Orbital Soft-Tissue Trauma

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KEYWORDS

• Orbits • Trauma • Computed tomography • Magnetic resonance imaging • Imaging • Soft tissue

KEY POINTS

- Significant morbidity is associated with orbital trauma, including permanent visual loss.
- In the clinical assessment of orbital trauma, visual acuity and extraocular muscle motility are critical for rapid evaluation of injury severity.
- Assessment of these parameters may be limited by edema and concomitant injuries.
- Imaging may further delineate the trauma pattern and extent of injury, and is performed early and often in patients with significant orbital trauma.

IMAGING TECHNIQUES

In patients with orbital trauma, physical examination and history should focus on rapid evaluation of injury severity. Mechanism and timing of injury may provide valuable clinical information in the workup of such patients. Visual acuity testing, intraocular pressure, slit-lamp examination with fluorescein and fundoscopy are helpful for assessment, but may be limited if severe trauma is present or globe rupture is suspected. Visual acuity and extraocular motility are the 2 most important functions to be evaluated emergently.[1] Evaluation of these clinical parameters may be limited by edema and concomitant injuries. Imaging may further delineate the trauma pattern and extent of injury, and is performed early and often in patients with significant orbital trauma.

ORBITAL ANATOMY

Familiarity with normal orbital anatomy is important for the interpretation of trauma studies (**Fig. 1**). The globe is a spherical 2- to 3-cm structure with 3 concentric layers: the outer sclera, the vascular choroid, and inner retina, continuous with the optic nerve. The sclera blends into the cornea and is covered by the translucent conjunctiva ventrally. The uvea describes the middle layer and consists of the choroid, ciliary body, and iris. The globe can be separated into the anterior and posterior segments by the lens. The anterior segment is further subdivided into the anterior and posterior chamber by the iris; aqueous humor is produced by the ciliary body and flows to the anterior chamber. The lens is suspended by zonule fibers, and muscular contractions of the ciliary body result in lens deformity. The posterior segment contains gelatinous vitreous humor.

CHOICE OF IMAGING

Radiology plays a critical role in the diagnosis and treatment planning of traumatic orbital injuries. Imaging may be indicated if the posterior chamber is not visualized on ophthalmologic examination, or if there is clinical concern for orbital fracture, intraocular foreign body, or occult globe rupture.[2]

Disclosures: None.

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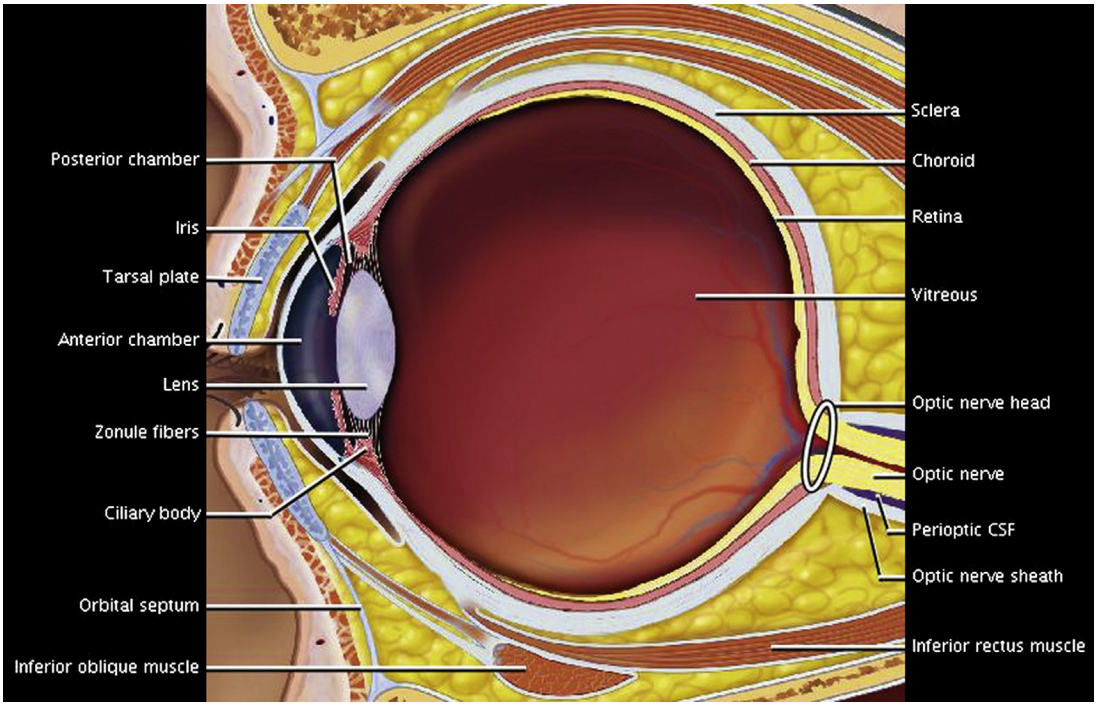

Fig. 1. Sagittal view of the orbit, illustrating the normal anterior and posterior segment structures separated by the lens. The 3 layers of the orbit are illustrated: outer sclera, vascular choroid, and inner retina. CSF, cerebrospinal fluid. (*Courtesy of* Amirsys Inc, Salt Lake City (UT); with permission.)

Computed tomography (CT) is typically the first-line modality, given its rapid acquisition and availability; however, other modalities may have distinct advantages.

Radiography

The role of conventional radiographs has diminished significantly, given the current availability of CT. Standard Caldwell (orbitofrontal view; beam tilted 15–20° caudal) and Waters (occipitomental view; chin raised) views have sensitivities ranging from 64% to 78% for detection of orbital fractures, with false-negative findings in 9% to 29%.[3] Given the moderate sensitivity and false negatives, plain films have limited clinical utility.

Computed Tomography

CT has become the first-line imaging study in patients with ocular trauma.[4] Rapid acquisition and thin-section multiplanar reformations of CT allow for accurate assessment of orbital fracture and associated injuries.[1] CT demonstrates a high sensitivity for fracture detection that is optimized by thin-section acquisition, multiplanar reformations, and 3-dimensional modeling.[5] Furthermore, for polytrauma cranial evaluation can be performed concurrently with CT imaging.

Although CT is very sensitive for detection of orbital fracture, its accuracy in soft-tissue injuries may be more limited. In a study from 2000, CT demonstrated 73% sensitivity and 95% specificity for the detection of open-globe injury.[6] Magnetic resonance (MR) imaging holds significant advantages for the assessment of retinal and choroidal detachments and nonradiopaque foreign bodies.

The lens is a radiosensitive organ, and efforts should be made to limit the radiation delivered. Modern multidetector helical CT scanning techniques demonstrate significant benefit in dose reduction over older single-detector setups. There is evidence that limiting the tube current to 100 mA instead of the conventional 300 mA setting may decrease the effective dose by 70% without compromising detection of traumatic injury.[7]

Ultrasonography

Ultrasonography can be performed rapidly at the bedside, and appears to provide accurate detection of retrobulbar hemorrhage, foreign body, lens dislocation, and retinal detachment.[8] However, it is contraindicated in the setting of globe rupture because of the pressure applied during sonographic assessment, possibly exacerbating an unstable globe.[8,9] Although ultrasonography is

limited in the assessment of complex facial fractures, it appears to provide accuracy comparable with that of CT in the detection of fractures of the infraorbital rim and orbital floor.[10]

MR Imaging

In the acute presentation of ocular trauma, MR imaging has limited utility because of its longer acquisition time and decreased sensitivity for fracture in comparison with CT. Furthermore, MR imaging is contraindicated if a metallic ocular foreign body is suspected.[9] MR imaging holds significant advantages in the detection of organic foreign bodies (eg, wood).[2]

ANTERIOR CHAMBER INJURIES

The anterior chamber is located between the cornea and iris and contains aqueous fluid. Clinically and by imaging, the anterior chamber should be assessed for the presence of abnormal material (eg, blood, foreign body) and abnormal depth. Deepening of the anterior chamber may result from lens dislocation, scleral rupture, or iridodialysis (rupture of the iris–ciliary body attachments). The anterior chamber may be abnormally shallow as a result of lens dislocation, corneal injury, choroidal hemorrhage, or acute angle closure.[2]

Anterior chamber depth (ACD) on CT appears to correspond with open-globe injury. An asymmetric difference in ACD of 0.4 mm or greater has 73% sensitivity and 100% specificity for detection of open-globe injury.[11] Corneal laceration may result in decreased ACD (**Fig. 2**) and may be accompanied by iris prolapse. Anterior lens dislocation may mimic traumatic corneal laceration, and the lens should be carefully examined.[12]

Traumatic Hyphema

A traumatic hyphema is caused by bleeding in the anterior chamber secondary to disruption of blood vessels in the iris or ciliary body.[9] A blood-aqueous level may be apparent on inspection, and increased intraocular pressure may result. Rebleeding may occur in 20% of patients, typically 2 to 5 days following injury when the initial clot retracts. Clinically it is important to assess for a bleeding diathesis, such as sickle cell disease, which can significantly increase the severity of the hyphema and the likelihood and rate of rebleed.[8] Hyphema may be classified in 4 grades, the most significant being an "8-ball hyphema" resulting from an anterior chamber completely filled with hemorrhage. More than half of traumatic hyphemas are the result of sports-related injuries.[13] Imaging is not required for the diagnosis of traumatic hyphema but is frequently performed to assess for more significant injury. The hyphema is manifested on orbital CT by increased attenuation in the anterior chamber. Disruption of these same iris and ciliary vessels may result in bleeding into the posterior chamber, a vitreous hemorrhage (see later discussion).

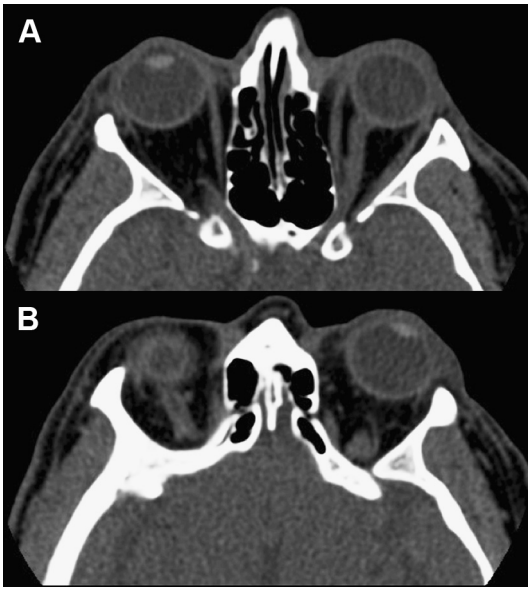

Fig. 2. (*A, B*) Axial noncontrast computed tomography (CT). Corneal laceration resulting in decreased anterior chamber depth in the left eye (*B*). Note that the normal density of the lens is preserved.

Subconjunctival Hemorrhage

Subconjunctival hemorrhage may occur from minor trauma such as sneezing, coughing, or Valsalva, and results from tearing of small subconjunctival blood vessels. The dramatic appearance may bring patients to medical attention when a painless, red conjunctival lesion is evident. The area should not be raised, and visual acuity should be preserved. Cross-sectional imaging is not necessary, and the hemorrhage is self-limited.[8]

INJURY TO IRIS AND CILIARY BODIES

Injuries to the iris and ciliary bodies are typically assessed on clinical examination. Thorough evaluation of the iris and ciliary body is performed with a mirror/prism (gonioscope) in conjunction with a slit lamp to evaluate the iridocorneal angle.[14] Cross-sectional imaging may be normal, unless associated vascular injury is present with anterior and/or posterior chamber hemorrhage.

Traumatic iridocyclitis results from blunt trauma to the iris and ciliary body that may cause spasm

and blurry vision. This condition is typically self-limited; short-term cycloplegic (dilating and paralyzing) therapy may be given. Traumatic mydriasis and miosis result from similar traumatic injury to the iris sphincter. Although this condition is self-limited, imaging may be performed to exclude cranial nerve injury as the underlying cause.[8] Traumatic iridodialysis is the result of a tear in the peripheral iris resulting in separation of the iris and ciliary body.

Glaucoma can result from traumatic obstruction of aqueous outflow from blood (hyphema), disruption of the trabecular meshwork, or lens dislocation. Delayed-onset glaucoma can occur from angle recession, lens dislocation, or various hemolytic manifestations. Delayed glaucoma is also seen following open-globe injury, owing to formation of synechiae.[14]

LENS INJURIES

Subluxation and Dislocation

The zonule fibers responsible for holding the lens in place may be disrupted by blunt trauma, resulting in lens detachment. Patients will clinically display blurred vision, monocular diplopia, or distortion if partial lens dislocation is present. Most commonly the lens will dislocate posteriorly because the iris prevents anterior translation. However, anterior lens dislocations can occur, and may mimic decreased ACD caused by corneal laceration. Anterior lens dislocation may also result in acute angle closure glaucoma by mechanical obstruction of aqueous outflow, a condition requiring emergent ophthalmologic attention.[15]

With complete dislocation, the lens is typically displaced posteriorly and lies dependently within the vitreous humor (**Fig. 3**). When the zonule fibers partially tear, there is asymmetric displacement of the lens away from the ruptured ciliary attachments and posterior displacement into the vitreous humor (**Fig. 4**).[9]

Trauma is the most common cause of lens dislocation, but bilateral nontraumatic subluxations may be seen with Ehlers-Danlos syndrome, Weill-Marchesani syndrome, aniridia, homocystinuria, and Marfan syndrome.[16]

Traumatic Cataract

The lens capsule normally maintains a dehydrated environment. When the capsule is disrupted, the lens may absorb fluid and swell. This condition manifests clinically as a cloudy edematous lens and is demonstrated on CT by unilateral lens hypoattenuation, a finding specific for traumatic cataract (**Fig. 5**). A decrease in attenuation of 30 HU may be seen in the abnormal lens. Treatment is surgical lens replacement.[8] The pathophysiology of traumatic cataract is similar to that of osmotic cataract seen with hyperglycemia, in which elevated glucose in the aqueous humor creates an osmotic gradient that draws fluid into the lens; however, this finding is bilateral in diabetic hyperglycemia.[17]

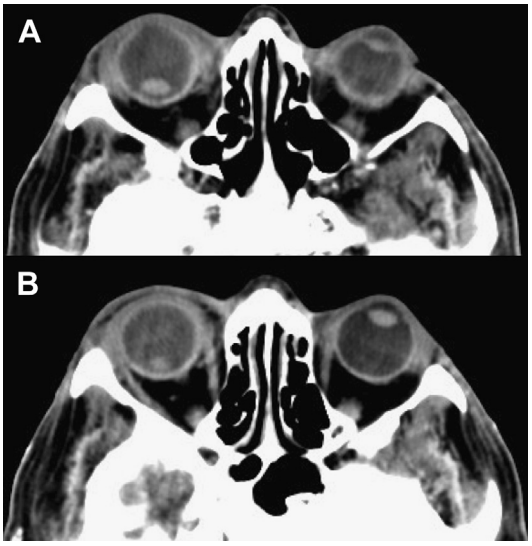

Fig. 3. (*A*, *B*) Axial noncontrast CT. Complete dislocation of the right lens following trauma. The dislocated right lens lies dependently within the vitreous humor.

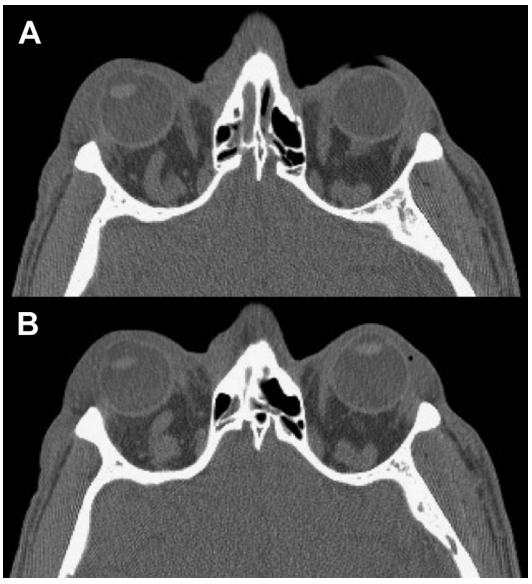

Fig. 4. (*A*, *B*) Axial noncontrast CT. Partial dislocation of the right lens is evident, with rupture of the medial zonule fibers and lateral and slight posterior displacement of the lens. The left lens appears normal.

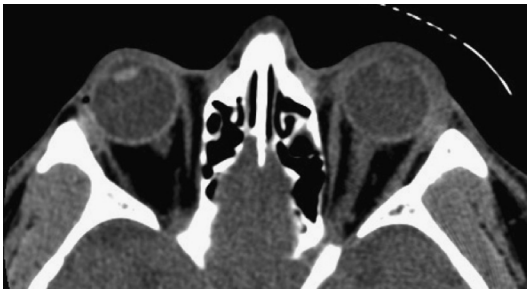

Fig. 5. Axial noncontrast CT. Corneal laceration and decreased left anterior chamber depth with traumatic cataract. When the lens capsule is disrupted, a traumatic cataract results from a swollen edematous lens with low density on CT.

GLOBE INJURIES

Globe Rupture

Open-globe injury, or globe rupture, is a surgical emergency because of the risk of endophthalmitis and the associated high rate of monocular blindness. The standard practice of ophthalmologists worldwide is to undertake a primary surgical repair to restore the structural integrity of the globe. This action is best undertaken at the earliest opportunity, regardless of the extent of the injury and the presenting visual acuity.[18] Although diagnosis of an open-globe injury is obvious when intraocular contents are visualized on ophthalmologic examination, it can be challenging when a patient is unable or unwilling to cooperate, or if severe facial injuries make examination unsafe.[6]

CT is the imaging test of choice for the diagnosis of an open-globe injury and assessment for associated injuries. Findings include a change in globe contour with an obvious loss of volume ("flat-tire" sign), scleral discontinuity, intraocular air, and intraocular foreign body (**Figs. 6** and **7**).[9] Absent or dislocated lens, vitreous hemorrhage, and retinal detachment are additional predictors of open-globe injury.[19] In a review of 46 patients who underwent surgical exploration, moderate to severe change in globe contour (flattening or concavity of the sclera in at least 1 quadrant), obvious volume loss, total vitreous hemorrhage, and absence of lens were seen only in eyes with globe rupture.[19]

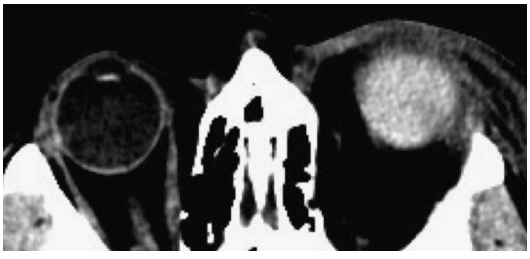

Fig. 6. Axial noncontrast CT of a patient with left facial trauma and left globe volume loss. Total vitreous hemorrhage is present and the lens is absent, both specific for open-globe injury.

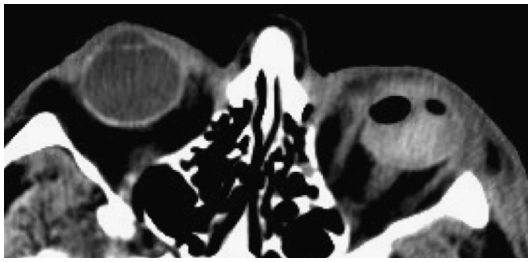

Fig. 7. Axial noncontrast CT of a patient with left facial trauma shows an obvious loss of volume of the left globe, contour change, intraocular gas, and hemorrhage.

CT has historically been an imperfect diagnostic modality for ruptured globe. A retrospective review of 200 patients from 1989 to 1993 found that CT had sensitivity of 75% and specificity of 93% for the diagnosis of open-globe injury in the absence of clinical information.[6] In the setting of massive subconjunctival and/or anterior chamber hemorrhage precluding adequate clinical examination in 59 patients, sensitivity was found to be 70%.[20] Sensitivity may be especially limited in the setting of equivocal clinical examination. A review of 46 patients found that when analysis was limited to patients with clinically occult globe rupture, sensitivity decreased from 71% to a range of 56% to 68% between observers, with a negative predictive value of 42% to 50%.[19]

Recent literature, however, suggests better performance for CT when certain findings are considered. Kim and colleagues[11] found 92% sensitivity, 85% specificity, and 89% accuracy for globe rupture in 56 patients when 1 or more of the following are present: change in ACD; change in globe contour; obvious loss of globe volume; dislocated/deformed lens; intraocular foreign body/air; or intraocular hemorrhage. The key finding among these factors was change in ACD. An earlier study reported increased ACD in globe rupture, thought to result from posterior scleral injury decompressing the vitreous, allowing for retropulsion of the lens and deepening of the anterior chamber.[21] However, Kim and colleagues[11] found that 13 of 15 patients (86%) with globe rupture had decreased ACD, a result of either corneal perforation with collapse of the anterior chamber or increased intraocular pressure from hemorrhage (**Fig. 8**). Regardless of whether ACD increased or decreased, any difference between globes

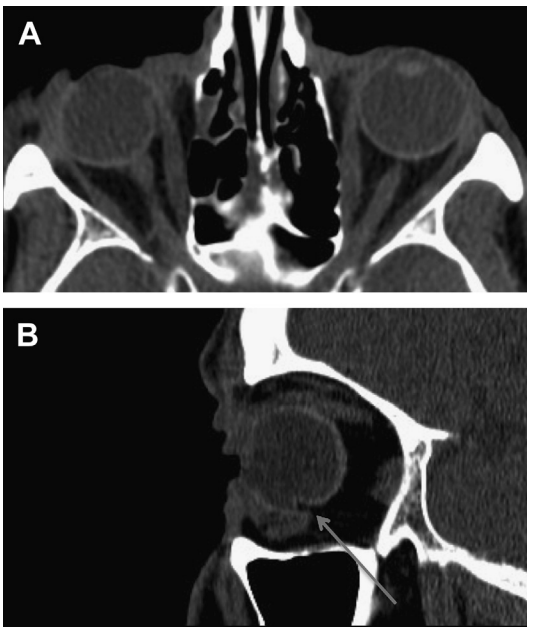

Fig. 8. (*A*) Axial noncontrast CT of a patient with right facial trauma shows asymmetric decreased right anterior chamber depth, which can result from either corneal perforation or increased intraocular pressure. (*B*) Medial scleral discontinuity of the right globe (*arrow*) is compatible with globe rupture, also shown at the inferior margin of the right globe on sagittal CT reconstruction.

0.4 mm or larger had 73% sensitivity and 100% specificity for globe rupture.

Potential pitfalls in the diagnosis of open-globe injury include nontraumatic causes of globe-contour deformity, mimics of intraocular air, and mimics of traumatic foreign body. Nontraumatic causes of globe-contour deformity include orbital mass or hematoma, buphthalmos, staphyloma, and coloboma (**Fig. 9**).[9,16] Mimics of intraocular air include gas injection into the vitreous to tamponade the retina in the treatment of retinal detachment (**Fig. 10**).[16] Mimics of traumatic foreign body include scleral banding (**Fig. 11**), silicone oil (**Fig. 12**), eyelid implants, and glaucoma drainage devices.[22] Surgical history and knowledge of characteristic findings can help suggest the correct diagnosis.

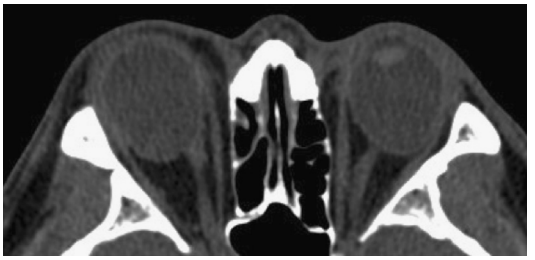

Fig. 9. Axial noncontrast CT of a patient with abnormal contour of the globe secondary to staphylomas. The bilateral nature of this finding helps to exclude open-globe injury. The right lens was normal but out of the imaging plane.

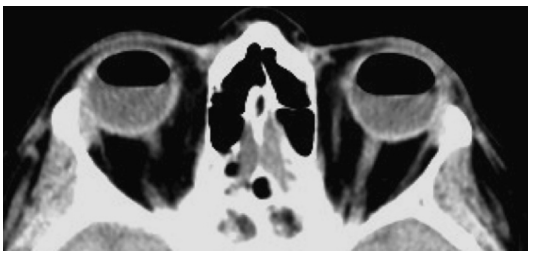

Fig. 10. Axial noncontrast CT of a patient with intraocular gas for treatment of bilateral retinal detachment. The pressure created by injected gas creates a tamponade, forcing the retina into choroid apposition.

Globe Luxation

Globe luxation, or anterior dislocation of the globe beyond retracted eyelids, results from an extreme proptosis that allows the lids to slip behind the globe equator. It places traction on the optic nerve and retinal vasculature, making early reduction important for maintaining visual acuity.[23] It can occur in the setting of trauma, Graves ophthalmopathy, or orbital mass, among other causes. Reported cases in the literature do not include radiologic imaging, but clinical appearance is characteristic.

POSTERIOR SEGMENT INJURIES

Vitreous Hemorrhage

In the setting of trauma, orbital hemorrhage can accumulate within the posterior segment in multiple anatomic spaces. Blood accumulating in the vitreous is simply called vitreous hemorrhage

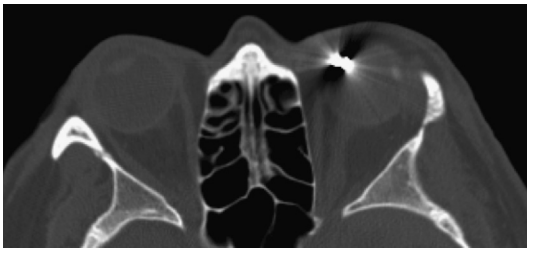

Fig. 11. Axial noncontrast CT of a patient with left medial scleral band used to treat retinal detachment mimicking foreign body. In the setting of trauma, eliciting a history of scleral banding may be crucial to differentiate from traumatic foreign body. Knowledge of the characteristic peripheral location can also aid in the correct diagnosis.

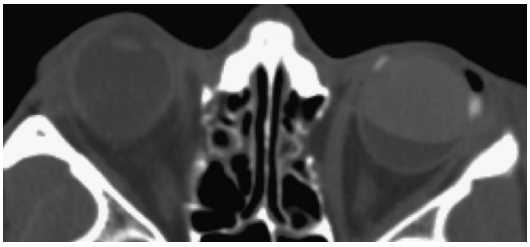

Fig. 12. Axial noncontrast CT. Silicone oil injected into the left vitreous, mimicking hemorrhage.

(Fig. 13). Hemorrhage can also collect in the space between the vitreous and retina. In addition, hemorrhage and fluid may seep into the potential spaces between the 3 layers of the globe, particularly when there has been laceration to 1 or more layers.

CHORIORETINAL INJURY

The retina is the innermost layer of the globe, and is securely attached to the choroid at its anterior margin, the ora serrata, and its posterior margin at the optic disc. The remainder is relatively loosely connected to the choroid. In the setting of trauma, a retinal tear can allow vitreous fluid and hemorrhage into the subretinal space. If the retina remains attached at the ora serrata and optic disc, hemorrhage lifting the retina off the choroid will have a characteristic V-shaped appearance on CT (Fig. 14).[9] If these attachments are also severed, the retina may become free-floating. The presence of subretinal hemorrhage in a child should raise suspicion for child abuse (Fig. 15). Nontraumatic causes of retinal detachment include inflammation and neoplasm.

Similarly, choroidal detachment occurs when hemorrhage or fluid collects in the suprachoroidal space between the choroid and sclera. The normal choroid extends from the ciliary body anteriorly to the optic nerve head posteriorly.

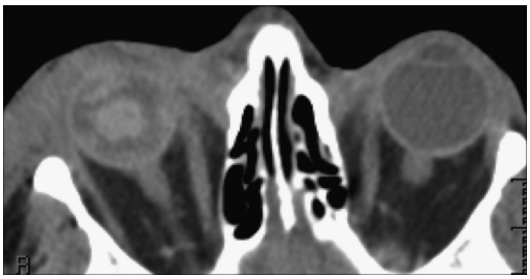

Fig. 13. Axial noncontrast CT of a patient with right-sided facial trauma shows right globe rupture evidenced by decreased globe volume and medial scleral discontinuity. There is prominent right vitreous hemorrhage.

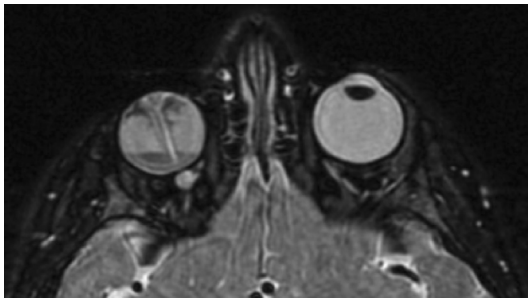

Fig. 14. Axial fat-suppressed T2-weighted image showing complete right retinal detachment with layering subretinal hemorrhage. This pediatric patient had primary hyperplastic vitreous with secondary retinal detachment; however the characteristic V-shaped detachment is illustrated.

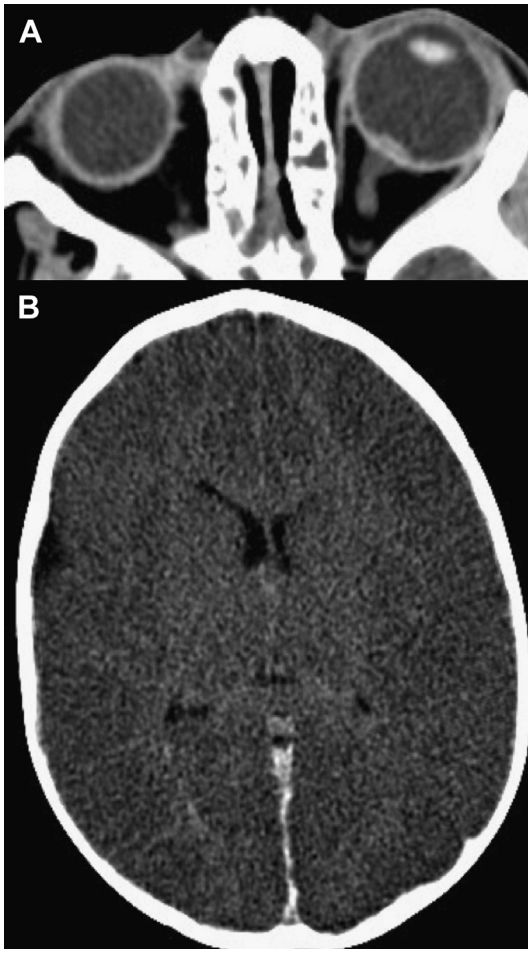

Fig. 15. (*A*) Axial noncontrast CT of a pediatric patient with left-sided subretinal hemorrhage. (*B*) Head CT of the same patient shows hemorrhage in the posterior horn of the right lateral ventricle in addition to posterior parafalcine subdural hemorrhage. The patient was later confirmed to be a victim of nonaccidental trauma.

Choroidal detachments occur in the setting of ocular hypotony, or decreased intraocular pressure. Trauma is one cause of ocular hypotony, which results in decreased pressure within the suprachoroidal space, allowing transudate to enter. If bridging arteries and veins between choroid and sclera are torn in the process, suprachoroidal hemorrhage can also occur. Suprachoroidal collections appear as biconvex, extending from the ciliary body anteriorly to the vortex veins posteriorly (Fig. 16).[9]

The morphology of hemorrhage can help distinguish the location in chorioretinal injury. Characteristically, choroidal detachment with suprachoroidal hemorrhage demonstrates a biconvex shape as opposed to a V-shaped configuration of retinal detachment and subretinal hemorrhage (Fig. 17).

Commotio Retinae

Commotio retinae, or Berlin edema, is a zonal area of retinal whitening caused by outer photoreceptor disruption and retinal pigment epithelial damage sustained by blunt trauma, often from assault, motor vehicle accident, or sports injury. It can result in loss of vision when affecting the macula.[24] Although there are no typical radiologic imaging findings, commotio retinae is often associated with preretinal, retinal, and subretinal hemorrhages and choroidal rupture, and, as such, a careful search for these findings should be made in this setting.[25]

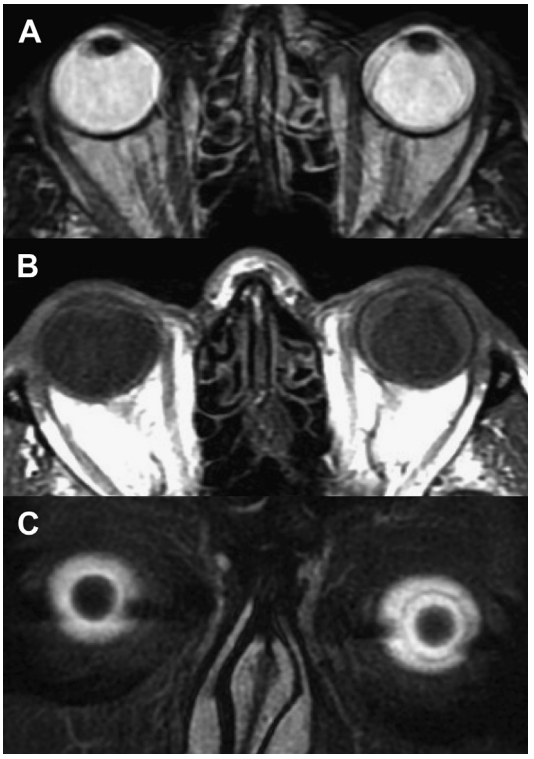

Fig. 16. Choroidal hemorrhage. Axial fat-suppressed T2-weighted (*A*), axial T1-weighted postcontrast (*B*), and coronal T2-weighted fat-suppressed (*C*) images of the orbits show biconvex collections in the left globe characteristic of suprachoroidal collections. The T2 signal is hypointense to vitreous and the T1 signal is intermediate, reflecting either hemorrhage or proteinaceous fluid.

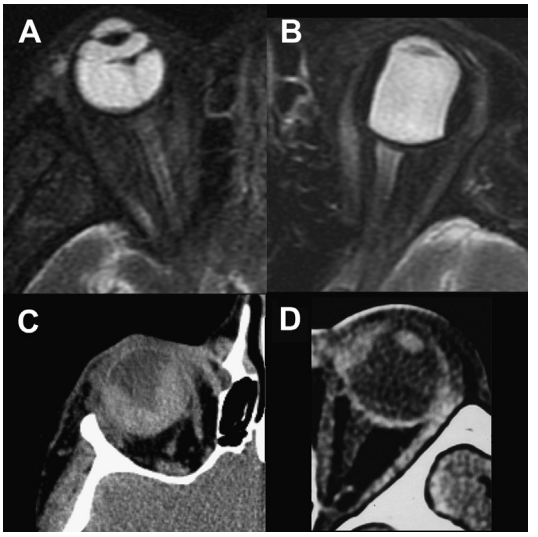

Fig. 17. Choroidal versus retinal hemorrhage. Axial fat-suppressed T2-weighted images (*A, B*) and axial noncontrast CT images (*C, D*) in 4 different patients. Characteristic V-shaped retinal detachment and hemorrhage is shown (*A, C*) in comparison with the biconvex shape of choroidal hemorrhage (*B, D*).

PENETRATING OCULAR INJURIES

Penetrating ocular injuries can be obvious when large foreign bodies remain present (Fig. 18). However, they can be missed on physical examination if the injury is sealed off or if clinical signs are subtle.[26] In these cases, careful attention should be given to the detection of an intraorbital foreign body (IOFB), which is common, occurring in 1 of every 6 cases of orbital trauma.[27] Knowledge of the precise location and number of IOFBs is also important preoperatively. Extraocular IOFBs are typically managed conservatively, whereas those that are intraocular may require surgical intervention.[28] Sequelae of undetected IOFBs include posttraumatic endophthalmitis, potentially leading to permanent loss of vision, in addition to hyphema, cataract formation, vitreous hemorrhage, and retinal tears and detachment.[28] Failure to detect metallic foreign bodies on CT is potentially devastating if the patient goes on to MR imaging.

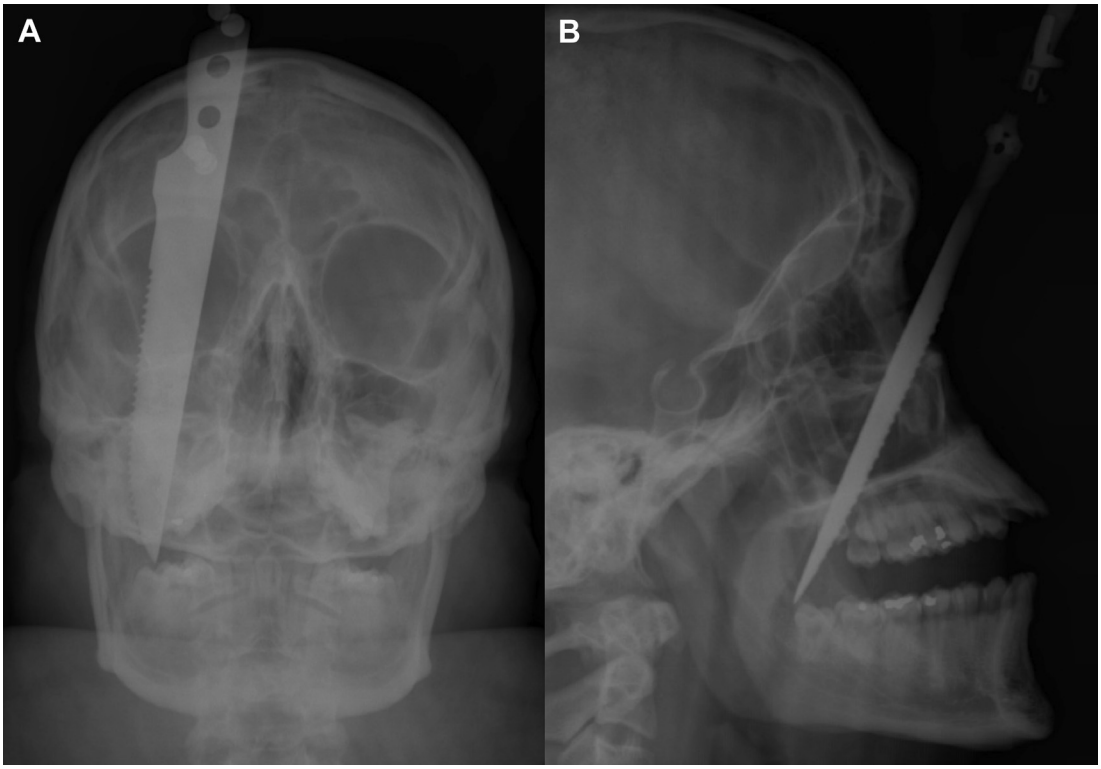

Fig. 18. (*A*) Anteroposterior and (*B*) lateral radiographs of a patient with transorbital penetrating knife injury extending through the maxillary sinus into the oral cavity.

CT is the gold standard for the detection of IOFBs,[27] with the ability to detect objects as small as 1 mm.[9] Metallic foreign bodies are well discerned on most CT window settings because of their high attenuation compared with the intraorbital soft tissues (**Fig. 19**B). MR imaging of a metallic IOFB demonstrates susceptibility artifact, which can limit evaluation of the orbit and surrounding soft tissues (see **Fig. 19**A).

Nonmetallic foreign bodies can present a diagnostic dilemma because they are less apparent on imaging. CT is more sensitive than MR imaging or ultrasonography for the detection of intraorbital glass. Sensitivity increases with increasing size (96% at 1.5 mm vs 48% at 0.5 mm), higher attenuation (range 80–550 HU), and anterior chamber location.[29] Wooden foreign bodies display a range of appearances on CT. Wood is a porous structure, and the relative amount of air and water within wood accounts for its attenuation on CT. For example, dry pine appears similarly to air on soft-tissue window settings (HU −656), whereas fresh pine, with greater water content, will appear at nearly water density on CT (HU −24).[27] A wooden foreign body can be difficult to identify when it mimics air, and should be scrutinized with wider window settings, especially in the

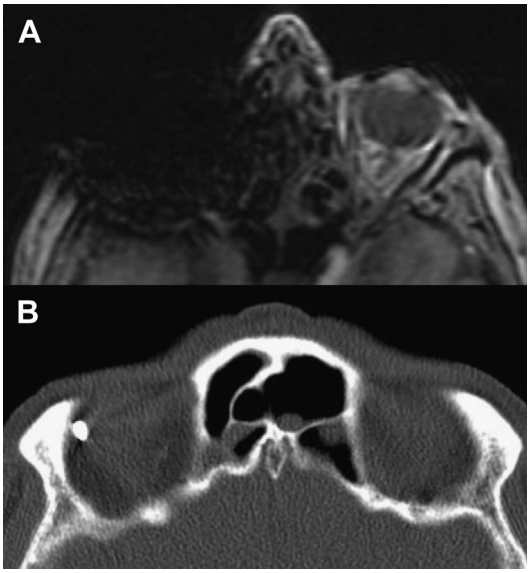

Fig. 19. Patient with right-sided orbital penetrating injury resulting in metallic foreign body. A magnetic resonance image (*A*) was first obtained without proper screening, showing significant susceptibility in the right orbit. Subsequent CT (*B*) shows the metallic foreign object in the right lateral orbit.

absence of fracture or if there are geometric margins (**Fig. 20**). The attenuation of wooden IOFBs has been shown to increase over time, a reported specific finding for wood; this likely results from body fluid displacing air within the porous foreign body.[30] Some advocate for MR imaging when CT is negative or equivocal in searching for a wood IOFB, because fat-suppressed postcontrast images can demonstrate surrounding inflammatory changes.[9]

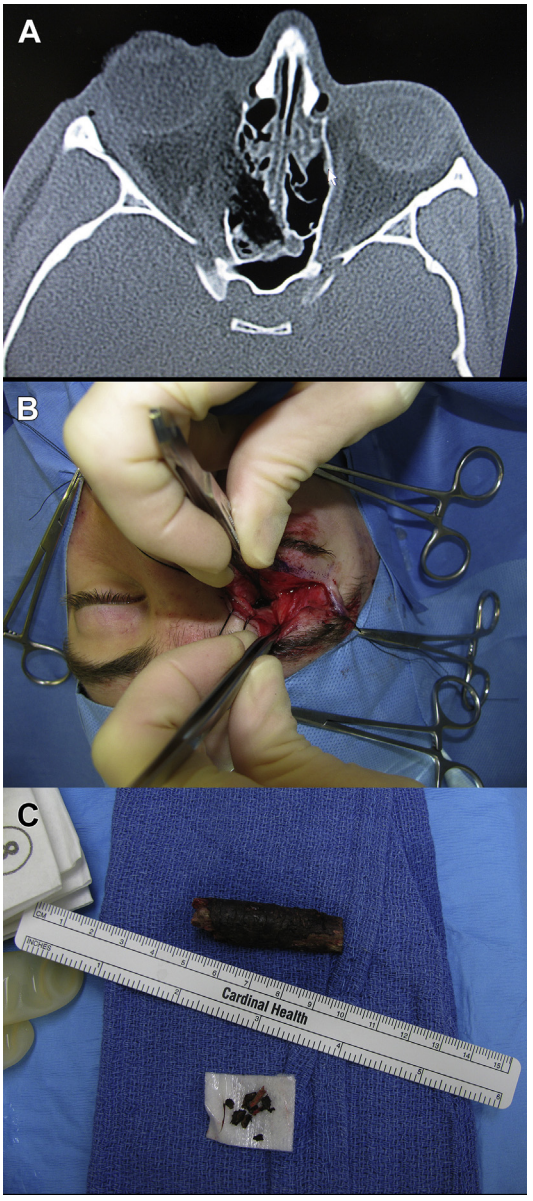

Fig. 20. Axial noncontrast CT (*A*) showing a hypodense foreign body traversing the orbit and right lamina papyracea into the ethmoid sinus. Intraoperative view (*B*) shows the deep penetrating injury and wooden foreign object that was removed (*C*).

CAROTID-CAVERNOUS FISTULAS

A carotid-cavernous fistula (CCF) forms when there is disruption of the wall of the cavernous internal carotid artery (ICA), resulting in arteriovenous shunting from the ICA into the cavernous sinus. Although CCF in most cases is not secondary to orbital trauma, it can result in multiple orbit-related findings, and therefore is an important entity to consider in posttraumatic orbital imaging. The typical mechanism for a posttraumatic CCF involves a skull-base fracture extending through the sphenoid bone and bony carotid canal, or penetrating injury to the posterior orbit or cavernous sinus.[31] When arterialized flow develops in the cavernous sinus, increased venous pressure can lead to flow reversal in the veins draining into the cavernous sinus, including the ophthalmic veins. If untreated, a progressive increase in intraocular pressure can ultimately cause retinal hypoperfusion and retinal ischemia. Ocular symptoms, including ocular chemosis, proptosis, glaucoma, and visual loss, typically do not present immediately but rather days or weeks after the initial trauma.[31] Pulsating exophthalmos and objective pulsatile tinnitus are also occasionally seen along with deficits of cranial nerves III through VI.[9]

Imaging of the head and orbits is valuable in the diagnosis of CCF. Typically CT or MR imaging demonstrates dilation of the superior ophthalmic vein, proptosis, and extraocular muscle enlargement, all findings potentially demonstrable on noncontrast studies.[9] Contrast-enhanced CT or MR imaging can provide additional diagnostic clues, including illustration of cavernous sinus distension and improved visualization of ophthalmic vein enlargement (**Fig. 21**). Definitive diagnosis requires digital subtraction angiography (DSA), which shows early aberrant filling of the cavernous sinus and dilated veins leading away from the sinus, including the superior and inferior ophthalmic veins and the superior and inferior petrosal sinuses. DSA can also provide access for endovascular therapy for CCF, with treatment options including detachable balloon embolization, transvenous embolization, or covered-stent repair of ICA laceration.[32]

OPTIC NERVE INJURY

Traumatic optic neuropathy (TON) is a rare but important potential result of head trauma, with recent estimates suggesting that optic nerve injury occurs in approximately 0.5% to 5% of all closed head injuries and up to 10% of patients with craniofacial fractures.[33] TON can be subdivided into 2 main types, direct or indirect, defined by mechanism of injury to the optic nerve.[34] Direct TON

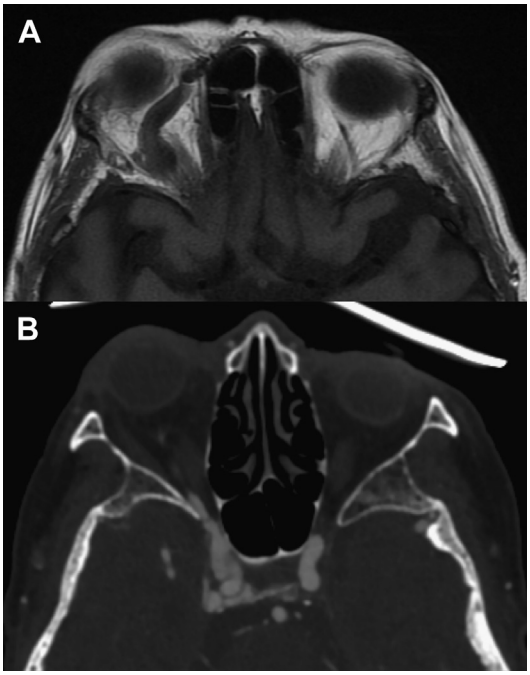

Fig. 21. (*A*) Axial T1-weighted image shows right-sided proptosis and marked dilatation of the right superior ophthalmic vein in this patient after direct facial trauma. (*B*) Axial CT angiography shows an abnormal enlargement of the right cavernous sinus with a convex lateral border. Note the abnormal early venous enhancement in the right side of the cavernous sinus, consistent with arteriovenous shunting in this patient with a direct posttraumatic carotid-cavernous fistula.

occurs when the optic nerve is damaged by a transorbital penetrating injury such as from a bullet or knife. Indirect TON includes all other nonpenetrating sequelae of trauma, including mass effect from retrobulbar hemorrhage and deceleration injuries that subject the optic nerve to violent rotatory forces. Such indirect mechanisms of injury may compress or tear the optic nerve or its associated vascular supply. TON typically produces clinical findings common to most optic neuropathies, including decreased visual acuity, color, and visual field, with visual acuity often 20/400 or less on the affected side.[33] Patients with TON often have severe injuries requiring immediate attention; the diagnosis of TON may be delayed until a detailed visual assessment can be performed.

In patients with facial trauma and subsequent decrease in visual acuity, neuroimaging is used to evaluate for potential causes of TON. In particular, noncontrast CT through the orbits is valuable in the assessment of any fracture involving the orbital apex that could compromise the optic nerve (**Fig. 22**).[33] In cases of TON related to a retrobulbar hemorrhage, noncontrast CT will allow for the characterization of the volume and extent of hemorrhage in the orbit, potentially guiding surgical decompression (**Fig. 23**). In situations where a trauma patient is clinically stable and able to tolerate MR imaging, direct visualization of the optic nerve can reveal abnormal hyperintensity in the nerve on T2-weighted images. In cases of posttraumatic compromise of the vascular supply to the optic nerve, diffusion-weighted imaging can demonstrate reduced diffusivity in the optic nerve, suggesting ischemic injury (see **Fig. 22**B, C). The ability to integrate detailed multisequence MR imaging of the orbits, however, is often unfeasible, despite its advantage in the demonstration of soft-tissue abnormality.

In patients sustaining traumatic injury to the optic nerves, the main treatment options remain close observation, high-dose corticosteroid therapy, surgical decompression, or a combination thereof.[33] However, given the lack of large observational studies or randomized controlled trials assessing patient outcomes after treatment of TON, the optimal treatment algorithm remains controversial. Future treatments currently being investigated include neuroprotective drugs and other therapies to enhance visual recovery following trauma.[33]

Diagnostic checklist orbital CT

ACD: Ensure the ACD is symmetric bilaterally. Corneal laceration, globe rupture, and lens dislocation will result in ACD asymmetry.

Lens density: A traumatic cataract will manifest with unilateral lens hypodensity.

Lens position: Careful attention should be paid to lens position. Complete dislocation is most common posteriorly into the vitreous. Partial dislocation may show subtle asymmetry away from the side of zonule fiber disruption.

Globe shape: The globe should demonstrate a smooth contour. Globe rupture may show focal discontinuity. Staphylomas have a characteristic location and morphology.

Foreign body: IOFB is an operative emergency and should be reported immediately. Metallic foreign bodies are usually conspicuous on CT and preclude MR imaging. Wood or other hypodense organic foreign bodies can be subtle.

Posterior segment density: Vitreous, retinal, and choroidal hemorrhage each have a characteristic location and configuration.

Fracture: Careful attention should be paid to the osseous structures surrounding the orbit, which are commonly fractured in facial trauma.

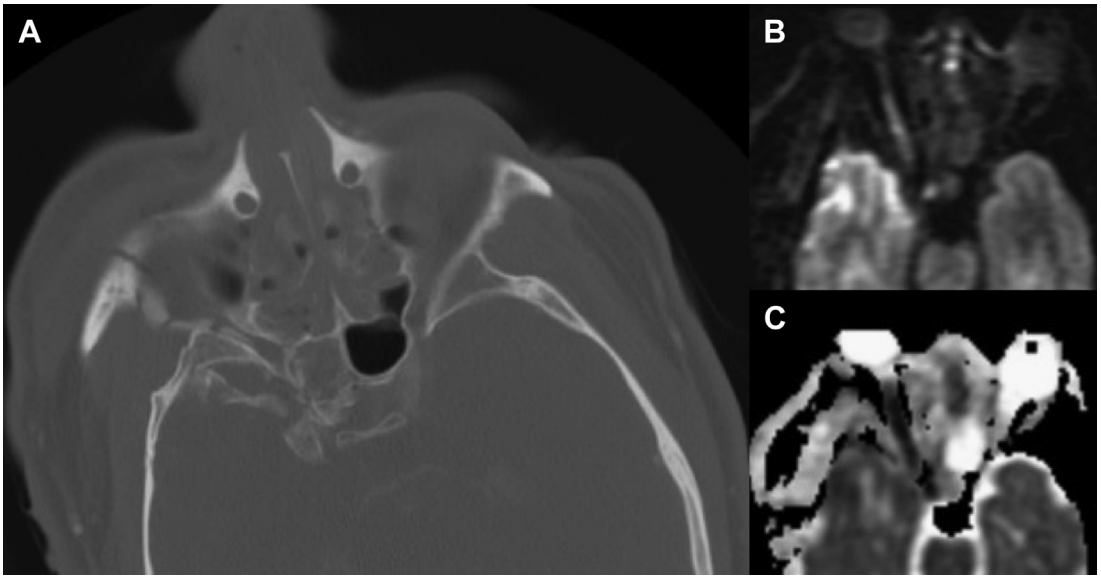

Fig. 22. A patient with right-sided visual loss after facial trauma. (*A*) Axial CT shows a complex orbital fracture with a bony fracture fragment from the right orbital apex situated within the optic canal. Axial diffusion-weighted imaging (*B*) and apparent diffusion coefficient map (*C*) show diffusion restriction with the intracanalicular and orbital segments of the right optic nerve, a finding that confirms traumatic disruption of the vascular supply to the optic nerve and resultant nerve ischemia. Right anterior temporal traumatic injury is also present.

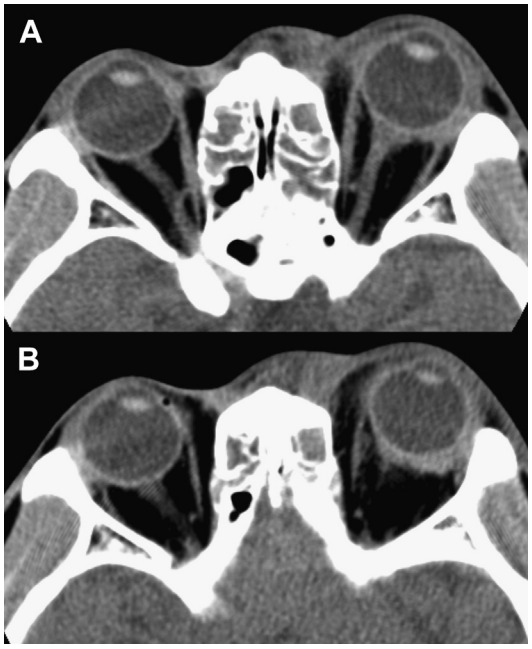

Fig. 23. (*A*, *B*) Axial noncontrast CT showing high-density left orbital retrobulbar hemorrhage.

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