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Case report

Bilateral carotid-cavernous fistula following head trauma: Possible worsening of brain injury following balloon catheter occlusion?

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Abstract

A 68-year-old woman developed right pulsatile exophtalmos in the early course of facial and head trauma. Investigations by visual evoked potentials suggested the presence of a bilateral prechiasmatic lesion. Intraocular pressure rapidly increased and a high-flow carotid-cavernous fistula was evident at carotid angiography. Immediately after the treatment of the right fistula by balloon embolisation, a systolic bruit was noted over the left eyeball and angiography disclosed the presence of a left carotid-cavernous fistula that was treated by the same approach. The patient developed brain oedema leading to death and we postulated that reestablishment of normal cerebral perfusion after abrupt closure of the fistulas may have played a deleterious role.

Keywords: Carotid-cavernous fistula; Head trauma; Visual evoked potentials; Embolisation; Perfusion pressure breakthrough

Carotid-cavernous fistulas are rare complications of craniofacial trauma, which may lead to serious complications. Clinical signs are often delayed and the diagnosis is not easy in comatose patients. We report a case of bilateral carotid-cavernous fistula complicating head trauma and leading to a fatal outcome.

1. Case report

A 68-year-old woman was admitted in emergency after a traffic road accident. The initial Glasgow Coma Scale (GCS) was 4/15. Bilateral peri-orbital hematoma was associated, with right dilated and fixed pupil; there was no evidence of exophtalmos. Cerebrospinal fluid rhinorrhea was also noted transiently. Neurological status improved gradually soon after admission and the patient became reactive to stimuli. As the GCS had risen to 10/15, an invasive monitoring of intracranial pressure was not proposed. The initial brain computed tomodensitometry (CT) disclosed a post-traumatic subarachnoid haemorrhage, with also multiple contusions in the left fronto-temporal and right frontal areas; however, there was no sign of brain oedema (Fig. 1). The patient had a history of bilateral cataract and diminished vision at the left eye due to degenerative lesions of the macula; signs of macular degeneration of the retina were found at fundoscopy. Visual evoked potentials (VEPs) were performed the day following admission. Flash VEPs were abnormal with absence of cortical response on both hemispheres after stimulation of the right eye and absence of cortical response on the left occipital lead after stimulation of the left eye; they were well defined but delayed on the right occipital lead after stimulation of the left eye. This pattern suggested the presence of a bilateral pre-chiasmatic lesion predominating on the right side or of an opto-chiasmatic lesion. A "second look" at the initial brain CT failed to reveal any lesion of the orbita or optic duct, but the right superior ophthalmic vein appeared to be dilated. On day 3, while neurophysiological abnormalities (VEPs) seemed to slightly decrease, pulsatile exophtalmos and abnormal bruit were noted for the first time over the right eyeball. A carotid-cavernous fistula was therefore expected. Intraocular pres-

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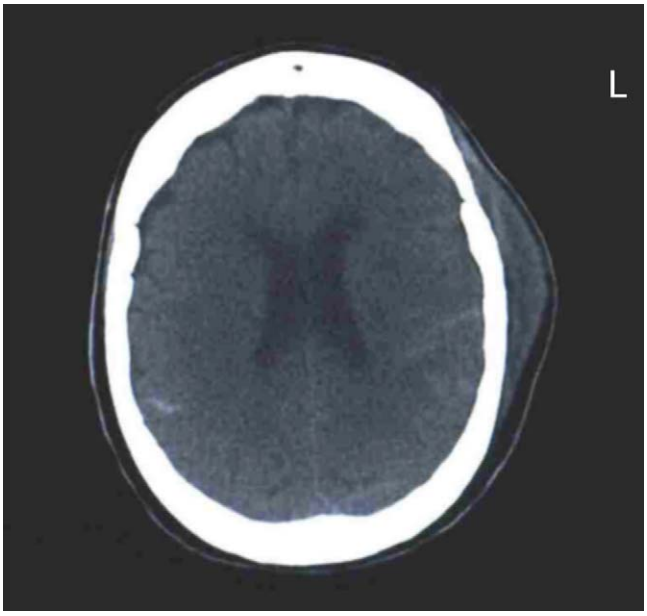

Fig. 1. Brain computed tomodensitometry obtained on admission.

sure was measured at 50 mmHg in the right eye (10 mmHg in the left). Acetazolamide and topical beta-blocking agents were prescribed to decrease the ocular pressure. The control brain CT obtained after 72 h remained unchanged. The patient underwent a carotid angiography on day 7: it showed a right high flow carotid-cavernous fistula. Embolisation was performed using two detachable balloons (DRT/GVB 15, 16 [2], 17 from BALT, France) by transarterial approach (Fig. 2a and b). Heparin was administered during the procedure (5000 units as a bolus dose, followed by a continuous infusion at a rate of 1000 U/h according to the results of the activated partial prothrombin time. Immediately after the procedure, while the bruit had disappeared at the level of the right eye, auscultation of left supraorbital area revealed for the first time a systolic bruit. The angiography revealed a left carotid-cavernous which was therefore treated using the same technique (Fig. 2c and d). On the post embolization angiograms (Fig. 2b and d), a narrowing of both carotid arteries was observed, probably due to abutting of the detached balloons into the arterial lumen.

Immediately after this procedure, intraocular pressure fell to 13 mmHg in the right eye and to 16 mmHg in the left. The worsening of the neurological status became evident only 24 h later, after the complete withdrawal of the sedative drugs, which were used during angiography. The patient was unresponsive to painful stimuli. No bruit could be heard over both eyes and intraocular pressure remained low. A brain CT scan revealed no recent intracranial bleeding, but diffuse brain swelling was noted. The balloons were correctly positioned (Fig. 3). Despite intensive therapy, the patient died 5 days later from intracranial hypertension. A post-mortem examination was obtained. Meningitis was ruled out by the macroscopical findings. No evidence of recent intracerebral bleeding was found, neither of focal ischemic lesions. There was also no sign of migration of the balloons neither of thrombosis in the cavernous sinus; the internal carotid artery was permeable at both sides. A fracture was seen on the left part of the sphenoid boon, extending from the processus clinoideus posterior to the facies temporalis of ala major. The autopsy findings were consistent localized hematomas (left frontal and temporal) with diffuse brain swelling and subsequent brain herniation (subfalcine, uncal and tonsillar). Microscopic examination was not obtained.

2. Discussion

Although carotid-cavernous fistula is the most common type of intracranial arteriovenous fistulas, it remains a rare complication of head trauma with a global occurrence rate ranging from 0.17 to 1.01% [1,2]. Spontaneous fistulas are occasionally observed following Ehlers-Danlos syndrome [3].

Usually, carotid-cavernous fistulas are found after fractures of the middle third facial bone, but they may also occur in patients who have sustained fractures of the frontal, cranial base, mid facial, and mandibular regions or maxillofacial injuries without evidence of a fracture [4]. Traumatic fistulas tend to be of “high flow” type (Barrow type A), with flow from the internal carotid artery directly into the cavernous sinus [5]. The delay for the occurrence of the clinical symptoms depends upon the size of fistula and the velocity of blood flow. It ranges from a few hours to several weeks [6].

The diagnosis of carotid-cavernous fistula may be difficult in comatose intubated patients, but the clinical examination remains the best way to detect such fistulas, even if the final diagnosis should be made by carotid angiography. In the present observation, the loss of pupillary-light reflex at the right eye was the only sign present on admission and pulsatile exophtalmos with systolic bruit developed only later on.

The complications of carotid-cavernous fistulas are not frequent, but potentially life-threatening. They may include intracranial haemorrhage, worsening pulsatile proptosis, exposure keratitis, neovascular glaucoma, blindness, cranial nerves deficits and fatal epistaxis [7]. In minor cases, however, spontaneous healing, probably by thrombosis, can be observed. The indications of early operative intervention are especially: epistaxis, visual loss, brain ischemia, cortical venous drainage and cavernous varices. Most of these signs are uneasily appraised in comatose patients.

The technique of endoarterial detachable balloon embolisation has become the treatment of choice for traumatic carotid-cavernous fistulas [8] and results in low mortality and morbidity [9].

Several complications may occur after transvascular or surgical closure of carotid or vertebral fistulas [10]. Ischemia may develop secondary to balloon migration, spasm, narrowing, or occlusion of surrounding vessels supplying or draining normal parenchyma. Thrombus formation on catheters or propagation of clot may cause distal embolisation. Such complications can be suspected by clinical or angiographic