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Clinical Communications: Adults

A 32-YEAR-OLD MAN WITH DELAYED ONSET POST-TRAUMATIC PROPTOSIS AND DIPLOPIA

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☐ **Abstract—Background: Proptosis and motility deficits are common findings in the setting of craniofacial trauma, but can indicate the presence of vision and even life-threatening pathology. Objective: Our aim was to identify presentations consistent with traumatic carotid cavernous fistula (CCF) and to review the appropriate initial work-up and management. Case Report: A 32-year-old man came to our emergency department with proptosis, ocular motility deficits, and decreased vision 1 month after a restrained motor vehicle accident. An orbital bruit was auscultated and four-vessel angiography revealed a CCF. Covered stents and an embolic agent were used to abolish the arteriovenous communication and the patient rapidly returned to his premorbid baseline. Conclusions: CCF is a relatively rare but important consequence of craniofacial trauma that must be recognized promptly in order to minimize the likelihood of serious sequelae. It should be suspected in patients with antecedent trauma presenting with exophthalmos, arterialized conjunctival vessels, and orbital bruit.** © 2014 Elsevier Inc.

☐ **Keywords—craniofacial trauma; carotid cavernous fistula; angiography; proptosis; vision loss**

INTRODUCTION

Proptosis, ocular motility deficits, and transient reductions in vision are very common in the setting of craniofacial trauma. Some degree of periocular edema, chemosis, and mild proptosis is frequently observed, even in the absence of serious ocular or orbital pathology.

Clinically significant fractures and retrobulbar hemorrhage must be identified and treated appropriately. Management of these conditions has been addressed extensively in the trauma literature.

Traumatic carotid cavernous fistula (CCF) is a less common and often neglected cause of post-traumatic proptosis and motility deficits that results from the formation of an arteriovenous communication between the internal carotid artery (ICA) and cavernous sinus (CS). It must be recognized due to the potential for vision-threatening and even life-threatening sequelae.

CASE REPORT

A 32-year-old African-American man reported to our emergency department with a 1-month history of double vision and redness of the right eye after a high-speed automobile accident. He lost consciousness after hitting his head on the dashboard, but was not ejected from the vehicle. Per the patient, he spent several days in a trauma intensive care unit before discharge.

Uncorrected visual acuity at the time of presentation was 20/50 in right eye and 20/20 in the left. Intraocular pressures were 27 and 17 mm Hg, respectively. In primary gaze, he had a slight right exotropia (out-turning of the eye), along with moderate motility deficits in all cardinal gaze directions. Pupils were 3 mm with brisk

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light reactivity and absence of an afferent pupillary defect. Visual fields by confrontation were grossly normal. Apart from dilated conjunctival blood vessels and 3 mm of proptosis in the right eye, the remainder of his ocular examination was unremarkable (Figure 1).

Despite his motility deficits, the absence of eyelid ptosis and pupillary changes was not consistent with a traumatic or compressive third cranial nerve palsy. Orbital cellulitis and inflammatory conditions, such as orbital pseudotumor or scleritis, were not thought likely based on the absence of pain, chemosis, and periocular soft-tissue changes. Given the combination of proptosis and unilateral arterialization of the conjunctival blood vessels, a vascular process of the orbit was suspected. Subtle pulsatility of the right eye was observed. A right orbital bruit was auscultated, and he was referred to a neurointerventionalist with the tentative clinical diagnosis of traumatic CCF. Four-vessel digital subtraction angiography revealed profound dilatation of the right superior ophthalmic vein (Figure 2).

A covered stent was deployed in the intracavernous ICA via a femoral approach after ethylene vinyl alcohol copolymer embolic agent was introduced into the ipsilateral CS with a microcatheter. Post-procedure angiography confirmed successful ablation of the abnormal arteriovenous connection, as well as preservation of distal arterial flow (Figure 3).

On repeat examination, the patient's visual acuity improved to 20/20 and his intraocular pressures were normal and symmetric. His motility deficits and double vision also resolved rapidly, as did his proptosis.

DISCUSSION

CCF is important to consider in the differential for post-traumatic proptosis. It is the result of an acquired or recanalized congenital arteriovenous communication between the ICA and the CS. The classic clinical triad of high-flow lesions consists of exophthalmos, arterialization of conjunctival blood vessels (due to ocular and orbital congestion), and a cranial bruit that diminishes with compression of the ipsilateral carotid artery.

Figure 1. Facial photograph demonstrating right-sided proptosis, moderate restriction in downgaze, and unilateral arterialization of conjunctival vessels.

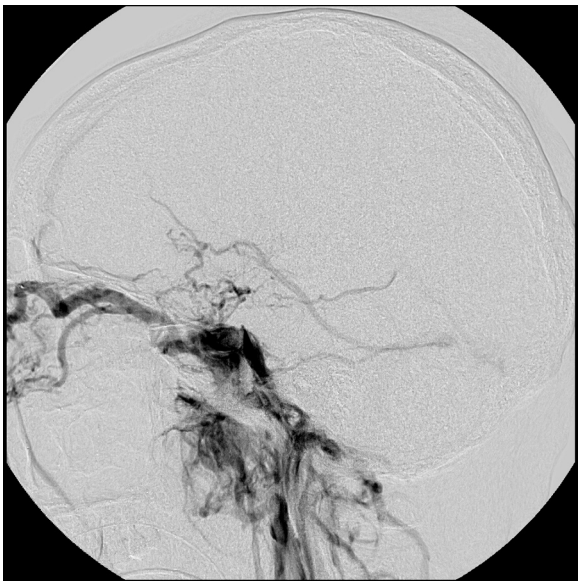

Figure 2. Digital subtraction angiography demonstrating dilatation and abnormal filling of the right superior ophthalmic vein during the arterial phase.

Common associated findings include elevated intraocular pressure, visible or palpable ocular pulsations, diplopia, and reduced vision (1).

Lesions are categorized according to the Barrow Classification, which is based on angiographic features. Barrow type A lesions are direct, high-flow lesions resulting from a wall defect between the ICA and CS. Barrow type B, C, and D lesions are indirect, lower-flow lesions resulting from abnormal communication between branches of the ICA or external carotid artery and the CS (2).

Type A lesions account for 70% to 90% of all CCF, and occur most commonly in younger males (3). Eighty percent are seen in the context of craniofacial trauma, with an overall estimated incidence of 0.2% (3). Onset is highly variable, ranging from hours to months after trauma. They are highly associated with skull-base fractures and penetrating injuries. The remaining 20% are generally the result of spontaneous rupture of occult intracavernous ICA aneurysms or weakened atherosclerotic arteries.

Direct lesions rarely close spontaneously, unlike the indirect lesions that generally represent the spontaneous recanalization of small carotid−cavernous connections in the context of hypertension and atherosclerosis, childbirth, or collagen vascular disease.

Natural variability of venous sinus anatomy results in several important variations in clinical presentation. Patients with widely patent superior or inferior petrosal sinuses might shunt arterial blood to the internal jugular vein, resulting in few or no ocular sequelae ("white eye shunt"). Those with limited petrosal sinus patency can