Globe Tenting as a Result of Head Trauma

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Summary: Ocular tenting in the setting of acute trauma, inflammatory processes, or carotid cavernous fistulas is reported to be associated with a poor outcome with respect to visual acuity. Therefore, acute decompressive intervention is recommended for these patients. We report a case of acute posttraumatic ocular tenting with acute visual compromise that resolved over 4 days with conservative management and intravenous corticosteroid therapy.

Index terms: Orbits, fractures; Eyes, computed tomography

Posterior globe tenting on computed tomographic (CT) scans has previously been reported to be associated with a poor outcome with respect to visual acuity. It is usually seen in the setting of acute intraorbital inflammatory processes, but has also been reported in connection with neoplasms, traumatic carotid cavernous fistulas, and orbital varices (1).

Case Report

A 37-year-old man was involved in a high-speed motor vehicle accident at which time he sustained trauma to the facial soft tissue, multiple maxillofacial fractures, including the orbit, and dislocation of the right hip. He was stabilized at an outside hospital and then transferred to our institution.

Upon arrival, the patient was alert and cooperative. His medical history was remarkable for a previous traumatic facial fracture requiring surgical reduction and fixation several years earlier. Both the patient and his family reported no history of vision loss. Physical examination revealed extensive lacerations and swelling of the facial soft-tissue with mild proptosis of the left eye. Visual acuity was difficult to assess because of periorbital edema, but it was concluded that he had no light perception in the right eye and 20/800 vision in the left eye. The pupils were equal but the right pupil was nonreactive to light. Intraocular pressure was 9 mm Hg in the right eye and 16 mm Hg in the left (normal, 10 to 20 mm Hg). An external eye examination revealed mild subconjunctival hemorrhage in the left eye and trace hyphema in the right eye without evidence of globe rupture. Fundoscopic examination showed vitreous hemorrhage in the right eye and a small amount of intraretinal hemorrhage lateral to the optic nerve head in the left eye.

An orbital CT scan showed extensive maxillofacial fractures involving all walls of the maxillary sinuses, the hard palate, the left medial and lateral pterygoid plates, the right lateral pterygoid plate, the left zygoma, the posterolateral walls of the orbits bilaterally, the lamina papyracea bilaterally, the ethmoidal air cells, and the sphenoidal and frontal sinuses. Moderate displacement of fracture fragments into the left orbit was seen, as were left-sided intraorbital streaky soft-tissue changes consistent with foci of intraorbital hemorrhage and/or edema. The left globe was proptotic, with straightening (tensing) of the left optic nerve accompanied by tenting of the posterior globe (Fig 1A). The angle of the posterior globe, measured as described previously (1), was determined to be 118° (normal, >130°). The right globe showed vitreous hemorrhage but was without globe tenting (posterior globe angle of 135°). A minimally displaced fracture fragment extending into the right optic canal could not be ruled out.

The patient was admitted to the trauma intensive care unit for further treatment. In the absence of a discrete intraorbital mass or hematoma, the ophthalmology service elected to initiate high-dose methylprednisolone therapy (400 mg/hour intravenously) to help alleviate swelling in the left intraorbital compartment and to treat potential edema associated with possible compression of the right optic nerve in the right optic canal.

The patient rapidly regained vision in his left eye, such that at 72 hours after admission, visual acuity was 20/30. Four days after admission, a repeat orbital CT scan (Fig 1B) revealed complete resolution of the left globe tenting with a posterior globe angle of 145°. No fracture in the region of the right optic canal could be identified. The facial fractures were surgically repaired and the patient was eventually discharged home. At follow-up 6 months later, the patient had 20/20 vision is his left eye (but still no light perception in his right eye).

Discussion

Globe tenting is a change in the shape of the posterior globe, resulting in a conical appearance with the apex at the site of the optic nerve insertion. This configurational change in the

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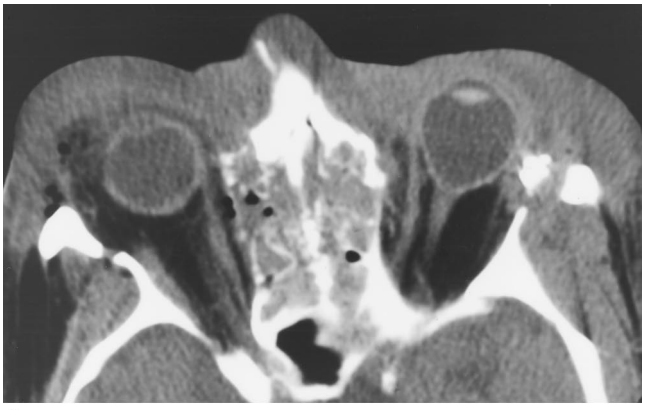

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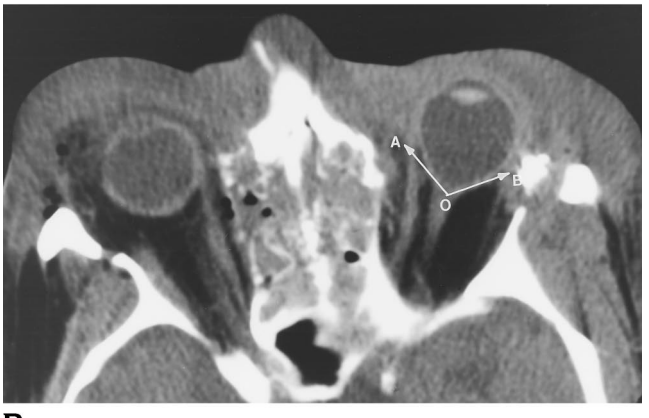

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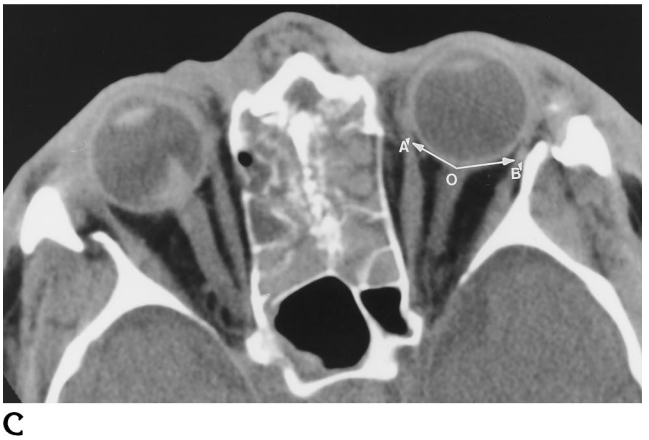

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Fig 1. Globe tenting caused by trauma.

A, CT scan without annotation.

B, CT scan with annotation obtained within hours of the patient's injury (left eye vision, 20/800) shows maxillofacial posttraumatic changes and a left posterior globe angle (*A-O-B*) of 118° (normal, >130°).

C, Repeat CT scan obtained 4 days later, after high-dose intravenous corticosteroid therapy (left eye vision, 20/30), shows significantly less exophthalmos. The left posterior globe angle (*A'-O-B'*) is now within normal limits (145°). Right vitreous hemorrhage is also seen.

globe is associated with an acute or subacute rise in intraorbital tension due to an intraorbital mass effect (1). Globe tenting has not been reported in association with slowly developing intraorbital masses (ie, Graves ophthalmopathy), presumably because of the optic nerve's ability to stretch as it accommodates to the gradual increase in tension.

Tenting has been seen as a qualitative feature on orbital CT scans in patients with rapidly developing intraorbital masses, most commonly abscesses (2, 3). In 1989, Dalley et al (1) attempted to characterize this abnormality fully and to determine its clinical significance. These authors reported a series of 11 cases of globe tenting, all of which involved the acute or subacute development of an intraorbital mass. These masses were primarily inflammatory (ie, subperiosteal abscesses), neoplastic (implantation cysts, hemorrhage into lymphangioma), or vascular (orbital varix, traumatic carotid cavernous fistula).

Dalley and coworkers also attempted to quantify the extent of globe tenting and to correlate this with clinical outcome (1). They measured posterior globe angles by drawing tangents to the medial and lateral aspects of the sclera at the site of optic nerve insertion. From a series of 62 normal CT scans, they defined a posterior globe angle of less than 130° as abnormal (ie, greater than 2 standard deviations from the mean). Using this measurement, these authors showed that patients with angles between 120° and 130° had uniformly excellent clinical outcomes (n = 4), whereas patients with angles less than 120° had less satisfactory outcomes (three of six patients were left with no light perception). From these data, the authors concluded that "a posterior angle of less than 120° constitutes a surgical emergency requiring intervention to preserve visual function."

Vision loss due to globe tenting may be the result of several different mechanisms of injury to the optic nerve. Intraorbital mass effect may directly compress the optic nerve, causing ischemia and structural disruption. Acute stretching of the optic nerve may itself cause ischemic nerve injury, although the optic nerve is able to accommodate this stretching if it develops gradually. Finally, any traumatic event that causes globe tenting may also result in direct contusion of the optic nerve (1).

The differential diagnosis of a decreased posterior globe angle includes presence of a

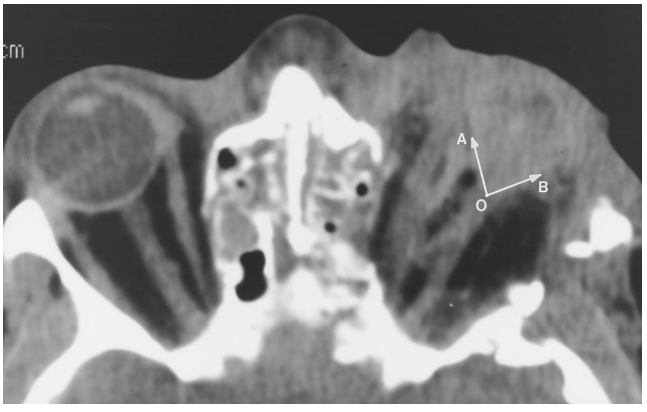

Fig 2. Pseudo–globe tenting caused by direct ocular trauma in a different patient. Posterior globe angle (*A-O-B*) measures 90° (normal, >130°). This globe was removed because of irreparable damage (frank globe rupture); therefore, the observed decrease in the angle of the posterior globe was probably directly attributable to the ocular injury and not to increased intraorbital pressure.

coloboma (a defect of the posterior aspect of the globe in the region of the optic nerve insertion); direct trauma to the globe resulting in loss of intraocular contents and thus "collapse" of the globe in a pattern mimicking increased tension on the back of the globe by the optic nerve (Fig 2); and a slow-growing mass that gradually deforms the posterior aspect of the globe in the region of the optic nerve insertion (however, such a deformity should be asymmetric with respect to the optic nerve and therefore should not be confused with true tenting).

Our patient differs from previous reports of globe tenting in that the deformation of the left globe was due to acute posttraumatic changes, including diffuse intraorbital edema and possibly hemorrhage, without a clearly demarcated mass such as an abscess or discrete hematoma. With a posterior globe angle of 118°, this patient was probably at increased risk for persistent vision loss. Despite the initial severity of his condition, the globe tenting (and the intraorbital edema responsible for it) cleared nearly completely over the first 72 hours. How much of this was due to the high-dose corticosteroid therapy versus the natural course of the injury is uncertain.

In the immediate posttraumatic period, ophthalmologists rely primarily on their examination to guide diagnosis and intervention; thus, if a trauma patient is unable to cooperate for an examination and local injuries limit physical evaluation of the orbits, the identification of globe tenting on a CT scan may be the only finding suggesting that a patient's vision is at risk. In such an instance, the radiologist may be the first to recognize the need for emergency clinical intervention.

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