CASE REPORT

A Time Bomb Defused, In Time! A Traumatic Optic Neuropathy To Be Wary Of

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ABSTRACT

Authors describe a case of traumatic pseudoaneurysm of cavernous internal carotid artery in a young male who presented with complaints of progressive decrease in vision, inability to move the eye, and delayed epistaxis 8 months after the head injury sustained during a motor vehicle accident. The presence of optic atrophy and epistaxis in the setting of head trauma alerted the authors to look for this potentially life-threatening condition. Timely intervention in the form of endovascular coil occlusion of the internal carotid artery was performed and the epistaxis stopped without any ischaemic or thromboembolic sequelae.

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Introduction

Aneurysm refers to persistent pathologic dilatation of the vessel wall. It may be a true or a pseudoaneurysm. In a true aneurysm, the vessel wall is intact whereas in a pseudoaneurysm the vessel is partially transected with a discontinuity in the vessel wall. Traumatic aneurysms of intracranial carotid artery (ICA) and its branches are rare lesions with an estimated incidence of 0.15% and 0.40% of all intracranial aneurysms.¹ These aneurysms can cause complications due to delayed rupture or mass effect on the adjacent cranial nerves. Associated injury to the ophthalmic artery can cause immediate blindness and the downward protrusion of the mass into the thin bone of ethmoid and/or sphenoid sinus can lead to mass effect on the adjacent structures like optic nerve and the oculomotor nerves causing vision loss and ophthalmoplegia which may be associated with massive epistaxis. We report one such case of delayed presentation of pseudoaneurysm of ICA who came to us for evaluation of blindness and ophthalmoparesis. Most of the traumatic pseudoaneurysm of internal carotid artery (TPICA) are diagnosed in the neurosurgical or otolaryngology clinics. The authors share an interesting case of TPICA presenting to an ophthalmologist with an orbital apex syndrome.

Case report

A 31-year-old male presented to us with a history of road traffic accident while riding a bike 8 months back. He had sustained a head injury with a major impact on the forehead region. Patient was managed locally for his head injury. The patient complained of gradual progressive decrease in vision after the accident for past 4 months. He complained of a recent onset headache and nasal bleeding since past 1 month.

On examination the best corrected visual acuity in right eye (RE) was 20/20. Left eye (LE) was no perception of light. Extra ocular movements in RE were full and LE showed an elevation limitation. External examination revealed mild ptosis and proptosis. Pupillary reaction showed a brisk reaction to light in RE and a relative afferent pupillary defect in LE. Intraocular pressure was within normal limits in both eyes (BE). Fundus in the RE was normal. LE revealed diffuse optic atrophy. Computed tomography of the brain and orbit was done at the time of initial trauma 8 months back locally in the patients home town and the films were not available for evaluation to look for the skull base fractures. Magnetic resonance imaging of brain and orbit showed a large irregular lesion in the posterior ethmoid and sphenoid sinus with irregularity of the left cavernous internal carotid artery (Figure 1). Magnetic resonance

Figure 1. Magnetic resonance imaging of brain (axial T2 weighted) showing a large irregular lesion of mixed intensity in the posterior ethmoid and sphenoid sinus with irregularity of the left cavernous internal carotid artery.

angiogram showed giant pseudoaneurysm arising from the cavernous segment of left internal carotid artery with a good cross circulation through circle of Willis's (Figure 2). Digital subtraction angiography (DSA) was done which confirmed the diagnosis of

Figure 2. Magnetic resonance angiogram showing a giant pseudoaneurysm arising from the cavernous segment of left internal carotid artery with a good cross circulation through circle of Willis.

pseudoaneurysm of the left internal carotid artery (Figure 3). An otolaryngologist's opinion was sought for the epistaxis and patient was advised nasal packing as a temporary measure. Patient was referred to an interventional radiologist for coil embolization of the feeding artery (Figure 4). With balloon occlusion technique, cross flow to left internal carotid artery territory through circle of Willis was tested. Since the pseudoaneurysm was involving left internal carotid artery, balloon occlusion test was performed by

Figure 3. Digital subtraction angiography showing the pseudoaneurysm and the circulation in the left carotid artery.

Figure 4. Post embolization digital subtraction angiography showing the left intracranial carotid artery. (lateral).

inflating the balloon in the left internal carotid artery temporarily for 30 minutes. During this 30-minute period, patient was assessed for development of untoward symptoms like headache, retro orbital pain, giddiness, and occurrence of any fresh neurological deficit. At the same time, collateral circulation to the left internal carotid artery through circle of Willis by performing selective right internal carotid artery and left vertebral angiogram. Since patient did not develop any untoward symptoms and there was excellent cross circulation to the left internal carotid artery territory through the circle of Willis, left internal carotid artery was permanently occluded using multiple coils by endovascular procedure.

Post embolization there were no systemic complications. DSA revealed good cross circulation from the right ICA and left vertebral artery. Post embolization, the epistaxis stopped but there was no improvement in the visual acuity or ophthalmoplegia in LE.

Discussion

A traumatic cerebral aneurysm is a dangerous lesion with rupture or increase in size probable and spontaneous thrombosis rare.² Early diagnosis and treatment is mandatory as the likelihood of exsanguinations increases with each subsequent episode of epistaxis.

Pathophysiology

TPICA results from direct injury to the arterial wall as seen in penetrating head injuries or due to the indirect injury caused by acceleration induced shear as in closed head trauma. It can develop within hours from trauma. In pseudoaneurysm, haemorrhage from a small, full-thickness arterial rent is adequately contained by the buttressing effect of local brain tissue.² The blood causes an inflammatory reaction in surrounding tissue with the formation of a fibrous capsular wall and development of an epithelial lining. The apparent lumen of the aneurysm is contained by an organized extraluminal hematoma. The hematoma liquefies in around 1 week and may rebleed. Continuous pulsatile forces can result in enlargement, weakening, and breakdown of the fibrous wall.³

Clinical course

Traumatic intracranial aneurysms are highly unstable lesions that may rupture within minutes after formation or remain quiescent for several weeks and may manifest with delayed haemorrhage and neurological deficits.⁴ Usual clinical course is delayed, recurrent and well-tolerated epistaxis to a severe bleed with haemodynamic instability, ranging from few days to several months although embolic phenomena and mass effect may also occur.⁵ Skull base TPICA can exhibit the classic triad of unilateral blindness, orbital fractures, and massive epistaxis. Eventual rupture into the sphenoid sinus results in massive and even fatal epistaxis with an associated 30–50% mortality rate.³

Evaluation

Although TPICA is rare, it must be investigated in high-risk patients like, 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. have seen the incidence of direct neurovascular injuries associated with blunt trauma to the skull base and have recommended screening for neurovascular injuries in all patients with fractures of the clivus and sella turcica-sphenoid sinus.⁶ TPICA is associated with craniofacial fracture nearby the course of ICA.⁷

Management

Occlusion of the parent artery is the most appropriate treatment of pseudoaneurysm anywhere in the body. An endovascular occlusion provides the quickest and effective treatment for long-term resolution of these dangerous lesions. It may be with coils, stent, covered stent, or ICA balloon occlusion.^{8–10} A 30-minute balloon test occlusion must be performed first. If the collateral circulation is not satisfactory, parent artery cannot be sacrificed. In such cases, flow diverter stent can be deployed across the pseudoaneurysm by vascular root in the extracranial segment of internal

carotid artery, and if no significant branches are arising from that segment, covered stent graft also can be used. Surgical ligation of the artery for permanent occlusion or trapping of the pseudoaneurysm also can be performed.^{8–10}

In this case, the pseudoaneurysm was diagnosed after 8 months of trauma with a head injury. In normal circumstances an 8-month-old traumatic optic neuropathy leading to no perception of light vision does not require any further evaluation. The presence of recurrent epistaxis, a history of progressive decrease in the visual acuity and ophthalmoplegia warranted a neuroimaging, which revealed the pseudoaneurysm of the internal carotid artery. An awareness of this condition can help in diagnosing and averting one of the delayed catastrophic complications of head injury.

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Conflict of interest

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