Recognition of transorbital intracranial injury

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Abstract: Eyelid or periocular wounds may be the only initial sign of occult, penetrating intracranial trauma. As in this case, the failure to recognize the injury may contribute to serious and potentially life-threatening complications. The discussion emphasizes that a high degree of suspicion and knowledge of patterns of occult penetrating orbito-cranial injury may help direct appropriate radiological imaging and lead to earlier, accurate diagnosis.

Keywords: Intracrancial penetration, transorbital, orbito-cranial

Introduction

Eyelid or periocular wounds may be only initial sign of occult deep intracranial penetrations. In these cases, the initial absence of ocular or neurologic signs contributes to a low suspicion for intracranial extension, and injuries are commonly repaired without further investigation (Guthkelch 1960; Bard and Jarrett 1964; Duffy and Bhandari 1969). Delay in the diagnosis of intracranial injury may lead to serious and potentially life-threatening complications (Bard and Jarrett 1964).

We recently reviewed a series of published cases of occult transorbital penetrating injuries (Turbin et al 2006). Our analysis of the anatomic patterns of injury characterized patterns of intracranial extension from superficial orbital wounds. Improved awareness of orbital bone and fissure anatomy, as well as common injury patterns, should increase first-line evaluator recognition of occult transorbital penetrating intracranial injury.

Case report

A 50-year-old male was beaten and found unconscious, sustaining multiple minute puncture wounds to the face and periorbital area. Although the attack was unwitnessed, a police report concluded that he sustained multiple facial and periorbital stabs from the sharp wire ends of a bundled metal tomato cage. He was first evaluated by the author (RET) in an outpatient setting six months after the attack. The patient had limited recall of the attack, but remembered falling and that he was neither able to speak nor move before becoming unconscious. Further details of the event are unavailable and were not known to the physicians initially evaluating him. A review of records revealed that he was intubated in the field, hospitalized, ultimately underwent a tracheotomy, and survived a prolonged ventilator-dependant comatose state. The injury left him wheelchair bound, ataxic, and hemiparetic with radiographic evidence of resolving subarachnoid and subdural hemorrhage attributed to blunt head trauma. After his recovery and discharge from the initial hospitalization, he subsequently developed aseptic (or occult septic) cerebrospinal fluid pleocytosis, basilar meningitis, and further neurologic decline requiring re-hospitalization at a major university medical center. Subsequent computed tomographic (CT) scan was reported to show residual

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subarachnoid and subdural hemorrhage, with enhancement of the basilar meninges. The patient was diagnosed with secondary complications of a "closed-head injury" and penetrating orbito-cranial injury was not considered during either of the initial hospitalizations.

After the second recovery period, the patient presented for outpatient evaluation of refractory diplopia. He complained of double vision and vertigo, left-sided weakness, ataxia, and remained wheel-chair bound. His visual acuity was 20/30 OD and 20/25 OS. Formal color vision testing was normal. His pupils were equal, round, and reactive to light without afferent pupillary defect. He had 3 mm of right upper eyelid ptosis and a puncture scar along the right medial canthus. Other stab punctures were healed and no longer visible. There was 3 mm of enophthalmos on the right. Ocular rotations were abnormal. He had a moderate right gaze paresis with underaction of abduction on the right, and abnormal adduction on the left. He had a right hypertropia that was incomitant in lateral gaze. He exhibited abducting dysconjugate and torsional nystagmus of the right eye. Findings on fundoscopic evaluation were normal. Automated visual field testing was normal.

The patient underwent subsequent evaluation with CT and magnetic resonance imaging (MRI) of the brain and orbit. Review of both images suggested an unusual obliquely oriented linear tract of signal traversing the brain stem and left cerebellar hemisphere. The linear tract projected directly back to areas of signal abnormality in the lateral aspect of the right orbit consistent with small metallic fragments, recognized only in retrospective review of magnified images of the

orbit. These subtle findings were consistent with a penetrating tract through the lateral orbit and superior orbital fissure into the brainstem, pons, and cerebellum (Figure 1A, 1B).

At the last follow-up visit, the patient ambulated with a walker and the strength of the left upper and lower extremity was improving. The slit lamp examination was unremarkable, and no visible eyelid scars were detected. The motility examination was notable for mild gaze paresis with nystagmus in left, down, and up gaze.

Discussion

In this case, radiologic evidence of a thin penetrating tract through the orbit, midbrain, and cerebellum suggest that the sharp end of the tomato cage pierced the orbit, leaving particulate matter (paint fragments?) visualized on the CT and MRI. Although it is also possible that the victim was stabbed with another unidentified object; that possibility was not supported by the forensic police report. The unrecognized orbito-cranial penetration tracked along the lateral orbital wall, and probably via the greater wing of the sphenoid or the orbital fissures, passed through the temporal lobe and pierced the tegmentum, medulla, pons, and cerebellum. The brainstem injury rendered the patient hemiplegic, ataxic, diplopic, and initially ventilator-dependant. Based on the location of the radiographic location of the tract, it is likely that the patient sustained myopathic damage to the right lateral rectus also resulting in mild enophthalmos. In addition, the patient sustained a central gaze palsy with nystagmus from the damage to the central pathways governing horizontal gaze. The
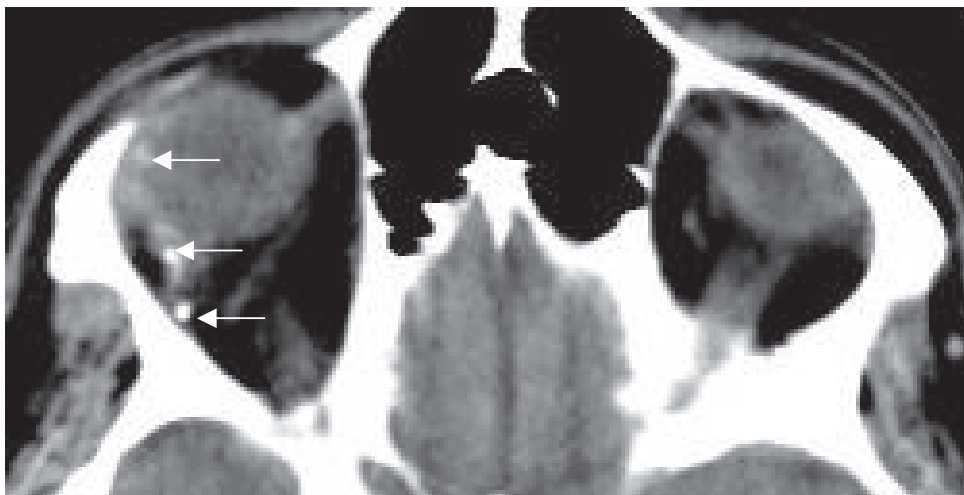

Figure 1A Axial computed tomography (1A) of the orbit performed more than eight months after the initial injury demonstrates high attenuation particulate matter in a tract along the right lateral orbit (arrows).
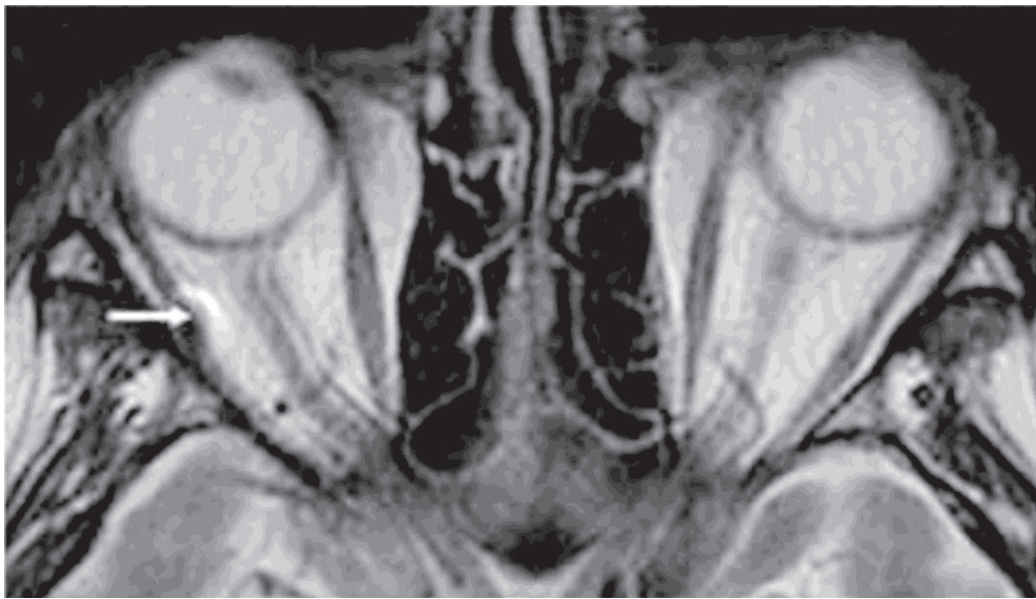
Figure 1B Axial T2 magnetic resonance imaging image illustrates area of signal abnormality in the lateral aspect of the right orbit. Foreign body induced signal abnormality (susceptibility artifact) represents metal particulate matter.
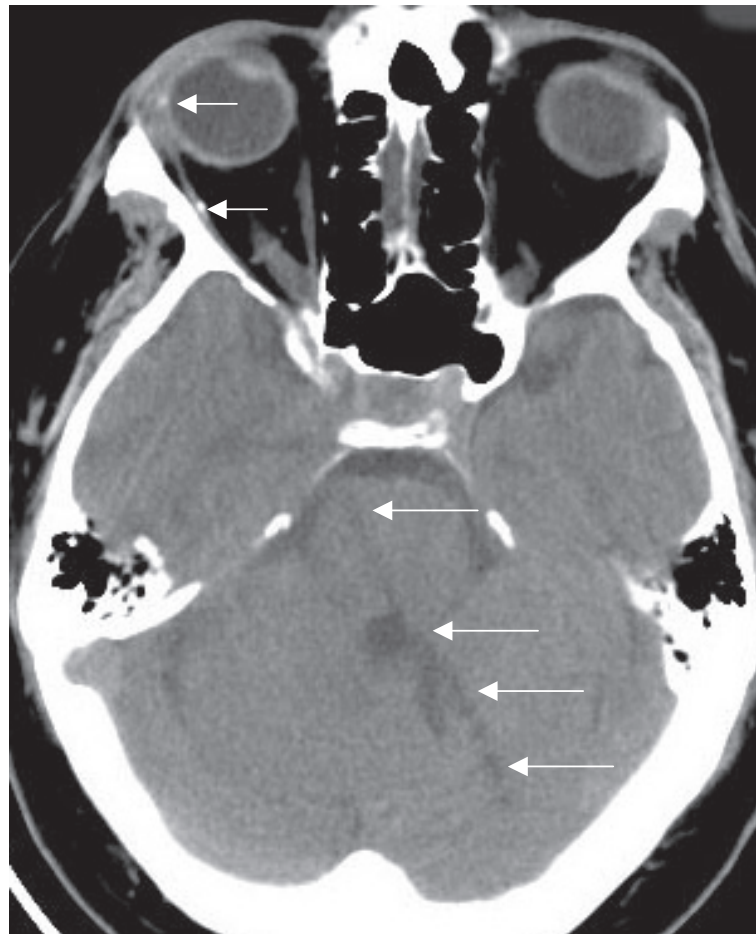

Figure 1C Axial computed tomography demonstrates that orbital particulate matter (small arrows) is aligned with a tract perforating the midbrain and cerebellum (Larger arrows).
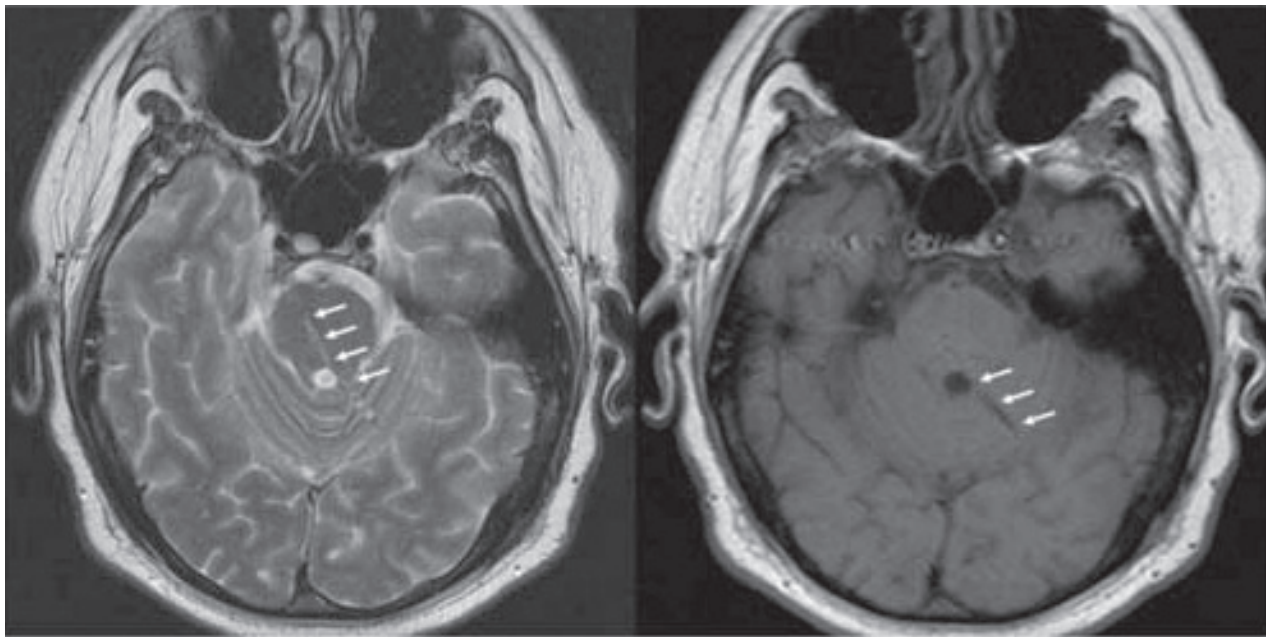
Figure 1D Axial T2 (left) and T1 (right) magnetic resonance imaging image illustrates linear tract traversing the brain stem and left cerebellar hemisphere.
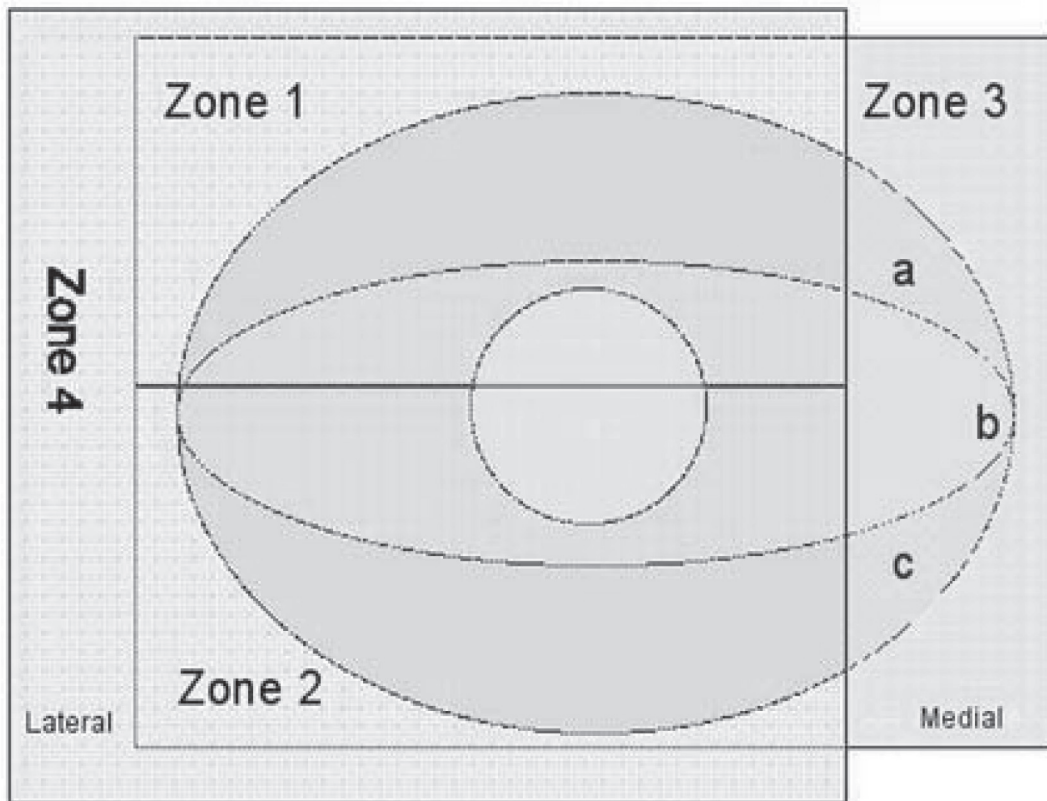

Figure 2 Schematic diagram describing cutaneous zones of entry.

Figure 2 *continued.*

1	Nonmedial UL	Orbital roof > Sphenoid wing	Frontal lobe > Temporal lobe
2	Nonmedial LL	Orbital roof > SOF > IOF/Orbital floor	Frontal lobe > Cavernous sinus/Temporal lobe > Pterygopalatine/Infratemporal fossa
3	All medial lesions	SOF > OC > Sphenoid wing > Orbital roof	Cavernous sinus > Temporal lobe > Brainstem > Frontal lobe > Basal cistern
3a	Medial UL	SO > Orbital roof > Sphenoid wing > OC	Temporal lobe > Frontal lobe > Cavernous sinus/Basal cistern
3b	MC	SOF > Sphenoid wing	Cavernous sinus > Temporal lobe > Brainstem/Basal cistern
3c	Medial LL	SOF > OC/Sphenoid wing	Brainstem > Temporal lobe/Cavernous sinus
4	All nonmedial lesions	Orbital roof > SOF > Sphenoid wing/Orbital floor/IOF	Frontal lobe > Temporal lobe> Infratemporal/Pterygopalatine fossa/Cavernous sinus

Abbreviations: IOF, inferior orbital fissure; LL, lower lid; MC, medial canthus; OC, optic canal; SOF, superior orbital fissure; UL, upper lid.

patient then developed a late sterile or a partially suppressed infectious basilar meningitis which lead to further neurologic decompensation.

Our previous analysis of the anatomic patterns of injury segregated the periocular surface into four zones (Figure 2) (Turbin et al 2006). Zone 1 injury penetrates central, lateral, and upper eyelid, or superior conjunctiva. Zone 2 injuries penetrate central, lateral, and lower eyelid or inferior conjunctiva. Zone 3 injuries penetrate medially, and are further divided into upper, middle, and lower entry points. Zone 4 injuries represent the combination of all nonmedial zones. Typical patterns of injury are presented in Figure 2. This case represents a Zone 4 injury, and could not be further subclassified because of the otherwise unknown origin of the entry point. Penetrating transorbital injury deep enough to injure intra-axial structures is rare, and is not typically occult. The small diameter of the proposed offending object is the likely reason the patient survived the injury.

In less dramatic cases, the emergency room physician is usually the first or only provider to examine and potentially identify cases of occult orbito-cranial injury. A high degree of suspicion and appropriate knowledge of recurring patterns of penetrating orbito-cranial injury is necessary to make the correct diagnosis. Knowledge of the superficial wound location and corresponding patterns of orbito-cranial damage should help guide the selection of appropriate radiographic studies and neuro-radiologic consultation. In fact, penetrating tracts may only be subsequently recognized in retrospective review of initial radiographic studies or orbital studies directed by a strong suspicion of this form of injury. We hope early recognition and appropriate management of such patients will prevent the sequelae of delayed diagnosis.

Disclosure

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