Direct Carotid-Cavernous Sinus Fistula Following Closed Head Injury

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ABSTRACT

A case of direct carotid-cavernous sinus fistula (CCF) after closed head injury is presented. A 22-year-old male presented to the emergency department of Shohada Tajrish Hospital with the chief complaint of blurred vision and pulsatile retro-orbital headache. The patient had closed head injury due to car accident 2 month ago with lower limb fracture. After a 2-week symptom-free period, he developed scalp and right facial tingling, along with pulsatile retro-orbital headache and vision problems. His vital signs were within normal limits, but on primary evaluation the patient orbital and carotid bruits could be recognized. Computed tomography (CT) scan and magnetic resonance imaging (MRI) suggested the carotid-cavernous sinus fistula, which was confirmed by brain angiographic imaging findings. Carotid-cavernous sinus fistula is an uncommon condition that is usually caused by head trauma but can advance spontaneously in about one fourth of patients with CCF. The connection between the carotid artery and cavernous sinus leads to increased pressure in the cavernous sinus and compression of its contents, and finally advances the clinical symptoms and signs seen. Diagnosis is based on clinical evaluation and neuroimaging techniques. The target of management is to decrease the pressure within the cavernous sinus, which results in gradual recovery of symptoms.

INTRODUCTION

A carotid-cavernous sinus fistula (CCF) is an uncommon condition well known in the neurosurgical territory, but not well described in emergency medicine literature. Without considering its nature, the symptoms related to CCFs are dangerous and potentially severe, from chronic headaches and diplopia, to intracranial hemorrhage and permanent vision loss. Hence the ability to recognize and supervise this disease is essential. The clinical presentations of CCFs can be varied as well, mimicking diseases like multiple sclerosis, brain tumors, or stroke, making it imperative that emergency physicians in the right setting include this disease in their differential diagnosis. We report a case of a patient with a direct CCF that presented to our Emergency Department (ED) 2 month after a closed head injury following car accident.

CASE PRESENTATION

A 22-year-old male presented to the emergency department with the chief complaint of “blurred vision and facial tingling”. He had closed head injury following car accident 2 month ago.” He remained symptom-free for the next 2 weeks, until he advanced facial tingling, and pulsatile headache. Over the next week his symptoms had increased to include: right-sided scalp and forehead paresthesia, blurred light photophobia vision, light
photophobia, right eye proptosis, right-sided droopy eyelid and a pulsatile retro-orbital headache. With increasing difficulty performing his job as a worker, he presented to the ED for receiving medical care.

His past medical history was significant for old temporal bone fracture following head trauma. He received no medications and had no significant family history. On physical examination, vital signs were stable. The visual field was normal as well, regarding to his age. His visual acuity was OD 20/40 and OS 20/25, and there were bilateral orbital bruits. The right eye showed: ptosis and chemosis (Figure 1). He had reduced sensation to fine touch over the entire forehead, nose, and right cheek, but his corneal reflexes were intact. No facial asymmetry was seen. The rest of the cranial nerve (CN) examination was intact. Clinical examinations revealed no focal neurological deficits, and deep tendon reflexes were 2/4 symmetrical in both the upper and lower extremities. The cerebellar examination was normal. Other systemic examinations were intact except for bilateral carotid bruits. Brain angiography reveal carotid–cavernous fistula (Figure 2).

**DISCUSSION**

We report a rare condition presented in the emergency department of a post-traumatic carotid-cavernous fistula. CCFs are formed when there is connection between arterial flow from the carotid artery into the venous system of the cavernous sinus. This pathological connection result in pressure elevation within the cavernous sinus and its contents compression, including CN III, IV, V, and VI. These nerves’ involvement results in ophthalmoplegia, facial sensory deficits, ptosis, and photophobia. Elevating venous hypertension conduct ophthalmic vessels engorgement leading to the classic triad of CCFs: orbital bruit, chemosis, and pulsating exophthalmos 4-7. Pressure can also be transmitted to the contralateral cavernous sinus through the presence of intercavernous bridging vessels 8, cause bilateral CN deficits and ocular findings, as is seen in this case report.

Most of CCFs follow blunt or penetrating trauma to the head 7,9,10, with only one quarter happening spontaneously (often from ruptured aneurysms) 11. Most cases described in the literature are secondary to closed head injuries related to motor car accidents 7,12, but as seen in this case, any form of closed head injury can result in a CCF. Symptom onset is naturally postponed until venous hypertension reaches a critical level, often days to weeks following the initial insult.

CCFs can be classified using the Barrow Classification System. Type A fistulas involve a direct connection between the intercavernous part of the internal carotid artery (ICA) and the cavernous sinus, whereas types B, C, and D are indirect connection between either dural branches of the ICA or external carotid artery and the cavernous sinus 7,13. Type A CCFs are common in young males, as this demographic has a higher incidence of closed head injuries, whereas indirect CCFs occur more often in the elderly 13.

It is important to recognize direct (type A) and indirect (types B-D) fistulas because of the prognostic significance. Direct CCFs are commonly high flow and give rise to significant venous hypertension, while indirect fistulas...
tend to be low flow. These low-flow lesions usually have fewer and less severe symptoms, they recover with time, and often can be medically managed. In contrast, surgical option is recommended for all high-flow lesions as these can result in intracranial hemorrhage, vision loss, and even life-threatening epistaxis.

The diagnosis of CCFs is based on clinical symptoms as well as neuroimaging. A Computerized tomography scans of the head without contrast may show proptosis, engorgement, and tortuosity of the superior ophthalmic vein, and affected cavernous sinus expansion. MRI images will typically show similar but particularly useful in categorizing CCFs. If a direct, high-flow CCF is recognized, the choice of treatment is endovascular embolization. When the lesion has been embolized, pressure within the cavernous sinus will normalize and symptoms will begin to recover.

REFERENCES