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# Nontraumatic Orbital Emergencies

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**Non-traumatic orbital pathology can often have overlapping imaging features and challenging timely diagnosis. Prompt recognition is necessary due to potential risk of permanent visual loss. In this article we present systematic evaluation of non-traumatic orbital pathology with attention to key imaging features, pathogenesis and potential complications.**
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## Introduction

Nontraumatic orbital pathology can often be a challenging diagnosis that warrants prompt recognition and timely management in the emergency setting. Delay in diagnosis may result in devastating long-term complications including permanent visual loss. Broadly, nontraumatic orbital emergencies can be categorized based on underlying etiopathogenetic factors as summarized in Table 1. Systematically evaluating the imaging pattern and their interaction with clinical features can serve as a key diagnostic tool. In addition, attention to the anatomic organization of the orbit can aid understanding of etiopathogenesis and scrutiny for detection of potential complications.

In this article, we will review clinical presentation and imaging features of common nontraumatic orbital emergencies with emphasis on diagnostic pearls and pitfalls.

## Inflammation

### Idiopathic orbital inflammatory syndrome

Idiopathic orbital inflammatory syndrome (IOIS), also known as orbital pseudotumor, is a benign, nonspecific, orbital inflammation without an underlying identifiable local or systemic etiology.[1,2] This distinguishes it from other disorders of orbital inflammation such as IgG4-related ophthalmic disease, (IgG4-ROD), Sjögren syndrome, granulomatosis with polyangiitis (GPA or Wegener's), and orbital manifestations of sarcoidosis.

Peak incidence of IOIS is in middle age, however rarely it can be seen in pediatric patients. It is more common in females than in males. Patients typically present with acute onset pain, swelling, erythema, ptosis, proptosis, and chemosis. There may be associated visual symptoms such as diplopia and loss of visual acuity. Often involvement is unilateral, or asymmetric. This is in contrast to children that can present with bilateral disease.[1]

Pathologically, the inflammation is nonspecific without features such as seen with a granulomatous or vasculitic processes. The management of IOIS entails corticosterioid therapy and/or NSAIDs to halt the inflammatory cascade. Patients with refractory course may be candidates for immunosuppression such as Rituximab and rarely orbital radiation. Unless there is a complication such as an orbital inflammatory mass or orbital compartment syndrome, surgical management is rarely indicated. However, there may be a rationale for biopsy for tissue diagnosis for pathologic confirmation of IOIS based on the individual clinical factors.

On imaging, both CT and MRI can provide clues to the diagnosis, however MRI with gadolinium enhancement and targeted high resolution orbital sequences provides much greater soft tissue detail and high sensitivity to detection of early changes. As seen in Figure 1, there is focal myositis involving the extraocular muscles (EOM) and associated inflammatory stranding of orbital fat. There may be other findings such as dacroadenitis, uveitis/scleritis, and optic nerve sheath involvement. Occasionally there may be an inflammatory orbital mass or associated osseous sclerosis changes.

### IgG4-related ophthalmic disease

IgG4-ROD is an immune mediated fibroinflammatory disorder characterized by tumefactive inflammation involving lacrimal glands, orbital soft tissues, scleritis, nasolacrimal duct system, infraorbital nerve, and EOM.[3] Rarely there may be associated osseous erosive changes.

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**Table 1** Categories of Nontraumatic Orbital Emergencies

| |
|---|
| **Inflammation** |
| Idiopathic Orbital Inflammatory Syndrome, (IOIS), pphthalmic complications of IgG related disease, sarcoid, granulomatosis with polyangiitis (GPA or Wegener's granulomatosis), allergic granulomatosis and angiits, (Churg Strauss syndrome) and rheumatoid arthritis |
| Optic neuritis, perineuritis, (involvement of optic nerve sheath) |
| Dacroadenitis, dacrocystitis |
| Myositis, (extraocular muscle involvement) |
| Scleritis, episcleritis (lies between Tenon's capsule and sclera, involvement of episcleral vascular plexus) |
| Orbital apicitis (one of the causes of orbital apex syndrome), Tolosa Hunt syndrome |
| **Infection** |
| Orbital, periorbital cellulitis |
| Intraocular, orbital, subperiosteal abscess |
| Dacroadenitis, dacrocystitis |
| Endophthalmitis, panophthalmitis |
| Infectious keratitis |
| **Vascular Conditions** |
| Cavernous sinus thrombosis |
| Cavernous carotid fistula |
| Retinal vasculitis, Retinal Vein occlusion |
| Hemorrhage or rapid proliferation of vascular malformation |
| **Miscellaneous** |
| Intravitreal hemorrhage |
| Retinal detachment |
| Choroidal detachment |
| Complications related to neoplasms such as lymphoma, metastasis, melanoma |
| Idiopathic intracranial hypertension |

Previously many of these cases were included under the umbrella diagnosis of orbital pseudotumor or IOIS. However, it is now increasingly recognized as a distinct pathophysiologic entity often seen with bilateral ocular adenexal involvement and systemic involvement such as autoimmune pancreatitis, lymphadenopathy, salivary gland involvement.[4]

IgG-ROD is commonly seen in the fifth to sixth decades of life, affecting men and women equally and is only rarely seen in pediatric age groups. Most common presentation is painless enlargement of lacrimal glands (Fig. 2). There may be periorbital edema, proptosis, diplopia, or pain related to EOM involvement, (EOM involvement due to IgG4-ROD shown in Fig. 2). Involvement of nasolacrimal duct can result in epiphora. There may be secondary changes to visual acuity, visual field defects due to compressive neuropathy from mass effect. EOM involvement presents a diagnostic overlap with Graves Orbitopathy, however systemic features, disproportionate involvement of lateral rectus muscle, involvement

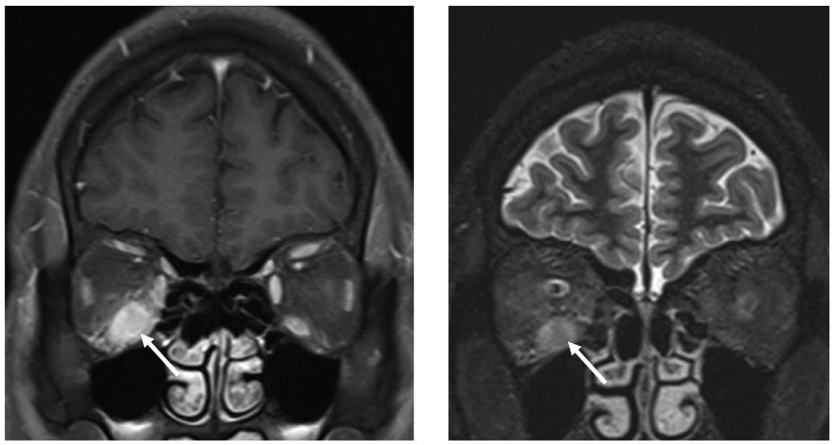

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**Figure 1** I: Coronal contrast enhanced T1w fat saturation sequence showing right inferior rectus muscle enhancement and stranding in the adjacent fat, (white arrow, A). Coronal T2w fat saturation shows edema and enlargement of the right inferior rectus muscle, (white arrow, B). IOIS, Idiopathic Orbital Inflammatory Syndrome.

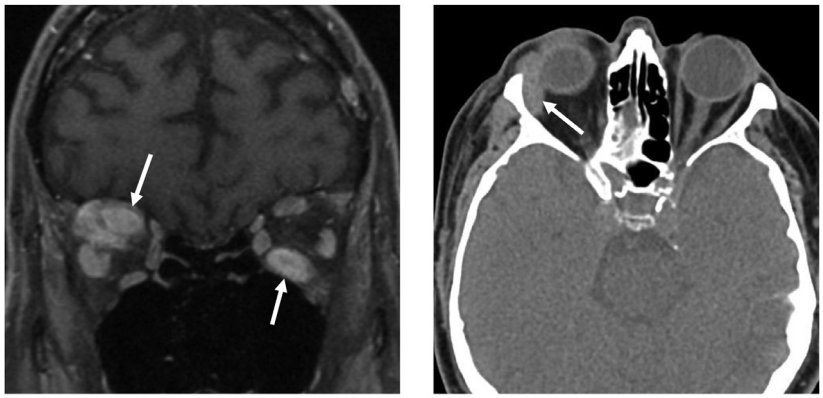

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**Figure 2** IgG4 ROD: Contrast enhanced coronal T1w fat saturation sequence shows enlargement, enhancement and adjacent fat stranding of the right superior rectus muscle and left inferior rectus muscle, (white arrow, A). An axial non-contrast CT showing diffuse enlargement of right lacrimal gland, (white arrow B). IgG4-ROD, IgG4-related Ophthalmic Disease; CT, computed tomography.

of myotendinous junction, and enlargement of infraorbital nerves, (greater in size than optic nerve) are diagnostic clues favoring IgG4-ROD.[5]

Pathologically, there is dense lymphoplasmacytic infiltrate with IgG4+ plasma cells, focal storiform fibrosis, and obliterative phlebitis. There may be associated elevation of serum IgG4, however often serum IgG4 is normal. Tissue sampling to confirm diagnosis and exclude malignancy such as Lymphoma may be necessary. Treatment strategies similar to IOIS rely on steroid, immunosuppression, and rarely orbital radiation.

## Orbital apicitis and Tolosa Hunt Syndrome

Orbital apicitis (OA) and Tolosa Hunt Syndrome (THS), involve inflammation within the orbital apex and cavernous sinus, respectively and can often coexist due to contiguous nature of these structures. These encompass a spectrum of abnormalities affecting the region of orbital apex, superior orbital fissure and cavernous sinus (OA shown in Fig. 3).

Inflammation within the orbital apex is one of the causes for orbital apex syndrome (OAS) which has a poorer prognosis due to potential of optic nerve invasion and intracranial extension through the superior orbital fissure and optic canal.[6] Other noninflammatory causes of OAS include infection, meningioma, lymphoma, trauma, and metastasis. Clinically patients with OAS can present with retro-orbital pain, combination of visual impairment due to optic neuropathy, and multiple other cranial neuropathies including III, VI, and V1 leading to EOM dysfunction.[7]

Surgical decompression of orbital apex is necessary to prevent further visual complications.

THS is characterized by peri-orbital or retroorbital pain, painful ophthalmoplegia, and cranial neuropathies involving III, VI, IV, and V1 due to idiopathic inflammation extending into the cavernous sinus. It is a rare disorder and can be seen in the 4th decade of life with no gender predispositions. Rarely it has been reported in pediatric population.[8] It is however essential to remember that both OAS and THS can have variety of noninflammatory causes including, neoplasm and infections, which must be excluded before the diagnosis is confirmed.

Steroid therapy is the main stay of treatment with other forms of immunosuppression and radiation used rarely as second line treatment for refractory cases. There is dramatic

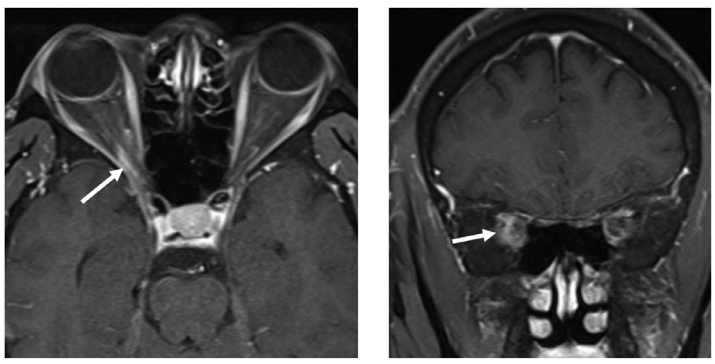

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**Figure 3** Orbital apicitis: Axial (A) and coronal (B) contrast enhanced T1w fat saturation sequences show enhancement in the right orbital apex, (white arrow in A and B). There is no evidence of retrograde extension into the cavernous sinus.

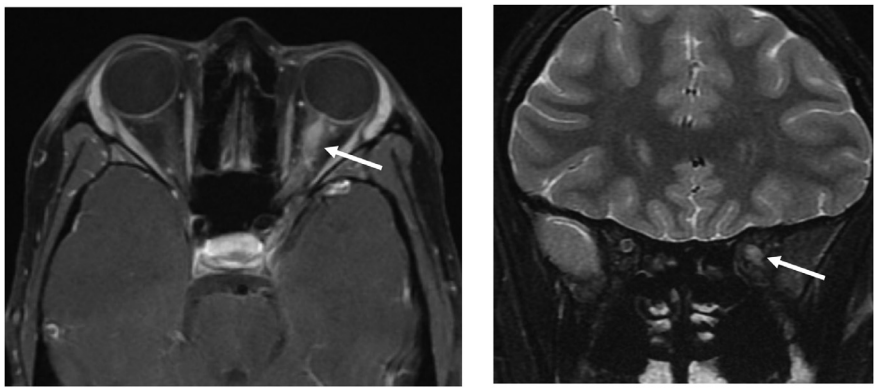

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**Figure 4** Optic neuritis: Axial contrast enhanced T1w fat saturation sequence shows enhancement within the left optic nerve (white arrow, A) and coronal T2w fat saturation sequence shows edema in the left optic nerve, (white arrow, B).

symptomatic recovery however 40%-50% of cases may experience a relapsing remitting course.[9,10]

## Optic neuritis

Optic neuritis refers to inflammation or demyelination within the optic nerve (Fig. 4). It is frequently associated with demyelinating conditions such as multiple sclerosis, in neuromyelitis optics spectrum disorder and Myelin-Oligodendrocyte Glycoprotein (MOG + disease) with the latter 2 associated with antiaquaporin 4 and anti-MOG antibodies. Other less common causes include sarcoidosis, SLE, Behcet's disease, GPA (formerly Wegener's disease), viral infections, radiation therapy, or idiopathic causes.[11,12]

Acute optic neuritis, (ON) is relatively common disorder frequently affecting young Caucasian women in their 30s. Clinically, patients can present with acute onset pain followed by blurring of vision or loss of visual acuity. Occasionally the visual symptoms can be painless. Most typical cases of acute ON are either idiopathic or related to multiple sclerosis. The atypical causes can range from neuromyelitis optics spectrum disorder to infectious/inflammatory causes as detailed above.

On MRI, there is enhancement within the optic nerve with edema and nerve enlargement in acute ON. In patients with chronic ON there may be T2w hyperintensity within the optic nerve with associated atrophy as opposed to enlargement. This is also in contrast to optic perineuritis where the inflammation is restricted to the optic nerve sheath and adjacent fat planes. CT has very limited role in detection of optic neuritis. The changes in the nerve itself may not be well seen while there may be nerve enlargement and adjacent inflammatory fat stranding.

Typical acute onset ON, has a self-limiting course, with treatment with steroids shortening the duration of symptoms. Other atypical causes often may require targeted therapy based on underlying cause, including prolonged steroids, immunosuppression, and plasma exchange.[12]

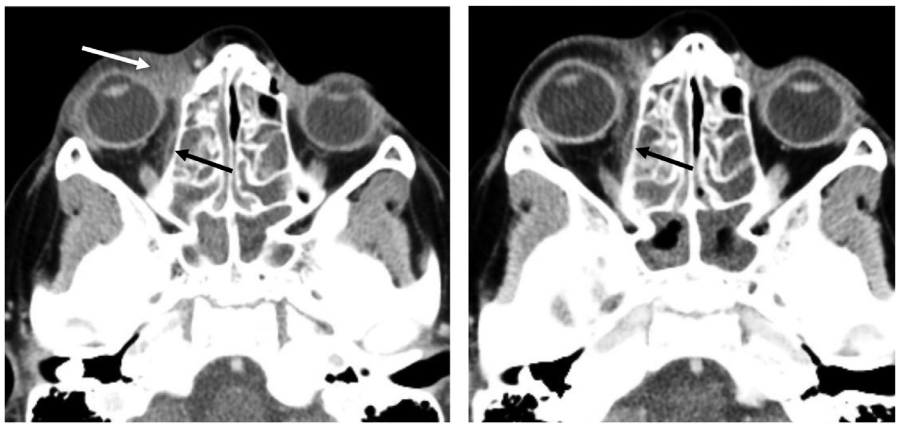

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**Figure 5** Periorbital and orbital cellulitis: Axial contrast enhanced CT of the orbits show soft tissue stranding and thickening in the medial preseptal region, (white arrow, A) and a thin subperiosteal abscess abutting the lamina papyracea, (black arrow A and B). There is adjacent sinus inflammatory disease.

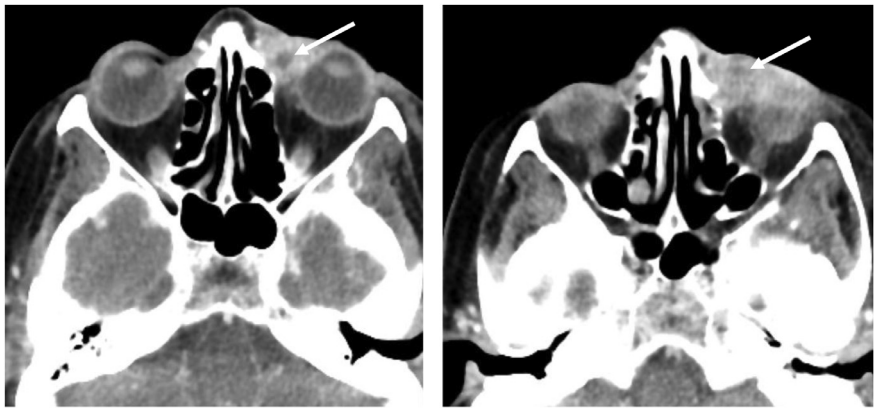

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**Figure 6** Acute dacrocystitis with lacrimal sac abscess: Axial contrast enhanced CT of the orbits shows rim enhancing collection in the left medial canthal region, (white arrow, A) and prominent surrounding inflammatory stranding and soft tissue thickening, (white arrow, B).

# Infection

## Periorbital and orbital cellulitis

Orbital infections can be broadly classified as preseptal (periorbital) and postseptal (orbital) cellulitis (Fig. 5). The orbital septum is thin sheet of fibrous tissue that attaches from the periosteum of the superior and inferior orbital rims and fuses into the aponeurosis of the levator palpabrae superioris for the upper eyelid and tarsal plate for the inferior eyelid. It provides a barrier for spread of infection from the preseptal soft tissues into the orbit proper.

Preseptal orbital cellulitis (periorbital cellulitis) can occur in any age group, but commonly seen in the pediatric age groups, spreading through trauma, or contiguous spread of face, dental or ocular adenexal infections. Orbital cellulitis is commonly a complication of bacterial rhinosinusitis, commonly ethmoid sinus via thin lamina papyracea and septic thrombophlebitis of the traversing valveless veins. Other etiologies include dental, middle ear, or facial infections, dacrocystitis, hematogenous spread from a distant source. Clinically, patients present with orbital pain, fever, diplopia, conjunctival chemosis, proptosis, and eyelid swelling/erythema.[13,14]

Diagnosis can be made both on CT and MRI, with superior sensitivity of contrast enhanced MRI toward complications such as subperiosteal/orbital abscess, intracranial extension such as meningitis/intracranial abscess, and septic thrombophlebitis.

Management consists of oral antibiotics and close clinical follow-up for patients with mild preseptal disease, other more severe preseptal or orbital cellulitis is treated with IV antibiotics. Surgical drainage may be needed for abscess formation.[13]

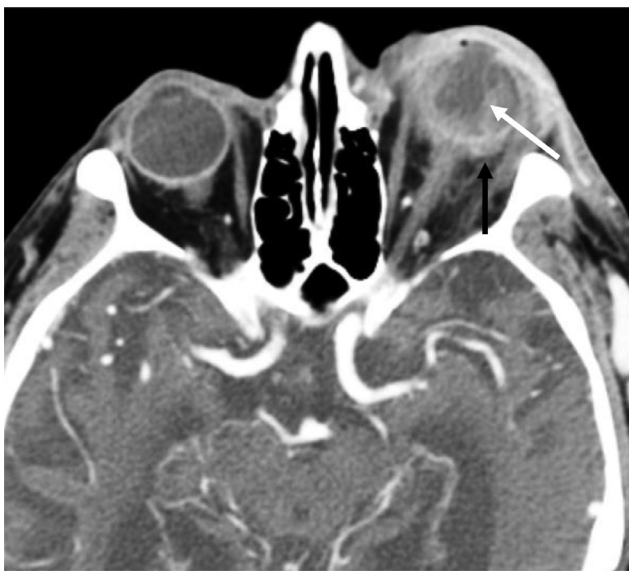

**Figure 7** Endophthalmitis: Axial contrast enhanced CT of the orbit showing enhancement and thickening of the posterior globe with episcleral extentsion, (black arrow), high density within the vitreous and choroidal detachment, (white arrow).

## Acute dacrocystitis with lacrimal sac abscess

Acute dacrocystitis is another form of preseptal cellulitis involving the lacrimal sac that lies anterior to the orbital septum (Fig. 6). It is commonly associated with nasolacrimal duct obstruction and confined to preseptal space due to the attachment of the orbital septum, lacrimal fascia, and medial canthal ligament. However, occasionally it can be complicated by postseptal spread commonly into the inferior and medial orbit. Management involves antibiotics and surgical treatment with dacryocystorhinostomy.[15,16]

## Endophthalmitis

Endophthalmitis refers to purulent inflammation usually due to infection within the aqueous and/or vitreous humors of the eye (Fig. 7). Further extension of involvement into the cornea, sclera, and orbital contents is referred to as Panophthalmitis.

Endophthalmitis, can occur from exogenous and endogenous sources. Exogenous sources include trauma,

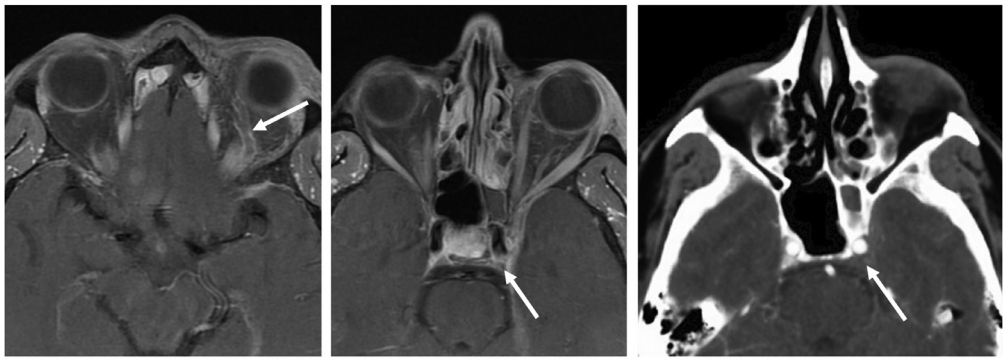


**Figure 8** Cavernous sinus thrombosis: Axial contrast enhanced T1w fat saturation sequences show occluded left superior ophthalmic vein (white arrow, A) and nonenhancing left cavernous sinus, (white arrow B and C).

surgery, or corneal infections. Specifically, cataract surgery, filtering bleb for glaucoma and intravitreal injections for macular degeneration have been complicated by exogenous endophthalmitis. Endogenous endophthalmitis can arise from bacterial seeding in patients with endocarditis, urinary tract infections, catheter associated infections, or injection drug use related bacteremia. Endophthalmitis is an emergency with intravitreal antibiotics and vitrectomy being the main treatment strategies. Systemic antibiotics can be used to treat the underlying infection in endogenous endophthalmitis.[17]

# Vascular conditions

## Cavernous sinus thrombosis

Cavernous sinus thrombosis can occur as a complication of sino-orbital infections, inflammation, hypercoaguable state, or extrinsic compression from a mass or infiltrative lesion. It may be associated with superior ophthalmic vein thrombosis, which can often be an earlier manifestation progressing to cavernous sinus thrombosis (Fig. 8). This can clinically present with orbital pain, proptosis, ophthalmoplegia, and visual disturbances. Diagnosis can be made on contrast enhanced MRI with high-resolution sequences through the orbits/cavernous sinuses and MR or CT venogram. Treatment includes, antibiotics for septic thrombophlebitis, anticoagulation therapy, and surgical intervention for associated abscess and sinusitis.[18]

## Cavernous carotid fistula

Cavernous carotid fistula (CCF) are a result of abnormal communication between cavernous sinus and carotid artery (Fig. 9). The CCF can be classified as low-flow or high-flow based on hemodynamic behavior or based on type of shunting as direct or indirect.

Barrow classification of CCFs defines 4 types, Type A CCFs are the most common with a direct fistula between the internal carotid artery and the cavernous sinus. Type B, C, and D comprise the indirect fistula. Type B CCF has a shunt between meningeal branches of the ICA and cavernous sinus. Type C CCF has a shunt between meningeal branches of the ECA and

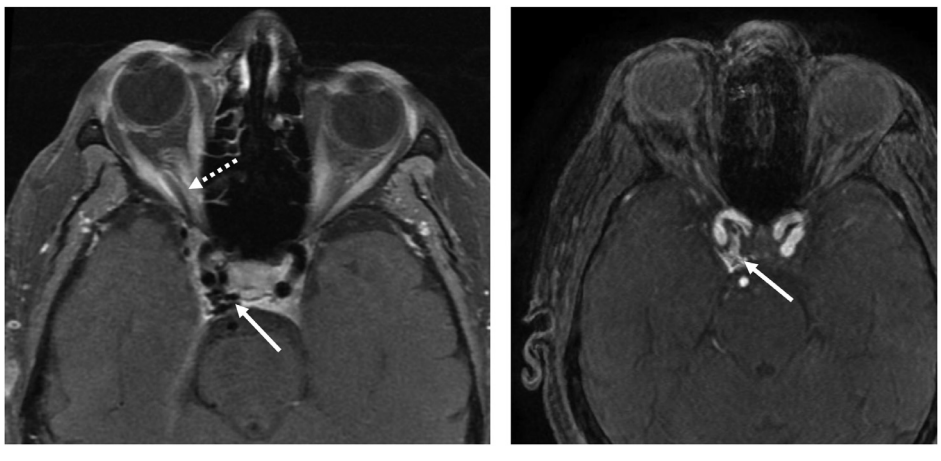


**Figure 9** Cavernous carotid fistula: Axial contrast enhanced T1w fat saturation sequences shows abnormal flow voids in the right cavernous sinus, (white solid arrow, A) and flow related signal within right cavernous sinus on time of flight MRA (white solid arrow, B). Note prominent right superior ophthalmic vein, (dotted arrow, A).

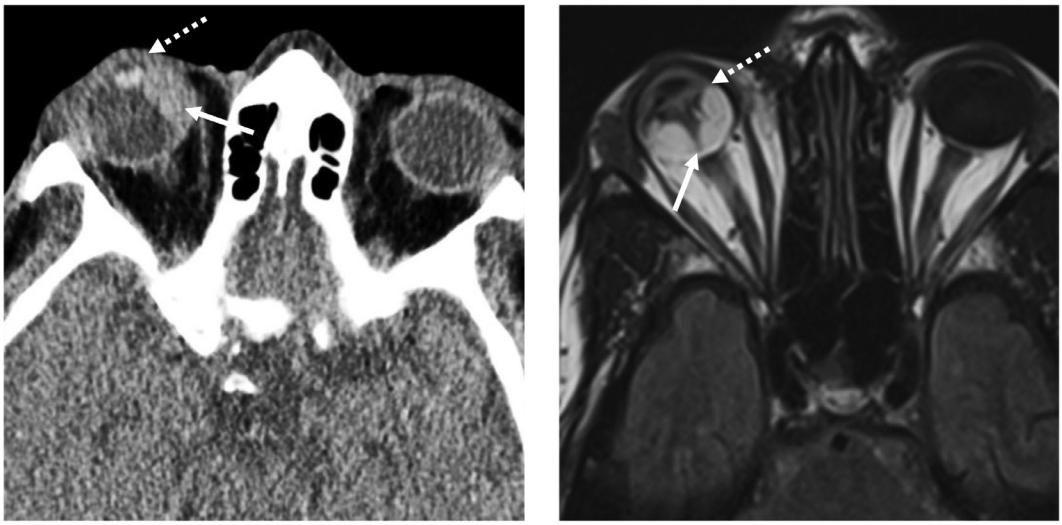

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**Figure 10** Choroidal detachment: Axial noncontrast CT of the orbit shows right medial globe lentiform high attenuation sparing the posterior globe and optic disc, (white arrow, A). Note extension of the detachment anterior to the ora serrata, (dotted arrow, A). Retinal detachment: Axial T2-FLAIR sequence showing V-shaped retinal detachment with apex at optic disc, (white arrow, B) and anteriorly bounded by ora serrata, (dotted arrow, B).

cavernous sinus. Type D CCF, has a shunt between meningeal branches of the ICA, ECA, and cavernous sinus. CCF can be a result of trauma, aneurysm, underlying vasculopathy, or connective tissue disorders. Clinically, the patients can present with orbital bruit, pulsatile proptosis, pain, ophthalmoplegia, and visual loss. High-risk CCFs are treated with endovascular embolization.[18,19]

# Miscellaneous

## Choroidal and retinal detachments

Choroidal detachment is characterized by lentiform morphology with detached surface sparing the optic disc and posterior third of the globe (Fig. 10A). Anteriorly it extends anterior across the ora serrata (junction of retina and ciliary body) to the ciliary body. Choroidal detachment can result from accumulation of fluid and blood in the subchoroidal space. The accumulation of fluid in the sub-choroidal space is also referred to as Uveal Effusion syndrome with underlying inflammatory and hydrostatic etiologies. Treatment entails steroids, surgical decompression, and full thickness sclerotomies.[20]

In contrast to above, the retinal detachment (RD), has a characteristic imaging appearance with a V-shaped morphology due to posterior attachment at the optic disc margin forming the apex of the V and anterior attachment at the ora serrata forming the base of the V (Fig. 10B). RD results from separation of the inner sensory retina from outer pigmented retina with subretinal space fluid or blood accumulation. It can be classified as rhegmatogenous, serous or exudative, and tractional based on etiology. The most common is rhegmatogenous due to a rent or tear in the sensory retina. Serous/exudative RD occurs due to fluid in the subretinal space that can occur as a result of tumors. Tractional RDs, occur to mechanical stress from adhesions and are seen in diabetic retinopathy.[21] Management is with surgical treatments including retinopexy, scleral buckling, and intraocular tamponade.

## Vitreous hemorrhage

Vitreous hemorrhage is one of the causes of acute visual loss and is therefore commonly encountered in the emergency setting (Fig. 11). It can result from diabetic retinopathy, posterior vitreous detachment, (may be associated with retinal tear), and trauma. Other less common causes include, retinal vein occlu-

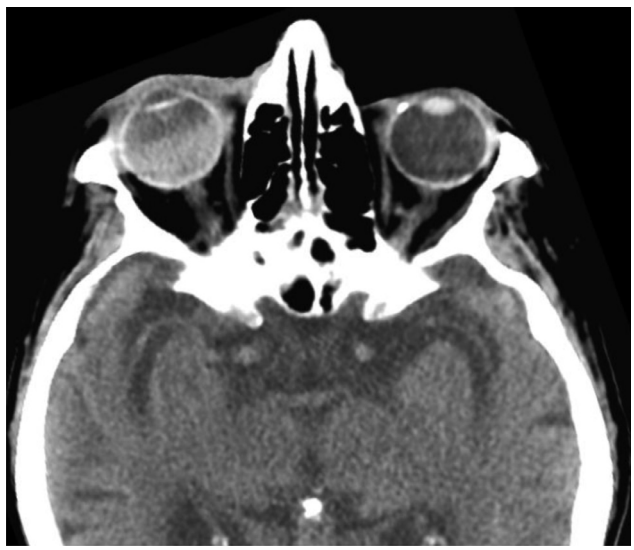

**Figure 11** Axial noncontrast CT of the orbits shows layering vitreous hemorrhage in the right globe.

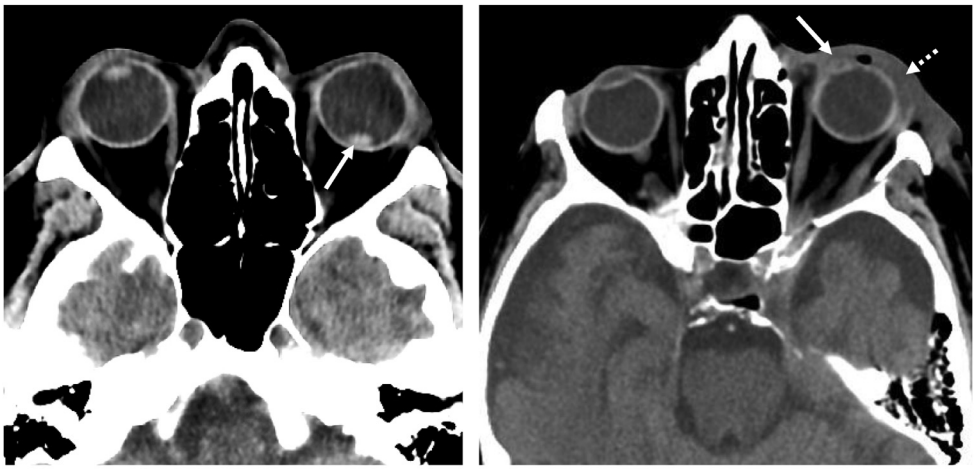

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**Figure 12** (A) Ectopia lentis: Posterior dislocation of the lens is seen in the left globe without history of trauma or other imaging findings to suggest trauma, (white arrow, A). (B) Anterior globe rupture due to corneal perforation: Apparent anterior positioning of left lens with reduced depth of anterior chamber, (white arrow, B) and adjacent fluid, (dotted arrow B). These findings were seen in the setting of infectious keratitis with corneal perforation leading to anterior globe rupture, rather than a true anterior lens subluxation.

sion, proliferative sickle cell retinopathy, neovascular age related macular degeneration, subarachnoid hemorrhage (Terson Syndrome). It commonly presents as sudden onset painless visual loss, with history of underlying risk factors such as diabetes, hypertension, prior surgery, or trauma. Management relies primarily on treating the underlying cause, observation for spontaneous vitreous clearing, and vitrectomy.[22]

## Lens dislocation

Lens dislocation or ectopia lentis, refers to dislocation or subluxation of the lens from its normal attachment to the ciliary body. Spontaneous lens dislocation without associated trauma has been seen with connective tissue disorders such as Ehlers Danlos syndrome, marfans syndrome, and homocystinuria.[18] Lack of other trauma related findings and appropriate clinical picture can help accurate diagnosis.

In cases of complete lens detachment from the ciliary body, it is often found displaced posteriorly in the vitreous (Fig. 12A). If the lens is displaced or subluxed anteriorly it may present as loss of volume in anterior chamber, which is difficult to differentiate from anterior chamber globe rupture (Fig. 12B). Additionally, recognition of anterior subluxation of lens is critical to detect secondary angle closure glaucoma.[23] Treatment includes intraocular lens replacement, lensectomy, vitrectomy, and management of underlying and associated disorders.[18]

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