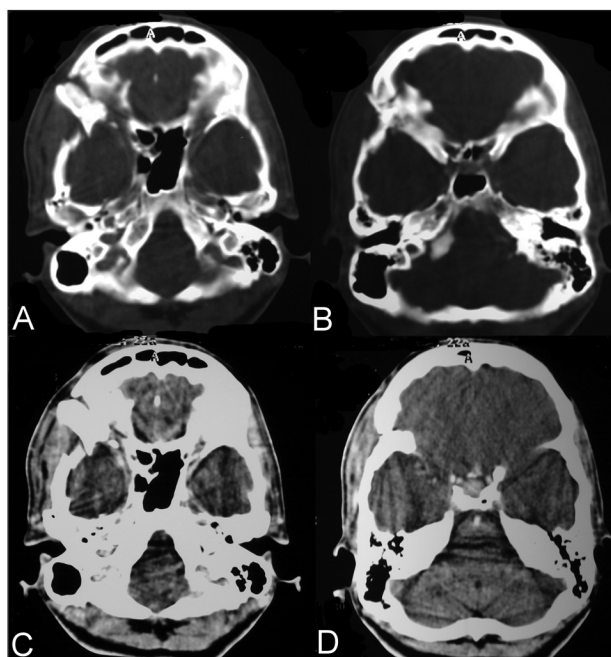


*Fig 1. Case 1. Admission CT scan. (A and B) Bone window and (C and D) contrasted CT scan showing orbitofrontal fracture with no abnormality in the sphenoidal sinus area.*

pressive surgery was performed. Over the next months she had some recurrent nasal bleeding episodes which were controlled by nasal packing. CT performed 5 months later showed an eroding mass in the sphenoidal sinus with enhanced contrast media inside (Fig 2). Magnetic resonance imaging (MRI) showed a flow void inside the mass, and a pseudoaneurysm was suspected
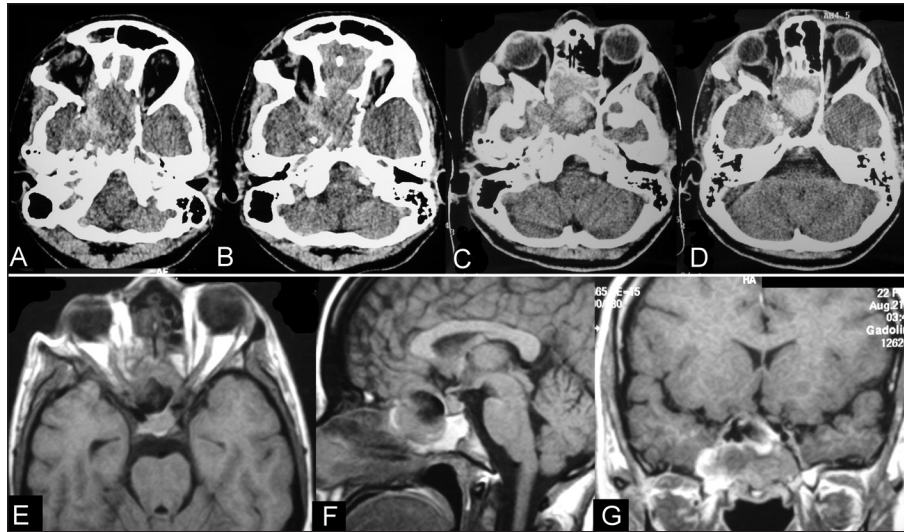
Fig 2. Case 1. Pseudoaneurysm images five months after trauma. (A–D) Contrasted CT scan showing cranial base eroding mass with enhanced contrast media. (E–G) MRI showing heterogeneous mass in the sphenoidal area with internal flow void.
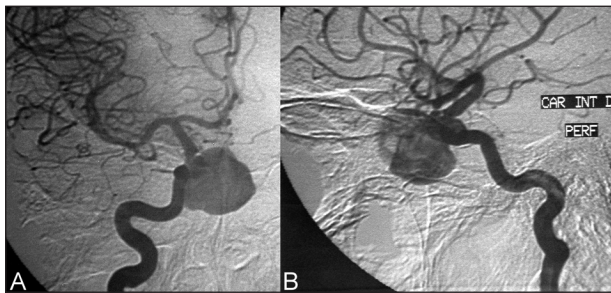


Fig 3. Case 1. Digital subtraction angiography. (A) Anteroposterior view (B) lateral angiography of the right ICA demonstrating the cavernous segment giant pseudoaneurysm.

(Fig 2). Digital subtraction angiography confirmed a giant traumatic pseudoaneurysm of the cavernous segment of the right internal carotid artery with extension into the sphenoidal sinus (Fig 3). After several episodes of severe epistaxis over 17 days which were treated by nasal packing and blood transfusion, the patient was transferred to the authors' facility. On admission, the patient was anemic and an abrupt and massive epistaxis occurred before proper treatment. The patient died despite continuous blood transfusion and resuscitation maneuvers.

### Case 2

A 23-year-old girl suffered two perforating knife wounds to the face, entering at the left nasal bone and left maxillary region. The blade crossed obliquely and cranially to the right, traversing the maxillary sinus and the pterygoid plate. On admission at another institution, she was hemodynamically stable with no evidence of other injuries. Conservative treatment was advocated as she was neurologically intact. Three months later she suffered headaches with recurrent massive epistaxis which was mistaken for upper gastrointestinal bleeding. Fifteen days later
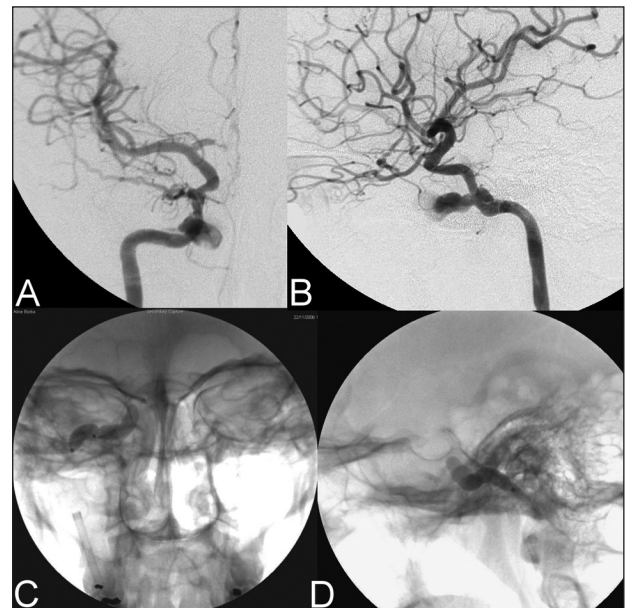


Fig 4. Case 2. Digital subtraction angiography. (A) Anteroposterior and (B) lateral angiography of the right ICA demonstrating the petrous-cavernous segment fusiform pseudoaneurysm. (C) Anteroposterior and (D) lateral radiographic view demonstrating the occlusion balloon.

she was transferred to our hospital after several blood transfusions. On admission at our institution, she presented epistaxis, headache and anemic. Digital subtraction angiogram showed a pseudoaneurysm of the petrous-cavernous transition segment of the right internal carotid artery (Fig 4A and 4B). She was submitted to balloon occlusion test followed by definitive occlusion of the internal carotid artery at the level of the lesion with two occlusive balloons (Fig 4C and 4D). Epistaxis and headache ceased immediately after the procedure. The immediate and 6

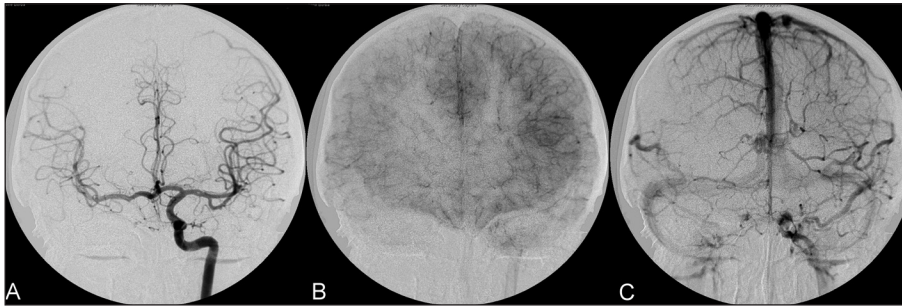*Fig 5. Case 2. Collateral circulation patency during six months follow-up, demonstrating aneurysm exclusion and proper collateral circulation from contralateral ICA. (A) Arterial phase. (B) Capillary phase. (C) Venous phase.*

months control angiogram showed no opacification of the aneurysm with proper collateral circulation (Fig 5). The patient is currently asymptomatic after seven months and performing her normal activities.

## DISCUSSION

The pathological substrate of direct arterial neurovascular trauma consists of transection, dissection, thrombosis, stenosis, arterio-venous fistulae and formation of a pseudoaneurysm[1,2,5]. The traumatic lesion of the internal carotid artery presents depending on the wall injury extension. Intimal lesions (incomplete wall tear and thrombus) presenting as thrombosis, stenosis or thromboembolic phenomena. Adventitial lesions also can occlude extrinsically the blood flow. Dissection occurs when an intimal tear permits penetration of blood into the vessel wall with or without formation of a pseudoaneurysm[6]. Transection occurs when complete wall compromising (adventitia, media and intimal) in a partial or complete circumference of the vessel and present as an acute bleeding. In these cases, formation of arteriovenous fistulae (carotidocavernous if occurs inside the cavernous sinus)[2,7] or a pseudoaneurysm[8,9] can be developed.

Traumatic pseudoaneurysm of the internal carotid artery (TPICA) pathogenesis involves partial vessel transection and formation of a hematoma. The bleeding stops with counterpressure. The unclotted portion of the hematoma located around the injured wall is filled by circulating blood in continuity with the arterial lumen. The hematoma liquefies in around one week and may rebleed. The blood causes an inflammatory reaction in surrounding tissue with the formation of a fibrous capsular wall and development of an epithelial lining. Continuous pulsatile forces can result in enlargement, weakening, and breakdown of the fibrous wall[8-10].

Usual clinical course is delayed, recurrent and well tolerated epistaxis to a severe bleed with hemodynamic instability, ranging from few days to several months, al-

though embolic phenomena and mass effect may also occur[11]. Skull base TPICA can exhibit the classic triad of unilateral blindness, orbital fractures, and massive epistaxis[12]. Bleeding occurs in the posterior septum similar to other vascular injuries: ethmoidal arteries, branches of the ophthalmic artery, sphenopalatine artery and internal maxillary artery. As a rule, traumatic arterial bleeding is more severe and more difficult to locate and control than in other epistaxis etiologies. Eventual rupture into the sphenoid sinus results in massive and even fatal epistaxis with an associated 30-50% mortality rate[8].

CT scan and MR provide initial information to suggest TPICA diagnosis[11,13,14] and minimally invasive imaging such as MR or CT angiography may confirm it[2,14]. However the gold standard is considered to be carotid angiography[1,2] and may provide the chance of prompt treatment by endovascular intervention. The angiographic features of pseudoaneurysm are: irregular contours of the aneurismal sac, lack of a defined neck, artery involvement, delayed emptying, and reduced opacity. Although TPICA is rare, it must be investigated in high risk patients: all knife penetrating trauma mainly if occurring in the orbitofrontal region or crossing the midline; blunt trauma patients with late neurological deterioration; cranial nerve palsy and amaurosis; and massive or mild recurrent post-traumatic epistaxis at any time in the past. Feiz-Erfan et al recently studying the incidence of direct neurovascular injuries associated with blunt trauma to the skull base recommended screening for neurovascular injuries in all patients with fractures of the clivus and sella turcica-sphenoid sinus[1].

TPICA can be treated by surgery or endovascular intervention. These are either deconstructive, in which the affected blood vessel is removed from the circulation, or reconstructive, in which the injured area is excluded from the vessel but circulation is maintained[13]. Surgical approaches are designed to either provide direct access to or bypass the lesion. A direct approach to the cavernous and petrous ICA is challenging because anatomic con-

straints limit direct operative exposure and distal control, and because of the fragile nature of the lesion preventing direct clipping[9].

Endovascular treatment of the pseudoaneurysm may be with coils, stent, covered stent, or ICA balloon occlusion[6,7,9,15,16]. Embolization with detachable platinum coil, sometimes associated with stent, provides intracranial pseudoaneurysm obliteration while maintaining ICA flow[14]. However there are reports of rebleeding after coiling[14,17,18]. The use of a covered stent allows immediate closure of the incompetent wall segment of the artery while preserving blood flow. However it may occlude arterial branches, many arterial stenoses have been currently reported and no data concerning the long-term patency are available[8]. Other disadvantages are difficulties of placement and navigation in tortuous segments and expense. Currently, parent vessel occlusion has been the optimal measure and can be done even with coils or detachable balloons[9]. Detachable balloon occlusion of the ICA at the level of the pseudoaneurysm is a quickly, easy, and less expensive procedure. It is important for occlusion to be performed at the level of the lesion, as well proximally, to avoid retrograde filling of the aneurysm by anastomoses from the Willis polygon or external carotid artery. A 30 minute balloon test occlusion (BTO) must be performed first. During the test, clinical and angiography features are analyzed. Clinically, the patient must have no neurological disturbance during the test. Angiographically, venous phase appearance must be symmetrical or with a delay of no more than 2 seconds on the occlusion side compared with the contralateral side. Even with the patient passing the occlusion test, there is risk of ischemic complications. If the patient cannot tolerate the occlusion test, extra-to-intracranial (EC-IC) bypass surgery should be realized[8,19]. Bypasses can be accomplished from the superficial temporal artery to middle cerebral artery (MCA), or from external carotid artery to MCA using either saphenous vein or radial artery grafts[19-22]. Reported long-term graft patency rates for saphenous vein grafts are 82% after 10 years[22]. The incidence of ischemic complications with carotid artery ligation has been reported as high as 41% without bypass[22]. A lower complication rate is expected if the BTO is performed[19]. In our case 2, carotid artery occlusion by balloon was used because the patient passed the BTO and prompt epistaxis healing occurred. Unfortunately, case 1 could not be treated due to delay on referring system, with a fatal outcome.

In conclusion, delay in diagnosis and treatment of pseudoaneurysm of the internal carotid artery can be very dangerous in patients and must be thoroughly investigated in patients with recurrent or massive epistaxis and trauma history. Because the onset of TPICA bleeding from the time of injury is variable, prompt diagnosis is clinically challenging. Optimal management demands rapid recognition and treatment to ensure the best outcome. Nowadays, endovascular occlusion of the pseudoaneurysm and parent vessel provides the most quick and effective treatment for long-term resolution of these dangerous lesions.

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