CT Demonstration of Optic Canal Fractures

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Traumatic blindness is a well recognized entity with a guarded prognosis. Previous studies have variously reported the incidence of concomitant optic canal fractures and response to surgical therapy. With the advent of CT scanning, a new technique for study of these severely injured patients has become available. Over a period of 20 months, optic foramen fractures were demonstrated in 10 such patients using finely collimated, high-resolution CT scans. Fractures were easily classified by location, relation to the optic nerve assessed, and associated facial injuries imaged. The technique is easy, rapid, and superior to polytomography in this setting. Possible implications for therapy are discussed.

Visual impairment due to optic nerve injury after blunt head trauma is a well described entity [1–5]. The clinical diagnosis of indirect optic nerve injury rests on an ophthalmologic examination that excludes globe injury and can demonstrate an afferent pupillary defect [1]. Injury to the nerve can occur anywhere along its length, but most such injuries occur in the nerve’s intracanalicular part [1, 2, 5, 6]. Demonstrating a fracture of the canal walls in this setting has been considered definitive proof of significant trauma [1, 7, 8]. Plain films and polytomography have been used in the past to depict the optic canal but with little documented success. We present 10 patients with visual impairment in whom fractures in or very close to the optic canal were diagnosed by computed tomography (CT).

Materials and Methods

We reviewed the CT studies of 10 patients who suffered optic canal fractures associated with head or facial trauma. All the patients experienced visual impairment. Indications for the study varied but included traumatically induced partial or complete blindness and the preoperative assessment of associated orbital fractures. All patients were examined within 1 week of their trauma and before any surgical intervention.

A G.E. 8800 CT scanner was used in all instances. The patients were studied in the supine position with contiguous 1.5-mm slices at 0° to Reid’s baseline. Examination was carried through the entire orbit and in many cases included the entire upper facial skeleton. Using an available software program, images were reconstructed using a high-resolution, bone-detail algorithm (G.E. Target Bone Review). The examinations were performed using a rapid-sequence protocol with automatic table incrementation, which incorporates reduced ampere-second settings to avoid excessive tube heating and minimizes patient motion. The axial data were reformatted into multiple planes through the optic canal, as needed, for better assessment of bony anatomy.

Results

Fourteen separate fractures in 10 patients were demonstrated to be in close proximity to the optic nerve or chiasm. Fractures were all easily identified on axial images and were classified into three categories on the basis of location.
Discussion

CT scanning with the use of a high-resolution reconstruction algorithm allows identification of canalicular fractures and pericanalicular fractures as demonstrated in our 10 cases. Such fractures should be suspected especially in those patients with tripod fractures and visual compromise not explained by globe trauma. CT scanning is superior to polytomography in demonstrating soft-tissue changes in the globe and the orbit and the relation of the intraorbital optic nerve to bony fragments [9–11]. Use of a rapid-sequence dynamic technique reduces the problem of patient motion, and the supine position is simpler and more reproducibly attained in these severely injured patients. CT can also identify associated injury to the adjacent carotid canal, which may produce a false aneurysm or fistula, and can assess damage to the brain itself.

Indirect optic nerve injury was described by Hippocrates: "Dimness of vision occurs in injuries to the brow and in those placed slightly above. It is less noticeable the more recent the wound but as the scar becomes old so the dimness increases" [12]. It is currently estimated that 5%–6% of patients with closed head injuries develop significant visual impairment [2, 5]. Previous reports have demonstrated autopsy evidence of infarction of the optic nerve in its intracanalicular part in many such patients [4]. Actual transection of the nerve or hemorrhage into the nerve sheath or subperiosteally in the optic canal has also been described but is uncommon [4, 5]. Although the exact pathophysiology of indirect optic nerve injury remains somewhat speculative, it is likely that either interruption of the vascular supply by direct arteriolar injury or compressive edema as a result of shear forces during injury result in ischemia of the intracanalicular nerve [4]. This mechanism would explain the particular vulnerability of the intracanalicular nerve and account for the clinical observation of delayed-onset visual loss with its better surgical prognosis [2, 4, 5] (compared to those patients with immediate loss of vision).

The incidence of concomitant fractures of the optic canal in traumatic blindness has been variably reported from 6% to 92% [2, 3, 7, 8]. That such a fracture is not necessary for nerve damage to occur has been demonstrated by Anderson et al. [3], who speculate that skeletal distortion about the canal as well as shearing and stretching forces caused by the momentum of the globe transmitted to the fixed intracanalicular part of the nerve result in enough injury to cause edema and/or infarction. Optic canal fractures when demonstrated can be viewed as more direct evidence of significant injury to the nerve in a location where it is extremely vulnerable, but such fractures are not obligatory in causing blindness. Similar fractures immediately anterior to or posterior to the canal also indicate that the nerve has been placed in peril.

Prognostic implications of the presence of a fracture are unclear in light of the variable incidence of fracture that has been reported in the literature. It is possible that the presence of a fracture implies greater force and hence greater likelihood of irreversible damage. Prognosis also may be influenced by the timing of surgical intervention and by the exact fracture site.
Current therapy depends on a combination of clinical and radiologic data and remains controversial. Fukado [7] has reported improvement of visual acuity in high percentage of patients undergoing decompressive surgery. More limited series in this country have not duplicated these results, and some authors advocate a trial of systemic steroids before surgery [3]. Those patients with delayed-onset visual loss who do not respond or only transiently respond to steroid
administration seem to be the best candidates for optic nerve decompression [3]. Therapeutic implications of the demonstration of a fracture remain unsolved. Surgical decompression was undertaken in two of our cases but was not helpful in restoring vision. In these two cases, the decision to decompress the canal was influenced by the presence of an associated intracranial injury that required surgery (in one, a frontal hematoma; in the other, a cerebrospinal fluid leak) and by the short time interval between injury and the diagnosis of optic nerve injury (both patients were operated on in less than 10 hr). In the other eight patients in our series, optic canal surgery was not undertaken either because of the presence of more life-threatening injuries or because the diagnosis of nerve injury was delayed beyond 24 hr, at which time the possible benefits of surgery were believed to be minimal. Two of these eight patients did subsequently recover some visual acuity without specific therapy. One of the patients who did spontaneously improve suffered delayed onset of visual impairment; in the rest of our 10 patients, visual loss was maximal at presentation. Although delayed-onset visual loss may carry a better prognostic outlook than immediate loss of vision, our experience is too small to substantiate this. Nothing in the radiologic profile of the two patients who improved would have predicted this result.

In addition, patients suffering optic canal fractures should be considered at risk for concomitant carotid artery injury. Arteriography was prompted in three of our patients by the proximity of associated fracture to the petrous and cavernous parts of the carotid artery. In two of these patients, carotid artery injury with carotid-cavernous fistula was demonstrated despite the lack of clinical findings suggesting the diagnosis. Because of this high incidence in our series and the potentially devastating effects of carotid injury, we strongly recommend that all patients suffering optic canal fractures undergo carotid arteriography.

Logical classification of traumatically blind patients into subgroups that may or may not benefit from decompressive surgery will require further study of a larger series of patients. In this setting, preoperative CT scanning can confirm trauma to the optic nerves by identifying fractures and their relation to the nerve. The method of choice for surgical decompression, whether transethmoidal or intracranial, also may be influenced by the identified fracture site. In view of the ability of CT to provide rapid and accurate assessment of this patient population, prospective studies of prognostic features and therapeutic approaches based on initial clinical and CT findings should be encouraged.

REFERENCES

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