Traumatic carotid cavernous fistula associated with a mandibular fracture


Abstract. Carotid cavernous sinus fistula is an abnormal vascular interconnection between a branch of the carotid artery and the cavernous sinus. This is an uncommon complication of craniofacial injuries, as it occurs in only 0.17–0.27% of cases, according to the literature. The differential diagnosis should include superior orbital fissure syndrome, orbital apex syndrome, orbital haematoma and cavernous sinus thrombosis. Specific diagnosis is usually not complicated but must be confirmed by angiography, because the complications of CCSF, although infrequent, are potentially life threatening. They may include intracranial hemorrhage, worsening pulsatile proptosis, exposure keratitis, neovascular glaucoma, blindness, cranial nerve deficits and fatal epistaxis. In minor cases, spontaneous healing, probably by thrombosis, can be observed.

Of 1800 patients suffering maxillofacial injuries (March 1999 to December 2004) who presented for treatment at the authors’ department, the first case of CCSF is presented as follows.

Case report

An 18-year-old white woman was referred for evaluation of injuries sustained in the craniofacial area, resulting from a motor vehicle accident. The patient had been in the intensive care unit for 2 days under the care also of the Neurosurgery Department. The patient was alert and oriented. In the maxillofacial region, there was periorbital edema and ecchymosis on the left side, left exophthalmos and subconjunctival hemorrhage, a closed laceration in the left frontal area, absent ophthalmoplegia and diplopia, normal visual acuity, malocclusion and mandibular crepitation.

Mandibular postero-anterior radiographs demonstrated left parasympyseal and body fractures. Head and face computed tomographic scan without contrast revealed no intracranial bleeding or other
cerebral pathology. No cranial, basilar skull or other fracture could be observed. The first diagnosis was left parasymphyseal and mandibular body fractures accompanied by a contusion in the left orbital area, without fractures. After 1 day the exophthalmos increased and the patient was unable to abduct the left eye in left lateral and superior gazes (Fig. 1). Diplopia was detected, and the patient was sent for ophthalmologic evaluation. Fundoscopic examination showed no disturbances.

Two days after first referral, extraocular movements were totally absent. Progressive chemosis and exophthalmos gave the left eye an irritated aspect, and the increased congestion and ptosis caused the eyelid to close completely. Magnetic resonance angiographic scanning was performed in order to diagnose an intracranial arterial aneurysm, suspected of being the cause of the subarachnoidal hemorrhage, but none was found. Neurosurgeons were also consulted, and clinically, on frontal auscultation, the so-called machinery-type murmur, pathognomonic for CCSF, could be heard. The patient was sent to the Hemodynamic Center for Internal Carotid Artery Angiography (Fig. 2).

A balloon catheter was introduced into the fistula through the femoral artery. The catheter was inflated with air to obstruct the arterial blood flow before leakage into the cavernous sinus, allowing flow control by reduced pressure (Fig. 3). Ten days after the CCSF was embolized, the patient was submitted to open reduction and internal fixation of the mandibular fractures, under general anesthesia, and was allowed to leave the hospital on the following day. Development was normal. The mandibular arch was completely restored and function re-established. Although the signs and symptoms of CCSF are often rapidly resolved, progress in this case was slow. Posis was completely resolved, and extraocular movements and vision were restored without disturbances within 90 days (Fig. 4).

Discussion
A CCSF may develop spontaneously, by rupture of a carotid aneurysm within the cavernous sinus, or result from trauma. The trauma may be direct, such as transorbital penetration, or indirect, such as impalement by a bone spicule or from fracture of the middle cranial fossa floor with a resultant tear in the muscular wall of the internal carotid artery and bleeding into the sinus8,9. In this reported case, head and facial trauma associated with a sudden impact of the skull against a rigid surface caused brain deceleration inside the cranium. This indirect trauma to the cavernous portion of the internal carotid artery resulted in tearing of the muscular wall of the artery and bleeding into the sinus.
The pressure gradient resulting from a CCSF creates a retrograde flow into the ophthalmic veins, with various associated signs and symptoms. Patients may have exophthalmos, chemosis, extraocular palsies, headache, ocular discomfort, or hear a noise synchronous with the pulse. The most striking symptoms are the ‘bruit de diable’ or ‘machinery-type murmur’, frontal headache and ophthalmoplegia. These symptoms may be present directly after the trauma or event, or may develop several days, weeks or months later, depending on the hemodynamic changes that are determined by the size and diameter of the fistula.

The abducens nerve (VI) is affected twice as often as the oculomotor (III) and trochlear (IV) nerves, probably because of the extreme medial position of the nerve in the sinus. Diplopia will also be noted as a result of the ophthalmoplegia and proptosis.

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**Fig. 3.** Angiography after embolization.

**Fig. 4.** Complete resolution with restoration of extraocular movements.
A complete history and careful examination should lead to the correct diagnosis. The case reported was remarkable in that it occurred in the absence of a cranial base fracture, and showed superficial periorbital contusion accompanying an isolated mandibular fracture. The patient also showed all signs and symptoms of CCSF, but the final diagnosis was difficult because of the absence of zygomatic, maxillary, cranial or basilar skull fractures. Definitive diagnosis was confirmed by bilateral carotid artery angiography.

References


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