Imaging of Orbital Trauma and Emergent Non-traumatic Conditions

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**INTRODUCTION**

Radiological imaging evaluation is crucial in the aid of clinical assessment in patients with orbital trauma or nontraumatic orbital emergencies. In the setting of orbital trauma, clinical assessment of the orbits may be hindered by extensive facial soft tissue injury, decreased level of consciousness, or life-threatening injuries to the remainder of the body. Multidetector computed tomography (MDCT) is the modality of choice in the assessment of orbital trauma\textsuperscript{1} because of its many advantages over other imaging modalities. MDCT is superior to conventional radiography due to its faster acquisition; it may be performed concomitantly with CT imaging of additional body parts frequently obtained in the setting of multi organ injuries. Additionally, it only requires 1 head position, and it allows for the assessment of the orbital soft tissues, is more sensitive for detection of fractures, and may be reformatted in multiple projections and in 3 dimensions. Ultrasound is user dependent and contraindicated in the setting of suspected open globe injury. MR imaging cannot be performed in the setting of potential intraorbital metallic foreign body.

Patients also seek emergency care for many nontraumatic orbital conditions causing a multitude of presenting symptoms to include: vision loss, scotoma, eye pain, ophthalmoplegia, diplopia, orbital bruit, proptosis, or enophthalmos. Cross-sectional imaging with either contrast-enhanced MR imaging or MDCT is helpful in differentiating the numerous etiologies of disease states that present with these symptoms. MR imaging is particularly useful in the setting of nontraumatic emergencies due to its superior evaluation of the orbital soft tissues with respect to MDCT. In addition, conventional angiography is useful for the diagnosis and treatment of vascular orbital emergencies, such as carotid–cavernous fistulae.
angiography may be indicated in select cases in which a vascular abnormality affecting the orbit is suspected, such as carotid-cavernous fistulae. Although invasive, conventional angiography has advantages over CT angiography (CTA) and magnetic resonance angiography (MRA), as it consists of real-time imaging and allows for endovascular treatment when appropriate.

**ORBITAL SKELETAL TRAUMA**

**Orbital Blowout Fracture**

Orbital blowout fracture is a displaced fracture of an orbital wall directed away from the orbit, which may be characterized as pure, if the orbital rim is spared, or impure, if the orbital rim is involved in the fracture (Fig. 1). The 2 mechanisms of the orbital blowout fracture are termed the hydraulic and the bone conduction mechanisms, which both have shown to result in orbital blowout fractures in experimental models with key differences (Box 1). The inferior orbital wall is most frequently affected by the blowout fracture, followed by the medial orbital wall. Although the medial orbital wall (lamina papyracea) is thinner than the inferior orbital wall, it is supported by osseous struts of the ethmoid sinuses, likely increasing its durability against fracture. Complications that advocate for early surgical repair include extraocular muscle entrapment and enophthalmos, which are typically seen in fracture fragments of greater than 1 cm in size. Internal fixation of orbital blowout fracture typically results in the placement of mesh material to restore orbital volume and provide a barrier against the herniation of intraorbital contents.

A special type of the orbital blowout fracture is the trapdoor fracture. The trapdoor fracture is an inferior orbital blowout fracture in which the inferior rectus muscle or infraorbital fat herniates through the fracture defect into the underlying maxillary sinus with return of the fracture fragment back to its original position; thus, the fracture fragment acts like a trapdoor. These patients will present with signs of entrapment caused by extraocular muscle restriction, resulting in diplopia. On coronal CT imaging, the inferior rectus muscle or extraconal fat herniates inferior to the orbital floor through a non-displaced inferior orbital wall fracture.

**Superior Orbital Wall Fracture**

The superior orbital wall or orbital roof is the only wall that forms a partition between the anterior cranial fossa and intraorbital contents. Fractures through the orbital roof are typically a result of a direct blow to the forehead and usually displace into the orbit, termed orbital blow-in fracture (Fig. 2). Associated frontal sinus fracture is a common finding. Potential complications include proptosis, diplopia, orbital emphysema, dural tear with resultant cerebrospinal fluid (CSF) leak or brain herniation, cerebral contusion, and extension of fracture to the orbital apex. Repair of these potential complications may require both intracranial and extradural approaches. The fractures are best visualized on coronal reformatted CT images.

**Box 1**

**Mechanisms of orbital blowout fractures**

- Hydraulic mechanism—posteriorly oriented force to the orbit causes an acute increase in intraorbital pressure with subsequent fracture of the weakest orbital wall to relieve this pressure.
  - Larger fracture defect
  - May affect the medial orbital wall
  - Commonly results in herniation of orbital contents
- Bone conduction mechanism—a force applied to the orbital rim is transmitted posteriorly until eventual buckling of the affected wall occurs.
  - Smaller, anterior fracture defect
  - Never involves medial wall
  - Rarely results in herniation

Fig. 1. Orbital blowout fracture. Coronal CT image demonstrates the inferiorly displaced fracture of the right orbital floor with herniation of intraorbital fat and inferior rectus muscle into the defect (arrow). Clinical correlation for entrapment should be performed.

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Zygomaticomaxillary Complex Fracture

Zygomaticomaxillary complex (ZMC) fracture is caused by a direct traumatic blow to the malar eminence, resulting in diastasis through the 4 sutures that attach the zygoma to the remaining face and calvarium (Fig. 3). Historically, this fracture pattern was described by the misnomer tripod fracture, as a fracture through the zygomaticofrontal, zygomaticomaxillary, and zygomaticotemporal sutures could be discerned by conventional radiography. However, fracture additionally extends through the zygomaticosphenoid suture. The zygoma forms portions of the inferior and lateral orbital walls, anterior and posterolateral maxillary sinus walls, and zygomatic arch. Displaced ZMC fractures that rotate along the axis of the zygomaticosphenoid suture or concomitant orbital floor blowout fractures can result in enophthalmos, which may necessitate intraorbital reconstruction in addition to ZMC fracture fixation.

Naso-Orbitoethmoid Complex Fracture

Naso-orbitoethmoidal (NOE) complex fracture is caused by posteriorly oriented high-impact force...
applied to the nasal region with transmission through the nasal cavity, medial orbital walls, and ethmoid sinuses (Fig. 4). This fracture complex typically results in severe comminution and telescoping of the bilateral nasal bones and septum, ethmoid sinuses including the cribriform plate, and medial orbital walls. Frequent complications caused by NOE fractures include proptosis due to a decrease in intraorbital volume, telecanthus from a medial canthal tendon injury, and CSF rhinorrhea caused by fracture through the cribriform plate and tear of the apposed dura. The Markowitz and Manson classification system separates NOE fracture complexes by comminution of the lacrimal fossa and involvement of the medial canthal tendon into 3 types (Box 2).

LeFort Complex Fractures

At the turn of the twentieth century, the French surgeon Rene LeFort described 3 types of midface fracture patterns resulting in varying degrees of craniofacial dissociation that resulted from high-impact forces applied to the midface of cadavers (Fig. 5). Disruption of the pterygomaxillary junction is the commonality shared by the 3 patterns, and it must be present to characterize a fracture pattern as a LeFort fracture. Although the 3 types of complex fracture patterns described by LeFort were characterized separately and symmetrically across the midface, these fracture patterns may be seen concomitantly and/or asymmetrically involving the 2 halves of the midface. Each of the LeFort fracture types affects unique portions of the midface, which can simplify characterization (Box 3). The LeFort type I fracture complex does not involve the orbit and will therefore not be discussed.

LeFort type II fracture complex, also known as the pyramidal fracture, results in a pyramid-shaped fracture of the central midface that may move independently from the remaining lateral face and skull base. Fracture extends obliquely inferolaterally from the nasofrontal suture (apex of pyramid) through the medial orbital walls, orbital floors, and maxillary sinus walls along the zygomaticomaxillary sutures. Involvement of the inferior orbital walls is unique to the LeFort type II fracture complex. Axial and coronal reformatted CT images enable detection of the obliquely oriented fractures extending through the medial and inferior orbital walls.

LeFort type III fracture complex produces true craniofacial dissociation. Fracture extends from the nasofrontal suture laterally through the medial orbital walls, lateral orbital walls, and zygomatic arches. Using axial and coronal reformatted CT images to detect extension of fracture through the zygomatic arch is unique to the LeFort III fracture complex, and is therefore a helpful discriminator in distinguishing between LeFort II and III fracture complexes, which both share fracture components through the nasofrontal suture and medial orbital walls.

Orbital Apex Fracture

Fractures of the orbital apex can extend through the optic canal or superior orbital fissure, resulting in damage to the cranial nerves that traverse these structures (Fig. 6). Fracture through the optic canal

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**Box 2**

Markowitz and Manson classification system of naso-orbitoethmoidal fractures

- Type I—medial canthal tendon intact and connected to single large fracture fragment
- Type II—medial canthal tendon intact and connected to single fragment of comminuted fracture
- Type III—medial canthal tendon disrupted with comminuted fracture of the lacrimal fossa
may result in monocular blindness from optic nerve injury caused by direct impactation from fracture fragments or subsequent compression from resultant edema or hemorrhage. In complex trauma patients in whom vision cannot be clinically assessed as a result of extensive soft tissue swelling or obtundation, detection of orbital apex fracture by MDCT may be the only method to diagnose potential vision-compromising injury.12

Two clinical syndromes may develop as a result of orbital apex fractures, the superior orbital fissure syndrome, and the orbital apex syndrome.13 The superior orbital fissure syndrome is caused by fracture-related injury to cranial nerves III, IV, V1, and VI as they traverse the superior orbital fissure, thus causing ophthalmoplegia, diplopia, and ptosis. The addition of injury to the optic nerve at the orbital apex is termed the orbital apex syndrome, and would add monocular vision loss to the patient’s symptoms. On CT, special attention is needed to assess the fat about the intracanalicular optic nerve in the setting of fracture. Any soft tissue attenuation along the optic nerve should be deemed suspicious for potential edema or hemorrhage within the confined space of the optic canal. On MR imaging, replacement of the normal T1 hyperintense fat within the optic canal or abnormal T2 hyperintensity within the optic canal or adjacent optic nerve also correlates to the injury. Injury to the optic nerve at the orbital apex is an emergency that may prompt high-dose steroid therapy or potential surgical decompression to prevent permanent blindness.

**TRAUMATIC GLOBE INJURY**

**Ocular Hemorrhage and Detachments**

Blunt and penetrating injury to the globe can result in several ocular hemorrhage patterns that are

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**Box 3**

**LeFort complex fractures unique features**

- Type I—lateral margin of nasal fossa and inferior nasal septum
- Type II—inferior orbital rim
- Type III—lateral orbital rim and zygomatic arch

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**Fig. 5.** LeFort complex fractures. (A) Coronal CT image demonstrates fractures through the pterygoid plates (black arrowheads), a requirement for LeFort complex fractures. In addition, right zygomatic arch fracture is present (white arrowhead). (B) Coronal CT image demonstrates obliquely oriented LeFort II fracture through the right medial orbital wall, inferior orbital wall (black arrow), and inferolateral maxillary sinus wall and transversely oriented LeFort III fracture through the right lateral orbital wall (white arrow). (C) Obliquely oriented 3-dimensional volume rendered CT of the same patient demonstrates fractures of the nasal bridge/right medial orbital wall, right inferior orbital wall (black arrow), right lateral orbital wall (white arrow), and right zygomatic arch (white arrowhead), consistent with a combination of right LeFort II and III complex fractures.

**Fig. 6.** Orbital apex fracture. Axial CT image demonstrates nondisplaced fracture through the medial aspect of the right optic canal (arrowhead), which may result in traumatic optic neuropathy.
distinguishable on cross-sectional imaging (Fig. 7A). Hyphema, hemorrhage within the anterior chamber of the globe, is recognized on CT as layering hyperdensity anterior to the lens. Although hyphema is easily recognized by clinical examination and often does not require radiological diagnosis, it should prompt the radiologist to pay close attention to the posterior segment, as ophthalmoscopic examination is compromised from the intervening anterior chamber blood products.

Vitreal hemorrhage, hemorrhage within the vitreous, demonstrates heterogeneous hyperdensity as blood products mix with the vitreous humor on CT (Fig. 7B). Subhyaloid hemorrhage, hemorrhage external to the hyaloid membrane that covers the vitreous and separates it from the retina, is defined on CT by a layer of hyperdensity that usually pools anterior to and covers the optic disc with a sharp margin between the subhyaloid hemorrhage and vitreous (see Fig. 7A).

Retinal detachment, hemorrhage or fluid between the retina and choroid or between layers of the retina resulting in separation of these layers, is characterized on imaging by lentiform-shaped collections (Fig. 8A). The retina extends to, but not across, the optic disc; thus diffuse retinal detachment gives a V-shaped appearance on axial cross-sectional images with apex at the optic disc. Choroidal melanoma is a potential malignant cause of spontaneous retinal detachment and will appear as a mushroom-shaped enhancing mass extending from the ocular wall into the vitreous (Fig. 8B).15

Choroidal detachment is a result of hemorrhage or fluid between the choroid and sclera with separation of these layers (Fig. 9). On CT and MR imaging, lentiform-shaped fluid/hemorrhage extends along the inner surface of the posterior aspect of the globe. In contrast to retinal detachment, choroidal detachment spares the posterior third of the globe without extension to the optic nerve insertion and may extend anteriorly to the ciliary body beyond the location of the ora serrata.16

Subtenon hemorrhage is a hematoma situated between the sclera and Tenon capsule (see Fig. 7C). On CT, subtenon hemorrhage appears as a lentiform-shaped hyperdensity along the posterior outer surface of the globe and may be difficult to distinguish from retrobulbar hematoma.

Intraocular hemorrhage may result from an acute increase of intracranial pressure that induces retinal venous hypertension from intracranial subarachnoid hemorrhage, a condition termed Terson syndrome (Fig. 10). This finding has been linked to increased morbidity and deserves consultation with ophthalmology to prevent permanent vision impairment.

**Traumatic Lens Injury**

Lens subluxation or dislocation is an injury associated with partial or complete tearing of the zonular attachments to the crystalline lens, respectively (Fig. 11A, B). On CT, lens dislocation is recognized by abnormal positioning of the lens, which more commonly is displaced posteriorly into the dependent vitreous. Anterior lens dislocation is less common and may demonstrate subtle asymmetric decrease in anterior–posterior dimension of the anterior segment, which may be indistinguishable from anterior segment open globe injury from open globe injury from

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**Fig. 7.** Orbital hemorrhage. (A) Axial CT image demonstrates irregular contour of the left globe, consistent with open globe injury. Hyperattenuation within the anterior chamber and in the dependent portion of the posterior segment with fluid/hemorrhage layer is consistent with hyphema (black arrow) and subhyaloid hemorrhage (white arrowheads), respectively. (B) Axial CT image demonstrates heterogeneous hyperdensity within the right vitreous body, consistent with vitreous hemorrhage. (C) Axial CT image demonstrates hyperdensity along the outer posterior margin of the left globe, consistent with subtenon hemorrhage (black arrowheads). Ill-defined hyperdensity in the retrobulbar soft tissues consistent with retrobulbar hematoma resulting in proptosis.
corneal laceration and requires correlation with ophthalmologic examination. The most severe form of lens dislocation is lens extrusion, in which the crystalline lens is ejected from the globe through an open defect and is therefore not visualized on imaging. Lens subluxation is distinguished on CT by appropriate positioning of a portion of the crystalline lens with abnormal angulation of the portion of the lens in which the zonular attachments are disrupted.

**Fig. 8.** Retinal detachment. (A) Axial T2 FLAIR MR imaging demonstrates V-shaped hyperintense collection along the posterior margin of the left globe that extends to, but not across the optic disc, consistent with retinal detachment. (B) Axial T1-weighted MR imaging demonstrates a hyperintense mass along the posteromedial globe with adjacent T1 hyperintense retinal detachment (arrow), consistent with choroidal melanoma (arrowheads).

**Fig. 9.** Choroidal detachment. Axial T1-weighted MR imaging demonstrates bilateral biconvex hyperintense collections along the inner lateral margins of the globes, which do not extend to the optic discs, consistent with choroidal detachment (arrowheads).

**Fig. 10.** Terson syndrome. Axial CT image demonstrates hyperintensity along the posterior ocular walls that spares the optic discs, consistent with subretinal hemorrhages (white arrowheads). Hyperdensity throughout the extra-axial space is a result of diffuse nontraumatic subarachnoid hemorrhage (black arrowheads) in a patient with a ruptured posterior communicating artery aneurysm (not shown).
Traumatic cataract is an opacity of the crystalline lens as a result of blunt or penetrating injury to the globe, or less commonly from electrical shock (Fig. 11C). On CT, decreased attenuation of the affected lens is the result of an ingress of fluid with ensuing edema.18

**Open Globe Injury**

Open globe injury or rupture is a full-thickness injury to the sclera, cornea, or both, as a result of blunt or penetrating injury to the globe, and it is associated with a high rate of monocular vision loss. On CT, when open globe injury is associated with loss of intraocular pressure, the prototypical flat tire sign is present, demonstrating a loss of volume and normal spherical shape of the globe (Fig. 12). Full-thickness corneal laceration can demonstrate decreased volume of the anterior segment with diminished anterior–posterior dimension of the anterior chamber on CT. Other CT findings that suggest open globe injury include lens extrusion, intraocular foreign body, vitreal hemorrhage, and intraocular air. CT is approximately 80% sensitive in the detection of open globe injury,19 and must be used in concert with intraoperative ophthalmologic examination for the complete assessment of occult injury.

**Retrobulbar Soft Tissue Injury**

Retrobulbar traumatic injuries include retrobulbar hematoma, subperiosteal hematoma, orbital emphysema, extraocular muscle injury, and optic nerve injury. Retrobulbar hematoma is typically ill-defined hyperattenuation within the soft tissues posterior to the globe (see Fig. 7C). Subperiosteal hematoma represents a collection of blood underlying the periosteum of an orbital wall. CT shows hyperattenuation along the affected orbital wall with convexity of hematoma projecting into the orbit, which may result in proptosis. Orbital emphysema is defined as the presence of air in the orbit, typically the result of orbital wall fracture in communication with an adjacent paranasal sinus. Orbital emphysema is typically a benign, self-limited condition; however, air tracking along the optic nerve sheath may result in optic nerve ischemia. Any space occupying material within the retrobulbar soft tissues, including retrobulbar hematoma and orbital emphysema, may result in orbital compartment syndrome, a potentially
devastating vision-threatening complication of acutely increased intraorbital pressure. Orbital compartment syndrome is a clinical diagnosis; however, CT is helpful in defining the location of the precipitating lesion or discovering findings that are associated with poorer prognosis, such as posterior tenting of the globe. Orbital compartment syndrome is a clinical diagnosis; however, CT is helpful in defining the location of the precipitating lesion or discovering findings that are associated with poorer prognosis, such as posterior tenting of the globe. The extraocular muscles can be directly injured from orbital trauma. CT findings of increased size and/or hyperattenuation within the muscle are consistent with edema/intramuscular hematoma. Partial or complete disruption of the muscle may result from laceration. Avulsion from tendinous insertion on the globe may also occur. Herniation of the inferior rectus muscle into the defect is a common complication of inferior orbital blowout fracture (see Fig. 1).

Traumatic optic neuropathy (TON) is a condition in which direct or indirect trauma results in acute injury to the optic nerve. Most commonly, trauma results in posterior indirect optic neuropathy, thought to be caused by transmitted forces resulting in shearing injury of the intracanalicular portion of the optic nerve. TON is a clinical diagnosis in which decreased visual acuity and afferent pupillary defect are present. MDCT is helpful in cases of TON to assess for optic canal fracture, edema/hematoma within the optic canal, optic nerve sheath or intracanal hematoma, or foreign body/fracture fragments impinging upon the optic nerve that would direct potential medical (eg, high-dose steroids) or surgical intervention (eg, optic canal decompression). Diffusion tensor imaging may demonstrate reduced diffusivity within the affected optic nerve. Optic nerve avulsion is an uncommon, severe injury resulting in transection of the optic nerve, which may occur at the level of the optic nerve head, intracanalicular portion, or anterior to the chiasm, thought to result from severe rotational forces to the globe. MDCT or MR imaging may demonstrate subtle discontinuity of the optic nerve with an intact optic nerve sheath. Optic nerve transection, a complete disruption of the optic nerve and sheath, may occur anywhere along the course of the optic nerve, often caused by the severing action of a fracture fragment or penetrating foreign body.

**Orbital Foreign Body**

Radiological evaluation of the orbit for foreign bodies is crucial in the setting of orbital trauma. Although conventional radiography is able to detect the presence of an opaque intraorbital foreign body (Fig. 13A), it does not assess the exact location with respect to the remaining orbital soft tissues. Therefore, conventional radiography is typically reserved in the nonemergent setting to prove the presence or absence of a suspected opaque foreign body prior to MR imaging. MDCT is the modality of choice in the initial assessment of the intraorbital foreign body, as it is sensitive, allows for detection of associated injuries to the orbit and soft tissues, and allows safe detection of ferromagnetic materials unlike MR imaging (Fig. 13B). Second-tier imaging modalities that

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**Fig. 13.** Intraocular metallic foreign body. (A) A frontal radiograph of the orbits demonstrates a punctate metallic foreign body in the right orbit (arrowhead). (B) Axial noncontrast CT image of the same patient demonstrates a metallic intraocular foreign body.
may be used to identify foreign bodies include ultrasound and MR imaging. B-mode ultrasound is useful for detection of intraocular foreign bodies, but it is user-dependent and not typically able to characterize the foreign material. Additionally, contact ultrasonography is contraindicated in the setting of open globe injury. MR imaging is reserved as a second-line modality to detect suspected nonmetallic foreign body, if CT is negative. MR imaging is contraindicated when there is suspected metallic intraocular foreign body, since electromagnetic torque forces may cause foreign body migration and resultant ocular damage and potential vision loss.

It is useful to categorize intraocular foreign bodies as metallic (eg, iron, copper, or metal alloys), nonmetallic inorganic (eg, glass or plastic), and organic materials (eg, wood, dirt, or plant material) for the purpose of preoperative planning. Metallic foreign bodies are more hyperdense than bone on CT, with Hounsfield units in the thousands (Fig. 14). Nonmetallic foreign bodies are also typically hyperattenuating when compared with orbital soft tissues, but less than metallic materials. Organic materials can have a wide range of attenuation. In particular, wood can be air attenuation and may be confused with orbital emphysema. Therefore, if the air conforms to a geometric shape or is not in a nondependent position, for the possibility of intraorbital wooden foreign body should be considered (Fig. 15).

The decision to undergo surgical extraction of intraocular foreign body depends on the size, location, foreign body material, damage to surrounding structures, and potential damage by surgical exploration. Metallic foreign bodies are typically well tolerated; however, intraocular copper may result in a sterile endophthalmitis (acute chalcosis), and intraocular iron may result in siderosis bulbi. Nonmetallic inorganic foreign bodies, such as plastic, are also typically well tolerated. However, organic foreign materials typically contaminate the globe with microorganisms, thus increasing the risk of endophthalmitis and necessitating prompt surgical removal.

It is important for the radiologist to be familiar with common sites and appearances of orbital calcifications and ocular postsurgical changes that may mimic intraocular foreign bodies. Trochlear calcifications are small curvilinear calcifications in the anteromedial orbit at the site of the trochlear apparatus, the fibrous sling containing the superior oblique tendons (Fig. 16A). Phthisis bulbi is the result of end-stage damage to the globe, which appears shrunken and irregular with dystrophic calcifications on CT and may mimic open globe injury with intraocular foreign body (Fig. 16B). Optic drusen are calcified mucopolysaccharides at the optic nerve heads and should not be confused with foreign bodies layering dependently in the vitreous (Fig. 16C). Retinal detachment tamponade is a procedure in which the posterior segment is filled with silicone oil or gaseous compounds (eg, air or sulfur hexafluoride) in order to appose the detached ocular wall layers. On MDCT, silicone oil is hyperattenuating, and the gaseous compounds have air attenuation. These compounds may be confused with vitreous hemorrhage or

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Fig. 14. Intraorbital metallic foreign body. (A, B) A scout image and 3-dimensional volume rendered CT images demonstrate left intraorbital extension of car keys in a patient status after motor vehicle collision.
Fig. 15. Intraorbital wooden foreign body. (A, B) Axial and sagittal CT images demonstrate a pencil within the medial aspect of the left orbit extending into the left anterior cranial fossa. The pencil graphite is hyperdense, and the wood component is air density.

Fig. 16. Potential orbital trauma mimics. (A) Trochlear calcification. Axial CT image demonstrates curvilinear hyperdensities in the anteromedial orbits at the site of the trochlear apparatus, consistent with trochlear calcifications (arrows). (B) Phthisis bulbi. Axial CT image demonstrates a shrunken and partially calcified globe. (C) Optic disc drusen. Axial noncontrast CT image demonstrates bilateral punctate calcifications at the optic discs, consistent with optic disc drusen (arrowheads). (D, E) Retinal detachment treatment. (D) Axial CT image demonstrates left intravitreal air status after vitrectomy and air tamponade. (E) Axial CT image demonstrates hyperdensity in the right vitreous body status after vitrectomy with intravitreal silicone oil tamponade.
ocular emphysema, and thus knowing the patient’s recent past surgical history is important in the setting of trauma (Fig. 16D, E).

NONTRAUMATIC ORBITAL EMERGENCIES

Orbital Infection

Radiological assessment of the orbit with contrast-enhanced CT or MR imaging is commonly used to differentiate preseptal cellulitis (Fig. 17A) from orbital cellulitis (Fig. 17B) in the emergency setting. These entities are stratified by infection limited to the soft tissues superficial to the orbital septum (preseptal cellulitis) or infection extending deep to the orbital septum (orbital cellulitis). The orbital septum is a fibrous membrane extending from the orbital rim periosteum and along the tarsal plates of the eyelids that acts as a natural barrier to the spread of infection. Preseptal cellulitis is a less severe infection that may be treated with oral antibiotics in an outpatient setting. On the other hand, orbital cellulitis is treated more aggressively with intravenous antibiotics, as infection may be complicated by vision loss, ophthalmoplegia, or intracranial spread of infection. The potential intracranial complications of orbital cellulitis are identifiable on MDCT or MR imaging (Box 4). Surgery may be required to drain concomitant subperiosteal or orbital abscesses.

The orbital septum is usually not visualized by MDCT or MR imaging, but may be imagined as a line connecting the margins of the orbital rim and anterior to the globe. Preseptal cellulitis is characterized by soft tissue swelling, stranding, and patchy enhancement of the soft tissues superficial to the orbital septum on contrast enhanced MDCT or MR imaging. Orbital cellulitis will demonstrate inflammatory stranding and enhancement posterior to the orbital septum. A subperiosteal abscess is defined as a rim-enhancing fluid collection along the orbital wall with adjacent paranasal sinus infection, which is most commonly seen along the medial orbital wall with associated acute ethmoid sinusitis (Fig. 17C). On MR imaging, subperiosteal or intraorbital abscesses will demonstrate rim enhancement with internal T2 hyperintensity and restricted diffusion.

Acute invasive fungal sinusitis is an aggressive infection with high mortality affecting immunocompromised patients, particularly those with uncontrolled diabetes or neutropenia (eg, bone marrow transplant recipients or patients on chronic immunosuppression), which may result in intraorbital extension (Fig. 18). On contrast-enhanced MDCT, intraorbital involvement is seen as variably enhancing soft tissue within the orbit and adjacent opacification of the involved paranasal sinus with or without intervening osseous destruction. MR imaging demonstrates T2 hyperintensity and postgadolinium enhancement of the involved intraorbital soft tissues. Treatment includes radical debridement.

**Box 4**

Intracranial complications of orbital cellulitis

- Cavernous sinus thrombosis
- Intracranial abscess
- Subdural empyema
- Meningitis

![Fig. 17. Preseptal and orbital cellulitis.](image)
intravenous antifungals, and treatment of the underlying immunodeficiency.\textsuperscript{30,31}

**Orbital Inflammatory Conditions**

Idiopathic orbital inflammatory syndrome (IOIS), otherwise known as orbital pseudotumor, is a nonspecific inflammation of the orbit without local or systemic causes (Fig. 19). Patients present with orbital pain and a wide variety of additional symptoms depending on the location of inflammation, including proptosis, restricted eye movement, conjunctival injection, or impaired vision.

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**Fig. 18.** Invasive fungal sinusitis. (A) Axial T1 postgadolinium fat saturated image demonstrates ill-defined enhancement in the right orbital apex at the optic nerve (black arrowheads) extending intracranially along the anterior margin of the right middle cranial fossa (white arrowhead). (B) Coronal T1 postgadolinium fat saturation image demonstrates inflammatory changes of paranasal sinuses and prior bilateral uncinectomies and ethmoidectomies. Nonenhancement of the right aspect of the hard palate is consistent with necrosis secondary to angioinvasion (arrow).

**Fig. 19.** IOIS. (A) Axial T2-weighted fat saturated image demonstrates enlargement and hyperintensity involving the left medial rectus muscle including the anterior tendinous portion, a distinction that differentiates IOIS from thyroid opthalmopathy. (B) Coronal T1 postgadolinium fat saturation image demonstrates ill-defined enhancement and thickening involving the left medial rectus muscle, consistent with myositic subtype of IOIS.
Contrast-enhanced MR imaging demonstrates nonspecific, ill-defined inflammatory stranding and enhancement that may affect a number of orbital subsites, including: extraocular muscles, lacrimal glands, retrobulbar fat, ocular walls, orbital apex, optic nerve sheath (e.g., optic perineuritis), or a combination of these structures. Differential diagnostic considerations of orbital inflammation include orbital cellulitis, granulomatosis with polyangiitis, sarcoidosis, and lymphoma. Apparent diffusion coefficient (ADC) values may be helpful in differentiating IOIS from lymphoma and orbital cellulitis.32 Although idiopathic orbital inflammatory syndrome is a diagnosis of exclusion, empiric treatment with systemic steroids is attempted in suspected cases. Biopsy and second-line treatment with low-dose radiotherapy or cytotoxic chemotherapy are reserved for refractory cases. Tolosa-Hunt syndrome is a subtype of idiopathic orbital inflammatory syndrome involving the cavernous sinus, resulting in painful ophthalmoplegia.33

Granulomatosis with polyangiitis is a syndrome associated with granulomatous inflammation and small- or medium-sized vessel vasculitis that predominantly involves the kidneys and respiratory tracts and is associated with cytoplasmic staining antineutrophil cytoplasmic antibodies (c-ANCA). On imaging, nonspecific mass-like inflammation may involve any regions of the orbital soft tissues (Fig. 20). Attention to the adjacent paranasal sinuses and nasal cavity may demonstrate extensive mucosal inflammation with variable destruction of the nasal septum and sinus walls.34,35

Ocular Infection/Inflammation

Infection or inflammation may affect any of the globe layer, the anterior or posterior segment, or a combination. Scleritis, inflammation of the sclera, may be designated as anterior or posterior if the inflammation occurs anterior or posterior to the extraocular muscle attachments. Anterior scleritis results in eye pain and erythema of the visualized sclera. On the other hand, posterior scleritis presents with eye pain, but without erythema. Uveitis is defined as inflammation involving the uvea, a combination of the iris, ciliary body, and choroid. Uveitis can be subdivided into anterior, intermediate, and posterior subtypes depending on the components of the affected uvea. In general, diagnosis of inflammation of the ocular layers is determined through ophthalmologic examination with the aide of B-mode ultrasound. However, eye pain and visual disturbance in the absence of erythema, such as in cases of posterior scleritis or posterior uveitis, may prompt contrast-enhanced CT or MR imaging in the emergency setting. Contrast-enhanced T1 and T2 weighted sequences with fat saturation in the axial and sagittal planes are the ideal sequences to assess inflammation involving the ocular layers of the posterior segment. In normal patients, the choroid is the only normally enhancing layer, given its rich vascularity. The inflamed ocular layer will demonstrate thickening, contrast enhancement, and T2 hyperintensity, which may be focal, diffuse, or nodular in appearance (Fig. 21).

Endophthalmitis is infection of the ocular cavities and adjacent structures resulting in pain, edema, and decreased vision (Fig. 22). This condition is typically the result of direct inoculation of bacteria or fungus into the globe from ocular surgery or trauma, termed exogenous endophthalmitis, and requires surgical intervention with administration of intracocular antibiotics or potential enucleation. Rarely, hematogenous seeding of the ocular cavities may affect immunocompromised or chronically ill patients, typical in the setting of uncontrolled diabetes, termed metastatic or endogenous endophthalmitis. Panophthalmitis is the most severe form of endophthalmitis, in which the infection encompasses the entirety of the structures of the eyeball. Acute endophthalmitis may be a diagnostic dilemma, particularly in cases of endogenous endophthalmitis, given its...
low incidence and nonspecific symptoms, prompting radiological assessment. Contrast-enhanced CT may reveal relative hyperattenuation of the affect vitreous, thickening and enhancement of the ocular wall, and/or adjacent periorbital inflammation. Contrast-enhanced MR imaging is the modality of choice in the assessment for endophthalmitis, with possible findings to include abnormal increased fluid-attenuated inversion recovery (FLAIR) signal within the vitreous, regions of restricted diffusion within the globe, ocular wall thickening and enhancement, and/or periorbital inflammatory changes.\(^{36}\)

### Vascular Orbital Emergencies

Carotid–cavernous fistulae (CCF) are an abnormal communication between the carotid arterial system and the cavernous sinus, typically resulting in conjunctival chemosis, pulsatile proptosis, pain, ophthalmoplegia, and progressive vision loss (Fig. 23). CCF can be classified according to the cause, traumatic or spontaneous, or whether the arterial supply arises directly from the internal carotid artery or indirectly from meningeal branches of the internal carotid artery, external carotid artery, or both (Box 5).\(^{37}\) CT angiography is more sensitive than MRA in the detection of CCF.\(^{38}\) Both can demonstrate enlarged cavernous sinus with increased internal vascularity, enlarged superior ophthalmic vein, proptosis, and possible enlargement/edema within the extraocular muscles of the affected side. These findings may be unilateral or bilateral. Digital subtraction angiography (DSA) is required for definitive diagnosis and possible endovascular treatment. DSA will demonstrate retrograde flow within the superior ophthalmic vein and corresponding cavernous sinus in the arterial phase of injection and allow for identification of the site of fistula, whether directly from the internal carotid artery or internal carotid artery (ICA)/external carotid artery (ECA) feeding vessels in the case of indirect fistula. Direct CCFs may be treated endovascularly with transarterial detachable balloon occlusion, transvenous coil embolization, covered stent across the fistula, or carotid sacrifice. Indirect CCFs may be initially treated with manual carotid compression. If this is unsuccessful,
Endovascular treatment with transvenous coil embolization is typically curative.\textsuperscript{39}

Orbital venous varices are congenital low-flow venous malformations with connection to the systemic venous system that dilate in the setting of increased venous pressure (e.g., Valsalva maneuver, coughing, etc.) and are the most common cause of spontaneous retrobulbar hemorrhage. Patients typically exhibit reversible proptosis, elicited by increased venous pressure. Patients may present to the emergency department with sudden painful proptosis caused by non-traumatic retrobulbar hemorrhage or variceal thrombosis. Contrast-enhanced CT performed with and without provocative maneuvers (e.g., Valsalva maneuver or internal jugular vein compression) will demonstrate increased size of the enhancing intraorbital mass during the increase in venous pressure.\textsuperscript{40} The orbital venous varix may not be visualized on the CT performed without provocative maneuver. CT may also demonstrate internal phleboliths within the varix, retrobulbar hemorrhage if present, or hyperattenuating clot without enhancement in the setting of thrombosis. Contrast-enhanced MR imaging will demonstrate variable T1 and T2 signal intensity within the varix, diffuse contrast enhancement, and enlargement of the varix with provocative maneuver.

Orbital venous lymphatic malformations (OVLMs) are benign congenital low-flow vascular malformations that may grow slowly with time, resulting in progressive proptosis (Fig. 24). OVLMs are characterized histopathologically by numerous dilated interconnecting vascular channels, which are not connected to the systemic venous system, in contrast to the previously described orbital venous varix. These masses typically demonstrate an infiltrative transspatial growth pattern in which they violate natural tissue planes that restrict other pathologies; therefore, they may involve both intraconal and extraconal compartments or preseptal and postseptal compartments. These lesions may acutely enlarge due to spontaneous internal hemorrhage or upper respiratory tract infection, resulting in sudden increase in proptosis or optic nerve injury, prompting emergent assessment.

**Box 5**

**Barrow classification of spontaneous carotid-cavernous fistulae**

- Type A—direct high-flow shunt between internal carotid artery (ICA) and cavernous sinus
- Type B—indirect low flow shunt between meningeal branches of the ICA and cavernous sinus
- Type C—indirect low flow shunt between meningeal branches of the external carotid artery (ECA) and cavernous sinus
- Type D—indirect low flow shunt between the cavernous sinus and meningeal branches of both ECA and ICA

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**Fig. 23.** Carotid-cavernous fistula. (A) 3-dimensional time-of-flight magnetic resonance angiography axial maximal intensity projection image demonstrates dilatation of the left superior ophthalmic vein (white arrow) consistent with arterialized flow. (B) Digital subtraction angiography, right internal carotid artery injection, frontal projection, demonstrates contrast opacification of the cavernous sinuses and left superior ophthalmic vein (black arrow) in the arterial phase, consistent with carotid-cavernous fistula.
MR imaging is the modality of choice in characterization of these lesions, which typically demonstrate multiloculated, trans-spatial T2 hyperintensity with thin, barely perceptible walls. In the setting of internal hemorrhage, multiple fluid–fluid levels with variable T1/T2 signal corresponding to hemorrhage products of varying ages are present. The venous components of these lesions will demonstrate enhancement, while the typically, nearly imperceptible walls and septations of the lymphatic components show thickening and enhancement in the setting of upper respiratory tract infection. Surgical removal is controversial and typically reserved for cases complicated by optic nerve compression, corneal exposure, or intractable pain, as complete resection is limited by the infiltrative, trans-spatial growth pattern; recurrence is common.

**SUMMARY**

In conclusion, radiological imaging is essential in the assessment of orbital emergencies. Multidetector CT allows for rapid assessment of osseous and soft tissue injuries in the setting of orbital trauma and can identify orbital or ocular foreign bodies in a majority of cases. The radiologist should be familiar with the midface complex fracture types that affect the orbits and commonly associated complications (eg, medial canthal tendon injury with NOE complex fracture). Contrast-enhanced MR imaging allows for the differentiation of many etiologies of nontraumatic orbital emergencies that present with similar clinical symptoms. Although CT angiography or MRA can typically diagnose carotid– cavernous fistula, conventional angiography is regarded as the gold standard in classification and is necessary for endovascular treatment.

**REFERENCES**