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Transorbital Herniation: An Unusual Mechanism of Intracranial Decompression Following Trauma

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Summary: This case illustrates decompression of contused, edematous brain through a "blow-in" fracture of the orbital roof, causing a different form of brain herniation.

Index terms: Brain, hernia; Skull, fractures; Orbits, fractures

Orbital roof "blow-in" fractures in adults are rare injuries (1, 2). They are usually associated with complex facial injuries and may be associated with a number of complications ranging from ocular laceration to cerebrospinal fluid rhinorrhea (1, 3, 4). Traumatic encephaloceles following orbital roof fractures are even rarer complications, having been reported with computed tomography (CT) only twice in the ophthalmologic literature (5, 6) and twice in the neurosurgical literature (7, 8). Although brain herniations are well recognized neuroradiologically, this report advances a case of transorbital herniation of the brain as another mechanism of decompression of focal cerebral edema, which superseded more characteristic pathways.

Case Report

A 32-year-old man was admitted to hospital after sustaining face and head injuries as an unbelted driver in a dune buggy. An initial physical exam revealed a comatose patient with a left temporal depressed skull fracture, bilateral orbital chemosis, and periorbital ecchymoses with normal appearing optic fundi. He minimally withdrew from noxious stimuli and had normal cranial nerve testing on limited examination. An emergency axial CT scan, 10-mm thick slices (G.E. 9800, Milwaukee, WI) of the head and upper face, performed soon after the accident, revealed a left temporal epidural hematoma, a depressed fracture of the left temporal and greater wing of sphenoid bones, a right frontal lobe contusion (Fig. 1A), and various fractures of the face including a right superior orbital rim fracture. Two days after evacuation of the left epidural hematoma, the patient developed contralateral proptosis. Axial CT using 3-mm slice thickness and coronal reconstructions (Figs. 1A, 1B, and 1C) demonstrated a large inferiorly displaced fracture of the right orbital roof that was not visualized previously. Portions of "pulped" frontal lobe had herniated into the superior right orbit, depressing the globe and stretching the optic nerve and ophthalmic artery (Fig. 1D). An open reduction of the orbit was performed with elevation of a 3-cm bone fragment of the orbital roof and debridement of the inferiorly herniated frontal lobe, restoring normal anatomic relationships. Clinical examination postoperatively documented absence of optic nerve injury.

Discussion

The term "blow-in" fracture was initially coined by Dingman (9) in 1964 to describe a specific fracture of the orbital floor and has become more generally used to describe a fracture of any orbital wall where the fragments are intraorbitally displaced. Schultz (2) estimated orbital fractures to occur in 5% of patients with facial bone fractures. However, Chirico et al discovered only 7 blow-in fractures in 3,666 consecutive adult trauma patients studied (3). Usually these fractures are associated with complex facial injuries but may be isolated, especially in children (10). Messinger et al (4) described three types of orbital roof fractures in children: nondisplaced type I fracture (48%); superiorly displaced type II fracture (20%); and inferiorly displaced type III fracture (32%). Only the patients with a type III injury exhibited the typical inferior intraorbital displacement of fragments that is seen in the classic blow-in fracture. Several mechanisms may cooperate to create a blow-in fracture of the orbit but the mechanism of injury remains incompletely understood (1, 4, 10–12). Anderson et al (13) demon-

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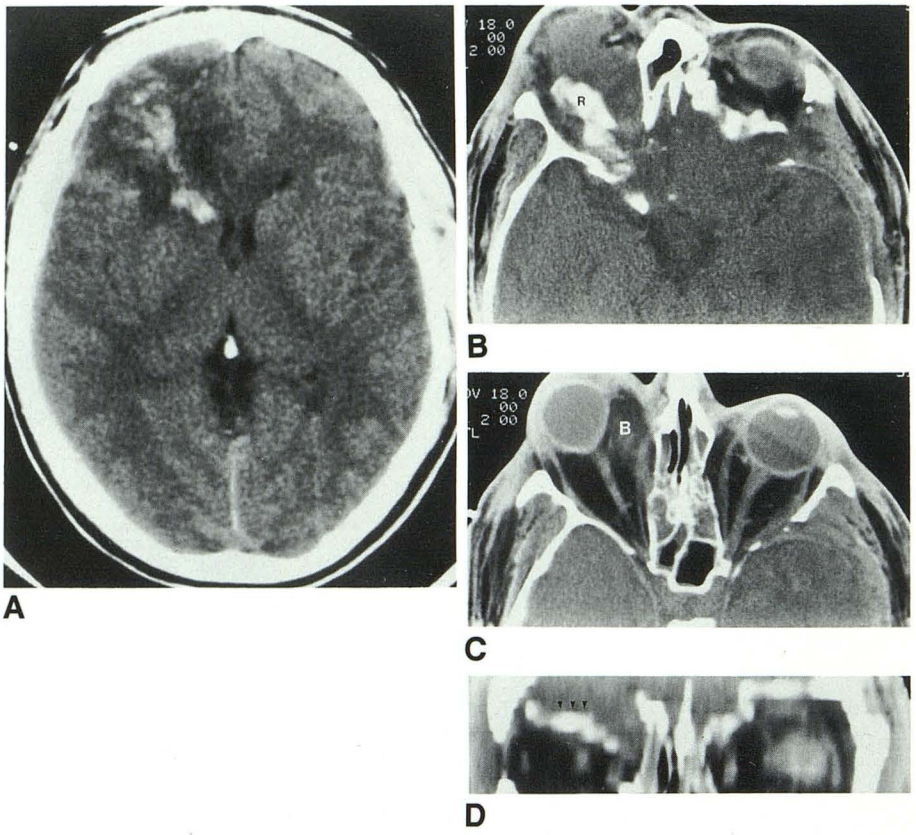

Fig. 1. *A*, Repeat NCCT 4 days post-injury reveals continued presence of extensive hemorrhagic contusion in the right frontal lobe. Edema extends into the genu of the corpus callosum indicating shear injury. There has been partial resolution of the initial midline mass effect during a period in which mass effect might have been expected to increase. This is because a portion of the frontal lobe has decompressed into the orbit.

B and *C*, Axial images through the right orbit reveal a large intraorbital bony fragment from the fractured orbital roof (*R*). Herniated brain (*B*) acts as a soft tissue mass deforming the globe, stretching the optic nerve and creating severe proptosis.

D, Coronal reconstruction reveals extensive depression of the orbital roof (*arrows*) by an extraconal mass representing herniated "pulped" frontal lobe.

strated by sophisticated holographic techniques that force applied to the superior orbital rim is transmitted to the roof leading to an orbital roof fracture without necessarily fracturing the rim. This concept can explain the presence of an orbital roof fracture with an intact supraorbital rim in all seven of the patients described by Chirico (3) and the 44% incidence of isolated roof fractures in the 32 pediatric patients described by Greenwald and Boston (10). The relative thinness and weakness of the orbital roof may also allow a remote force transmitted through the brain to fracture the roof (8).

A large number of complications of orbital roof fractures have been described including displaced bone fragments, orbital hematoma, cerebrospinal fluid rhinorrhea, pneumocephalus, dural fistulas, meningitis, and carotid cavernous fistula (14). A cephalocele might be anticipated complication of orbital roof fractures, but only four cases have been reported on CT (4, 6–8) and none have been reported on MR to our knowledge. Ocular complications occur in 14%–29% of orbital roof fractures (14). Since direct force to the intraorbital soft tissues is not thought to be the mechanism of injury in blow-in fractures, ocular injuries are not typically part of the spectrum of complications expected from an *isolated* orbital roof fracture (3, 10). However, force can be transmitted to the optic canal or vessels supplying the optic nerve, leading to decrease visual acuity or blindness. Gaze palsies may be created by mechanical or neural injury.

Antworth (5) reported a single case of traumatic encephalocele in a 39-year-old man with an isolated orbital roof blow-in fracture. Even though orbital roof fractures in children are felt to be more common than previously believed, Greenwald et al found post-traumatic encephaloceles in only two of 36 fractures in pediatric patients in their 5-year survey (6, 10). In these children, this complication was assumed to be secondary to a "growing" fracture from subarachnoid cerebrospinal fluid pulsations, since they were observed months after the initial injury.

After severe intracranial injury with the development of sufficient edema, the brain may herniate along several now classic pathways as initially described neuroradiologically by Azambuja et al (15) and, more recently, pathologically illustrated by Mastri (16). In addition to the classical: *tentorial* (descending or ascending); *occipital* (tonsillar); *sphenoidal* (transalar); and *lateral* (subfalcine) pathways, our case describes another brain herniation pathway—that of transorbital decompression. In our case, "pulped" frontal lobe

extended along the pathway of herniation depressing the orbital roof, compressing the orbital contents and leading to proptosis. Our patient presented with complex facial and calvarial fractures and cerebral injuries, including a contralateral epidural hematoma. Despite decompression of the extra-axial collection, due to the extent of intraparenchymal hemorrhage and edema present on our patient's initial CT scan, subfalcine or transalar herniation of the frontal lobe resulting from increasing edema in the first few days after the injury might have been expected. These findings did not develop in our case while subsequent "delayed" herniation of the frontal lobe into the orbit did occur as a means to decompress the increasingly edematous brain.

In conclusion, our case illustrates the decompression of contused edematous brain through a blow-in type of orbital roof fracture. While characteristic brain herniation pathways are well recognized and previously described, in our patient a different form of herniation prevailed.

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