Pulsating unilateral exophthalmos due to traumatic aneurysm of the intraorbital ophthalmic artery

Case report

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A case of pulsating unilateral exophthalmos is presented. It was caused by a traumatic aneurysm of the intraorbital ophthalmic artery, secondary to missile injury. Evolution of the aneurysm was observed clinically, by angiography, and by computerized tomography. The differential diagnosis of pulsating exophthalmos and the pathogenesis of such aneurysms are briefly discussed.

KEY WORDS • pulsating exophthalmos • unilateral exophthalmos • traumatic aneurysm • ophthalmic artery • missile injury

Pulsating exophthalmos can be caused by traumatic or spontaneous carotid cavernous fistulas leading to secondary intraorbital varices, primary intraorbital varices due to trauma, orbital or remote arteriovenous malformations, highly vascular tumors of the orbit, spontaneous or traumatic “bloodcysts” of the orbit with intracranial connection, traumatic arteriovenous fistulas between the ophthalmic artery and one of the ophthalmic veins and any primary or secondary bone defect of the orbital cavity leading to transmission of the cerebrospinal fluid pulsation.

A review of the available literature has revealed no clearly defined case of traumatic intraorbital ophthalmic artery aneurysm with or without pulsating exophthalmos. We report one such case that occurred secondary to missile injury and which was documented by repeated angiography and computerized tomography scanning.

Case Report

On November 5, 1982, a 34-year-old soldier was transferred from the battlefield to our hospital, 10 hours after missile injury. He was awake but confused and obeyed only simple commands. He had a linear scalp laceration about 5 cm long in the right pterional region, with burned margins caused by tangential injury of a shell fragment or a bullet. He had severe bilateral peri-orbital edema and ecchymosis. He appeared blind with dilated pupils, neither pupil reacting to light. Extraocular movements could not be evaluated. He had a mild left hemiparesis with an equivocal left plantar reflex.

Examination. Initial plain x-ray films of the skull and optic foramina were normal, except for fracture of the greater sphenoid wing on the lateral wall of the orbit, with vaguely identifiable inwardly driven bone chips. A plain CT scan of the brain performed on the following day showed mild generalized brain edema with contusion hemorrhage of the right temporal lobe. These changes had resolved markedly on the next plain CT scan performed 10 days later, while he was under conservative management with steroid, antibiotic, and antiepileptic drugs. By November 20, the peri-orbital ecchymosis had subsided gradually, but he remained totally blind with fixed dilated pupils. The extraocular movements were full in both eyes. Funduscopes revealed pale discs bilaterally, without any retinal or vitreous hemorrhages.

To investigate for possible traumatic lesions of the optic chiasm and/or circle of Willis as the cause for his total blindness, bilateral carotid angiography was performed on November 22, 1982. There were no pathological findings associated with the intracranial vessels, but a small aneurysm was vaguely visible within the apex of the right orbital cavity along the course of the ophthalmic artery (Fig. 1). During the subsequent week,
Traumatic ophthalmic artery aneurysm

FIG. 1. Initial right carotid angiogram, anteroposterior view (A) and lateral view (B), showing a small aneurysm in the apex of the right orbit (arrows).

FIG. 2. Sequential right carotid angiograms performed after development of exophthalmos, lateral projections, showing slow filling of the enlarged aneurysmal sac (arrows).

The patient developed a right-sided pulsating exophthalmos rather rapidly. The eye protruded directly forward some 6 to 7 mm. There was no limitation of extraocular movements and no audible bruit was detected. Repeat right carotid angiography on November 28 disclosed that the previously noted aneurysm had enlarged significantly and the slow turbulence of blood filled the lobulated aneurysmal sac mainly in the late capillary phase (Fig. 2). Contrast CT scanning at this time clearly showed the aneurysm lodged within the apex of the orbital cavity. In-driven bone chips could also be seen in juxtaposition to the aneurysm wall (Fig. 3A, B, and C).

Operation. A right frontotemporal craniotomy was performed and the right ophthalmic artery was clipped just at its origin from the internal carotid artery. No chiasmal injury was seen at the time of this exploration. The patient's postoperative course was uneventful and his exophthalmos subsided gradually. A control right carotid angiogram 5 days later demonstrated proper clipping of the ophthalmic artery. Control CT scanning showed a collapsed and clot-filled aneurysm sac which did not enhance after contrast injection (Fig. 3D). When visited again 3 months later he was still blind in both eyes with symmetrical eyeballs. Control CT scanning was unchanged.

FIG. 3. Computerized tomography scans of the orbit. A: Preoperative nonenhanced scan revealing a slightly hyperdense irregular retrobulbar mass. B: Preoperative scan after contrast perfusion revealing a lateral orbital wall fracture and in-driven bone fragment on the right side (arrow). C: Preoperative scan after contrast perfusion showing exophthalmos on the right and an enhanced retrobulbar mass. D: Postoperative contrast-enhanced scan. There is reduction of the exophthalmos and in the size and density of the aneurysm.
Discussion

Missile injury has been reported as an unusual cause of false aneurysms of the intracranial or extracranial carotid, vertebral, and peripheral arteries and arteriovenous fistulas. To our knowledge, no such aneurysm has been reported involving the intraorbital ophthalmic artery. Traumatic aneurysms can be true, false, mixed, or of dissecting type, depending upon the extent and severity of the primary damage to the arterial wall. It is hard to distinguish the type of aneurysm involved in this case without pathological confirmation.

Clinically, these aneurysms present as expansile pulsating swellings which may compress the neighboring structures and cause symptoms as in this case. The symptoms may occasionally be due to a steal phenomenon or propagation of the thrombus from within the lumen of the lesion into the main vessels and embolization. Finally, the aneurysm may leak or rupture. The time interval between the trauma and the onset of symptoms caused by the aneurysm varies from 5 days to 10 years; in the present case the interval was 2 weeks.

The diagnostic procedure of choice is angiography. Growth of the aneurysm is well shown in this case on repeated angiograms. Computerized tomographic scanning has been shown to be useful for the evaluation of the orbital trauma, by providing visualization of soft-tissue damage, fractures, and embedded foreign bodies. The CT scan had a unique place in the evaluation of this case by showing the close relationship of the small, in-driven bone chips to the aneurysm wall. This raises two possibilities. The bone chips might have initially injured the arterial wall leading to clot formation which later turned into a false aneurysm. Less probably, repeated pulsations of the artery against the piece of hard bone led to arterial wall injury and formation of a true aneurysmal sac.

References