A case of delayed carotid cavernous fistula after facial gunshot injury presented as loss of vision with symptom resolution after endovascular closure procedure

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ABSTRACT

Carotid cavernous fistulas (CCFs) are abnormal connections between the carotid artery and the cavernous sinus (CS), and can occur as a result of blunt and penetrating head injuries. While occurrence is rare, diagnosis can be made in the emergency department. Described in the present report is the case of a 26-year-old man who presented with complaints of pain, redness, blurred and loss of vision in the right eye, and swelling of the upper face due to a gunshot injury he had sustained 35 days prior.

Keywords: Carotid cavernous fistula; endovascular intervention; endovascular treatment; gunshot injury.

INTRODUCTION

Post-traumatic carotid cavernous fistulas (CCFs) are direct communications between the internal carotid artery (ICA) and the cavernous sinus (CS), frequently encountered as a complication of closed head trauma, though occurrence has been reported in relation to penetrating object or gunshot injury.[1] CCFs tend to be diagnosed a few weeks after trauma, and the majority of signs and symptoms result from increased venous pressure in an ophthalmic vein that lacks a valve.[2] While CCFs are not life-threatening, timing of diagnosis is extremely important, as permanent loss of vision may develop within hours or days of initial injury. The most common signs and symptoms are pulsatile exophthalmus, orbital murmur, conjunctival hyposphagma, ophthalmoplegia, orbital pain, and impaired visual acuity.[1,3]

Computed tomography angiography with pathognomonic radiological signs can aid diagnosis and may be used for screening. Precise location of the fistula and nature of the lesion can be studied further with digital subtraction angiography.[4] Endovascular embolization has been the preferred treatment approach throughout the past 2 decades, and surgical treatment remains an option when endovascular treatment fails or is not possible.[5,6] Discussed in the present report is the presentation, pathogenesis, and management of carotid CS fistulas.

CASE REPORT

A 26-year-old man presented to emergency services with pain, redness, and blurred and loss of vision in the right eye. Initial complaints were mild and first noted 15 days prior to clinical presentation. History was unremarkable with the exception of a gunshot injury to the face sustained 35 days prior, which had been managed through conservative measures.

On physical examination, general status was normal, and scar tissue was noted over the right nasal sulcus, indicating bullet entry. Neurological examination revealed no abnormality. Typical murmur was audible at the right orbit, suggesting diagnosis of CCF. Initial radiographic scans revealed a bullet in the right side of the face (Fig. 1). The patient was referred to ophthalmology, and upon examination, corrected visual acuity

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was 0.3 on the right side and 1.0 on the left. Biomicroscopic evaluation was compatible with eyelid edema, chemosis, proptosis, dilated fixed pupils, and negative light reflex in the right eye. Fundoscopic examination revealed marked venous congestion and increased tortuosity, arteriolar thinning, and preretinal hemorrhages in the right eye. Intraocular pressure measured with tonometry was 34 mmHg in the right eye and 16 mmHg in the left.

Color Doppler ultrasonography, orbital tomography with and without contrast on the axial plane, magnetic resonance imaging, magnetic resonance angiography, and digital subtraction angiography confirmed diagnosis of CCF. Examinations revealed fistula between the right ICA and right CS (Fig. 2a). Endovascular treatment was performed via femoral artery access under general anesthesia without complication. Postoperative course was uneventful, and typical murmurs disappeared on auscultation of the orbit. Complaints of pain, redness, and blurred and loss of vision in the right eye markedly improved within 3 weeks of intervention. Postoperative angiographic scans demonstrated complete obliteration of the fistula (Fig. 2b).

**DISCUSSION**

CCFs are identified as direct and abnormal communication between the CS and the ICA, one of its branches, or the external carotid artery.[7] CCFs are rare complications of head trauma, with reported incidence around 0.2–0.3%.[3] Several classifications based on angiographic features (high-flow vs low-flow fistulas), mechanism of onset (spontaneous vs traumatic), morphological features, and angioarchitecture (direct vs indirect fistulas) have been suggested.[8]

Drainage pattern and instant development of fistula are typically associated with the signs and symptoms. As a rule of thumb, direct fistulas usually exhibit more dramatic clinical presentation, not infrequently displaying the so-called “classical” triad of exophthalmus, chemosis, and loss of vision. A study of direct CCFs in a large patient series demonstrated that the most common symptoms at initial presentation were orbital bruit (80%), proptosis (72%), chemosis (55%), cranial nerve VI palsy (49%), complete ophthalmoplegia (24%), and loss of vision (18%).[6,9] In agreement with the literature, many of these clinical findings were observed in the present case.

CCFs may go undiagnosed after major craniofacial trauma, and eyelid auscultation for potential murmur is an appropriate clinical approach. CCFs are commonly accompanied by diplopia due to etiology of ischemic or compressive mechanical cranial neuropathy, as well as restricted orbital motion inside the eye socket secondary to venous hypertension.[10] Both direct and indirect fistulas with retrograde cortical venous drainage could lead to intracranial bleeding. The latter is a particularly ominous occurrence, with a high rate of rebleeding over a short time in cases of direct CCF. Therefore, appropriate therapy should be initiated at once if such a devastating complication occurs.[11]

Computed tomography and magnetic resonance imaging may reveal indirect signs of these fistulas, including engorgement of the CS region or abnormally dilated venous segments. Nevertheless, conventional transluminal angiography remains the gold standard for both detection and typing of CCFs. Complete and technically correct cerebellar angiography should provide information regarding internal and external carotid supply, and delineate contralateral side and posterior circulation.[12,13]

In instances of traumatic fistula, intervention is required in urgent conditions such as progressive loss of vision, intolerable murmur, and headache, as well as in cases of traumatic aneurysm showing signs of dilatation behind the CS, hemiplegia secondary to intracranial hematoma, impairment of cortical venous drainage, severe epistaxis, or intraocular pressure exceeding 40 mmHg. Dural sinus fistulas may spontaneously regress in 20–50% of cases. In cases not requiring urgent intervention, carotid jugular compression may be another palliative treatment option.[14]
In cases of traumatic CCFs, embolization of the fistula by transarterial placement of detachable balloons, platinum coils, polyvinyl alcohol particulates, and liquid embolic agents has become preferred procedure, while combined approaches including stenting, either alone or with coil placement, can also be used.[17,18] While transvenous embolization is the preferred approach in cases of indirect CCF, it also serves as an alternative approach when arterial route has failed in cases of direct CCF.[19] Possible complications of arterial approach include cerebral ischemia or infarction due to displacement of embolic material, and arterial dissection or formation of pseudoaneurysm due to arterial wall injury.[20]

Surgical treatment of CCFs must be limited to cases in which endovascular treatment fails or is not possible. Techniques may include the placement of packing inside the CS to occlude the fistula, suturing or clipping the fistula, sealing the fistula with fascia and glue, and/or ligation of the ICA.[18]

Described in the present report was a case of traumatic carotid CS fistula successfully treated with advanced radiological techniques and interventions in a short period of time. The potential for occurrence of CCF should be kept in mind following facial gunshot injuries, in an effort to avoid ocular and cerebral complications.

Conflict of interest: None declared.

REFERENCES

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