ABSTRACT

Epistaxis is a common ENT emergency. Various causes are mucosal trauma, anticoagulants and bleeding disorders. Vascular causes and trauma accounts for less than 5% causes. A 25-year-old man presented to emergency with intractable epistaxis. He had sustained road traffic accident 5 months back. His previous CT scan showed multiple facial bones fracture along with sinuses (frontal, maxillary and sphenoid sinus on right para clinoid area) fracture. Initial CT angiography showed no vascular defects. The latest episode of epistaxis was massive and he underwent nasal endoscopy and packing of nose. Pulsatile sphenoidal polyp was visualized, along with transmitted pulsations in the sphenoid sinus on the right side. Subsequent Magnetic Resonance Angiography (MRA) showed a 12x4 mm pseudoaneurysm of clinoidal portion of Internal Carotid Artery on the right side. Post-traumatic Pseudoaneurysm of the Internal Carotid Artery may be the differential diagnosis in traumatic epistaxis, especially the delayed type. If pseudoaneurysm is suspected, CT or MRI angiography should be performed immediately.

INTRODUCTION

Epistaxis is one of the most common emergencies in otorhinolaryngological practice which leads to distress in patients and treating clinicians. It may be caused by injury to nasal wall mucosal membrane, dryness of nasal mucosa, anticoagulation therapy and bleeding disorders. However vascular and trauma accounts to <5% of all the cases of nasal bleeding. Among them pseudoaneurysm affecting the internal carotid artery (ICA) is uncommon which accounts only 3%–5% of all the intracranial aneurysm. Therefore, delayed recurrent or massive epistaxis, especially after head trauma can be an alarming symptom of pseudoaneurysm. Here we describe a young man with delayed epistaxis in post-traumatic Internal Carotid artery Pseudoaneurysm, 5 months after head injury.

CASE REPORT

A 25-year-old man who met with a road traffic accident with facial bones, sinuses and cranial blunt trauma, 5 months back was admitted to a hospital, nearby. He was conscious, oriented, with normal speech and a GCS (Glasgow coma score) of 15. No motor deficits noted. Cranial Nerves and sensation were intact bilateral. Pupils were reacting to light, bilaterally. He complained of nasal and oral bleeding with melena. He underwent computed tomography (CT) scan of the brain and sinuses, which found a fracture of the bilateral frontal bone, maxillary sinuses, sphenoid wall on right side, around the clinoid process, medial orbital wall on right side, hemisinus in b/l maxillary, ethmoid, frontal and sphenoid sinuses, and right sided deviated nasal septum (Fig 1). Cerebral CT angiography showed no e/o aneurysm or av malformation (Fig 2). However, he developed gradual loss of vision of right eye.

Figure 1: Computed tomography scan image of fracture of right lateral wall of sphenoid sinus sphenoid fracture.
He was managed conservatively with antiepileptics, antibiotics and closely observed for Cerebrospinal fluid leak. Steroids were administered for his right traumatic optic neuropathy and he was discharged after few days of admission. But he had no perceivable vision in his right eye.

In the following months, he had recurrent nasal bleeding that stopped spontaneously. However, the last episode of nasal bleeding was uncontrolled, and he landed at emergency department of our hospital. On ENT examination, blood clots were found in the nasal cavity and mouth which was cleared. Hemoglobin level were decreased and blood transfusion given.

A rigid nasal endoscopy examination was performed which showed right nasal septum deviation. A yellowish white mass with a smooth surface was observed, possibly originating from the right sphenoid sinus (Fig 3). There was pulsation synchronous with the arterial beat. Based on the pulsation and previous recurrent bleeding, it was thought to be a Pseudoaneurysm. An Anterior packing with nonabsorbable material was applied to provide some pressure and delay potential life threatening hemorrhage. Magnetic resonance Angiography (MRA) of the neck was performed which revealed a 12mm x 4mm pseudoaneurysm, originating from the clinoid portion of the right internal carotid artery, and was directed anteromedially toward the sphenoid sinus (Fig 4). The patient was informed of the need for intervention in the form of bypass surgery or endovascular flow diversion.

DISCUSSION

Carotid aneurysm can have traumatic, infectious, or idiopathic causes as their etiologies. Parkinson classified aneurysms in this region as saccular and fistulous, and the fistulous variety can be further classified as spontaneous and traumatic. Traumatic aneurysms are pseudoaneurysms consisting of a hematoma surrounded by a fibrous layer, rather than a true arterial wall.

Continuous pulsatile arterial pressure will expand the aneurysm to a saccular shape. Pseudoaneurysm has a non-durable structure that can lead to massive bleeding, either spontaneously or associated with minor trauma.

Traumatic pseudoaneurysm of the internal carotid artery (TPICA) pathogenesis involves partial vessel transection and formation of a hematoma. The bleeding stops with counter pressure. The nonclotted 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 bleed again. 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 may cause enlargement, weakening, and dissolution of the fibrous wall.

In our case, we thought of pseudoaneurysm as a differential diagnosis because of the patient’s history of blunt head trauma, severe and recurrent epistaxis, and the presence of a pulsatile mass in the right nasal cavity. In addition, the presence of a period between the onset of persistent epistaxis and the trauma, supported the provisional diagnosis.

Han et al. reported that the time between trauma and the onset of massive epistaxis can vary from 1 week to 8 months. As in our case, one-sided blindness, orbital fractures and massive epistaxis, which is Maurer’s classic symptom triad, should bring the pseudoaneurysm into the clinician’s mind. Initial bleeding is rarely fatal, but the frequency and severity of bleeding gradually tends to increase. However rapid diagnosis and urgent treatment intervention is crucial as it is unknown which bleeding can be mortal. The pseudoaneurysm must be demonstrated with a radiologic method such as digital

Figure 2: CT angiography image of pseudoaneurysm of the carotid artery

Figure 3: Endoscopic image of nose showing sphenoid polyp

Figure 4: Carotid pseudoaneurysm
subtraction angiography, magnetic resonance angiography or CT angiography for definitive diagnosis.

CT scan and MR provide initial information to suggest TPICA diagnosis and minimally invasive imaging such as MR or CT angiography may confirm it. However, the gold standard is considered to be carotid angiography and may provide the chance of prompt treatment by endovascular intervention. 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.

In conclusion, posttraumatic pseudoaneurysm of the carotid artery may be the differential diagnosis in traumatic epistaxis. If pseudoaneurysm is suspected, CT or MRI angiography should be performed immediately. And appropriate treatment can be done with endovascular methods.

REFERENCES: