MRI of Orbital Pathology: An Overview for the Ophthalmologist

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Abstract

Meaningful interpretation of orbital pathology on Magnetic Resonance Imaging (MRI) is best done in a systematic approach. This involves differentiating pathology from normal, identifying the appropriate surgical space in which a lesion is situated, relationship to the surrounding structures as well as estimation of its size, signal and enhancement characteristics. This article aims to cover the MRI features of commonly imaged ocular and orbital pathologies.

Key Words: MRI; Orbital Pathology

Introduction

MRI has become the most favored imaging because of the explicit anatomic detail displayed and low radiation. It is imperative that the Ophthalmologists acquire the skill to order appropriate views and interpret an MRI scans. Systematic reading of an MRI has been discussed in a previous article [1]. We present some of the commonly encountered orbital lesions in the present article and discuss the salient features. Correlated with clinical data obtained by history and examination, MRI is of immense help in arriving at the most appropriate diagnosis, planning medical or surgical management including the surgical approach.

Surgical spaces of the orbit

The orbital septum separates the pre and post-septal spaces. Post-septal space (orbit) has four potential surgical spaces: 1) Subperiosteal space situated between bony orbit and periosteum 2) Extraconal space between the bony orbit and the muscle cone 3) Intraconal space behind the globe enclosed within the muscle cone and 4) Tenon’s space enclosed within the Tenon’s capsule around the globe.

Normal measurements

In diagnosing pathology, it is important to compare the appearance and sizes of the structures with the contralateral normal orbit. Measurements provide guidelines and may help in interpretation of conditions with bilateral involvement as in Grave’s ophthalmopathy, bilateral optic neuritis and papilloedema. Normal thickness of the extraocular muscles (in mm): [2] Medial rectus – 4.5 +/- 0.6

Lateral rectus – 4.8 +/- 0.7, Inferior rectus – 4.8 +/- 0.7 Superior rectus – 5 +/- 1.0 (If the muscles on one side are more than 1.4 times that of the other side or an absolute thickness of more than 8 mm are considered abnormal. [3]) Other structures: Superior-ophthalamic vein - 1-2.9mm [4] Optic nerve sheath complex – 3.4 - 5.5 mm [4]

Ocular and orbital pathology

A. Intra-ocular tumors: Primary, Metastatic
B. Inflammatory diseases
C. Infections
D. Vascular lesions
E. Lymphoproliferative and leukemic disorders
F. Optic nerve lesions
G. Peripheral nerve sheath tumors
H. Orbital apex and cavernous sinus lesions
I. Diseases affecting the lacrimal glands
J. Developmental anomalies
K. Miscellaneous

Intraocular tumors

Retinoblastoma: The sensitivity of MRI in diagnosing retinoblastoma is less than that of CT because of the difficulty of identifying calcifications on MRI. But, MRI is a better modality for evaluation of extension along the optic nerve, detection of other intracranial masses including pinealoblastoma and in differentiating retinoblastoma from other causes of leukocoria. On MRI, retinoblastoma is hyperintense to vitreous on T1W images, and hypointense on T2W images (Figure 1) [5]. Moderate to marked contrast enhancement can be seen in the mass. Associated retinal detachment is characterized by lack of enhancement. Calcifications may be seen

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as hypointense foci on all sequences. Normally, a contiguous linear enhancement is seen at the optic disc. Interruption of this linear enhancement pattern of the choroiretinal complex at the optic disc suggests prelaminar optic nerve involvement [6].

Figure 1: Retinoblastoma.

T2W axial (Fig A) and fat-suppressed post-gadolinium axial (Fig B) images showing left intraocular hypo-intense mass (thick arrow in A) with heterogeneous enhancement (white arrow heads and thick arrow in B) with extension along the optic nerve (thin arrow A and B).

Retinoblastoma must be differentiated from Coat's disease. The lipoproteinaceous subretinal exudates in Coat's disease appear hypointense on both T1W and T2W MRI and do not enhance. The detached retina in Coat's disease may show enhancement, reflecting the presence of abnormal retinal vessels. This may be seen as linear enhancement between the subretinal lesion and the vitreous.

Choroidal Melanoma: Uveal melanomas are hyperintense on T1W images due to the paramagnetic effect of melanin. They are hypointense on T2W images and show homogeneous enhancement with gadolinium. They are dome shaped with a broad base to the choroid and with destruction of Bruch’s membrane; they can attain a mushroom-shape. There can be associated haemorrhagic subretinal fluid which has variable intensities depending on the age of the haemorrhage [7].

Choroidal Hemangioma: Choroidal hemangiomas are also hyperintense to vitreous on T1W images, but, unlike melanomas, they are isointense with the vitreous on T2W sequences and enhance more strongly and uniformly than melanomas [8].

Uveal Metastases: Uveal metastases can be seen with primary tumours of the lung and breast and very rarely in other primary tumours. They can be bilateral and less well defined masses compared to melanomas. There may be associated retinal detachment (Figure 2). There can be considerable overlap of MRI findings with uveal melanomas. [9]

Inflammatory Diseases of the Extra-Ocular Muscles (EOM) and orbit

Thyroid ophthalmopathy and Idiopathic Orbital Inflammatory Syndrome (IOIS) are the common inflammatory affections of the EOM. Other differentials include 1) specific myositis in conditions like sarcoidosis, SLE, Crohns disease, scleroderma 2) neoplastic conditions like lymphoma, metastases, primary malignancy, leukemic deposits (chloroma) 3) Vascular causes like AV fistulas / AVM, hematoma and 4) infections: extension of infection from adjacent sinuses, cysticercosis, abscesses

Figure 2: Uveal Metastasis in a 38 year old male with carcinoma lung; (A) T2 axial image showing hypointense choroidal lesion (arrowhead) and retinal detachment (arrow). (B) Fat-suppressed T1W post Gadolinium images showing intense enhancement of the lesion (arrowhead) and nonenhancing subretinal collection (arrow).

Thyroid Ophthalmopathy: Proptosis with bilateral enlargement of the EOM can be seen. To measure proptosis, interzygomatic line is drawn between the anterior margins of the zygomatic process at the level of lens (Figure 3). Perpendicular distance between this line and the anterior corneal surface exceeding 21mm or an asymmetry greater than 2mm between the two sides indicates proptosis [10].

Figure 3: Measurement of proptosis on T2W axial image in a patient with right caroticocavernous fistula. Perpendicular distance (black lines) from anterior mid corneal surface to interzygomatic line (white line) measuring 25mm on the right side indicating proptosis. Note the normal measurement on the left side.

The EOM can be enlarged bilaterally. They appear isointense on T1W images and mildly hyperintense on T2W/STIR images in the acute phase. The muscle bellies are involved with sparing of the tendinous insertions (Figure 4). Inferior rectus is most commonly involved, followed by medial rectus, superior rectus, lateral rectus and then the obliques (pneumonic – I'M SLOW). Good correlation has been shown between clinical activity scores and maximum T2 relaxation time which could also be used to assess response to therapy [11]. The retrobulbar fat may increase in volume, but, usually there is no stranding in the fat (Figure 4). Crowding of the muscles at the orbital apex can compress the optic nerve [12] and the superior ophthalmic vein.

IOIS–Idiopathic Orbital Inflammatory Syndrome (Orbital Pseudotumour/ Myositis): IOIS can involve different anatomical parts (muscle, orbital fat, orbital apex and lacrimal gland) in the orbit in isolation or in various combinations. Periscleritis and perineuritis can also occur. The tendons enlarge along with the
Figure 4: Thyroid Ophthalmopathy.

(A) T2W axial image reveals enlargement of medial recti (MR) with sparing of tendinous insertion (arrows)-the 'coke bottle ‘appearance.

(B) Fat-suppressed post- gadolinium coronal image showing bilaterally symmetric enlargement of all extraocular muscles except lateral recti.

muscle bundles and lead to a tubular configuration (Figure 5). There is also stranding or infiltrates in the adjacent fat. These findings are in contrast to thyroid ophthalmopathy, in which the tendons are spared and fat stranding is absent. The enlarged muscles may regress in size after administration of steroids.

Figure 5: Orbital pseudotumor

Fat-suppressed post-gadolinium T1W axial (Fig A) and coronal (Fig B) images showing diffuse thickening of left superior rectus muscle (arrow). Note that the tendinous insertion is involved (arrowhead).

Idiopathic orbital inflammatory infiltrate on MRI demonstrates low signal intensity on T1W and frequently on T2W images. The signal characteristics depend on the degree of fibrosis, with the sclerosing variety being more hypointense on the T2W images (Figure 6). Diffusion Weighted Imaging (DWI) with apparent diffusion co-efficients (ADC) have been suggested in differentiating orbital pseudotumor, orbital lymphoma and orbital cellulitis. Brightness on DWI (lower ADC) is maximum in lymphoma with decreasing intensity in IOIS and orbital cellulitis. [14].

Orbital Cysticercosis: Extraocular muscle is the most common site of involvement in the orbit. The cyst contents are isointense to CSF on T1 (dark) and T2 (bright) images (Figure 7). On T2W images, scolex can be seen as an intracystic hypointense focus. Ring enhancement can be seen in dying cysts with associated inflammation. Cyst may get calcified in later stages, which is better appreciated on CT [15]. A careful search should be made for cysticercosis in brain and facial muscle.

Figure 6: Idiopathic sclerosing orbital inflammation in a 23- year-old male with no perception of light right eye.

(A) T2W axial image showing ill defined hypointense tissue replacing extracanal (arrowheads) and intraconal (arrow) fat.

(B) T1W fat- suppressed post-gadolinium coronal image shows diffuse enhancement of the intraconal (arrow) and extracanal soft tissue. Extraocular muscles are bulky and heterogeneously enhancing (arrowheads).

Figure 7: Orbital cysticercosis

(A) T2Wcoronal image showing bilobed cystic lesion (arrowheads) in the left inferior rectus muscle. The medial lesion shows a T2 hypointense scolex (arrow).

(B) Fat-suppressed post-contrast coronal T1W image demonstrates intense enhancement of cyst wall (arrowheads).

Infections

Orbital Cellulitis And Abscess: Contiguous infection from the para-nasal sinuses is the most common source of cellulitis and
subperiosteal abscess. Findings of sinusitis (hyperintesity on T2W and FLAIR images in the sinuses) suggest the primary infective focus. Role of imaging is to demonstrate the extent of disease and its complications. MRI with fat-suppressed and post-gadolinium sequences is highly sensitive in detecting early inflammatory changes and in assessing intracranial and cavernous sinus involvement. Imaging findings and staging [16] of different extents of involvement is given in table 1.

**Table 1: Staging of orbital cellulitis:**

<table>
<thead>
<tr>
<th>Extent</th>
<th>MRI Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preseptal cellulitis</td>
<td>Soft tissue thickening of the eyelids, isointense to muscle on T1W and hyperintense on T2W, postseptal tissues appear normal (Figure 8).</td>
</tr>
<tr>
<td>Inflammatory edema</td>
<td>Enhancement of edematous orbital fat and adjacent tissues, maximum in the extraconal fat near affected sinus (Figure 8).</td>
</tr>
<tr>
<td>Subperiosteal abscess</td>
<td>Peripherally enhancing T1 hypointense, T2 hyperintense extraconal collection. Adjacent rectus muscle is displaced inward (figure 9). Restricted diffusion on DWI sequence is diagnostic and helps in identification in a non-contrast study</td>
</tr>
<tr>
<td>Orbital abscess</td>
<td>True intraconal orbital abscess, proptosis, extraocular muscle enlargement.</td>
</tr>
<tr>
<td>Cavernous sinus thrombosis</td>
<td>SOV* thrombosis is seen as enlargement and altered signal compared to opposite side. Enlargement and abnormal hyperintense signal of cavernous sinus (Figure 10), dilated SOV, thickened extraocular muscles would suggest cavernous sinus thrombosis.</td>
</tr>
</tbody>
</table>

* SOV: Superior Ophthalmic Vein

Figure 8: Orbital cellulitis in a 19-year-old male with pain and restricted eye movements for 3 days.
Fat suppressed post-gadolinium T1W Axial (Fig A) and coronal (Fig B) images showing pacification and enhancement of left ethmoid sinus with extraconal extension (long arrow), medial rectus thickening (arrowhead) and preseptal edema (double arrows).

Figure 9: Sub-periosteal abscess.
(A) T2W axial images showing opacification of right ethmoid cells (arrow) with orbital subperiosteal abscess (*) which displaces the medial rectus (arrowheads).
(B) T1W fat-suppressed, post-contrast images showing rim enhancing collection in the medial orbit in contiguity with ethmoidal abscess.

Figure 10: Cavernous sinus involvement in 60-yr-old diabetic with multiple cranial nerve palsies and loss of vision in the right eye - rhino-orbito-cerebral mucormycosis.
Fat-suppressed post-gadolinium coronal (Fig A) and axial images (Fig B) showing thickening and enhancement of right cavernous sinus (thick arrow) and right orbital apex. Thickening of tentorium cerebelli is also seen (double arrows) Note opacification and enhancement of sphenoid sinus (thin long arrow) suggesting sinusitis.

**Vascular lesions**

**Cavernous Haemangioma:** They are solitary lesions, most commonly seen in the lateral intraconal space. They are well circumscribed due to distinct fibrous pseudocapsule. On MRI, they are usually isointense to muscle on T1-W images (Figure 11) and hyperintense on T2-W images (Figure 12) and have characteristic heterogeneous enhancement on delayed images (Figure 13).

Figure 11: Cavernous haemangioma
Well defined T2W hyperintense (arrowheads in A) and T1W isointense (arrow in B) intraconal lesion, displacing the optic nerve inferomedially (arrowhead in B). Immediate post contrast image shows small area of focal enhancement (C) and delayed image shows more widespread heterogeneous enhancement (D).
perintense on T2-W images. Progressive enhancement is seen on delayed dynamic contrast enhanced images [17] (where multiple rapid sequential images of the orbits are obtained after injecting contrast).

The other common intraconal lesion which shows similar signal characteristics is the schwannoma. Dynamic contrast-enhanced MRI can help in differentiating the two as in the early phase, hemangiomas start enhancement from one point or portion whereas schwannomas start enhancement from a wide area [18].

**Orbital Varices**: These consist of markedly dilated valveless orbital veins, with most channels communicating with the venous system [19]. Imaging performed with maneuvers that increase venous pressure can show distension of the varices. On MRI, varices appear smooth contoured channels or venous spaces or they may appear as a cluster of vessels. They have hypo- to hyperintense signal on T1W images, hyperintense signal on T2W images, and show intense enhancement with gadolinium (Figure 12). [17]

**Veno-Lymphatic Malformations**: They are slow-flow vascular malformations which are unencapsulated, diffuse and multicompartamental, often including both intraconal and extraconal components and insinuating between normal orbital structures (Figure 13). [20] Intracystic fluid-fluid levels produced by hemorrhages of different ages is a typical finding in the lymphatic component. Variable signal intensity is seen on MRI depending on the type of cyst fluid, presence and age of haemorrhages. On T1W images, lymphatic or proteinaceous fluid and blood products appear hyperintense. Post-contrast, the intervening septae of lymphatic component can show enhancement. Venous components show bright enhancement and phleboliths (Figure 14). Phleboliths are seen as hypointense foci on all sequences. They do not communicate with the normal veins and, unlike varices, do not distend with maneuvers that increase the venous pressure [21].

**Arterio-Venous Malformations**: These are high-flow vascular malformations and hence seen as tangle of abnormal tubular flow voids (Figure 15) or hypointense signal voids on MRI (better seen on T2W images). Magnetic Resonance Angiography (MRA) can demonstrate the hypertrophied and tortuous vessels.

**Carotico-Cavernous Fistula (CCF)**: CCF is an arterio-venous fistula, resulting from an abnormal communication between the arterial and venous systems of cavernous sinus usually following trauma. MRI findings include cavernous sinus enlargement, abnormal flow voids within the cavernous sinus and dilatation of

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**Figure 12: Orbital Varices.**

(A) STIR images showing tortuous vascular channels (arrow) along the superolateral extraconal space.

(B) Intense enhancement (arrows) is noted on post-contrast images.

(C) Doppler image with Valsalva maneuver, showing distension and homogenous colour uptake (arrow).

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**Figure 13: Venolymphatic malformation.**

(A) T2W axial image showing multicompartamental heterogeneously hyperintense lesion (arrows) with septations. Note involvement of eyelid (arrowhead).

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**Figure 14: Venous Malformation.**

(A) T2W axial image showing a multiloculated hyperintense lesion with fluid-fluid levels (arrow).

(B) T2W sagittal image showing focal hypointense phleboliths (thin arrows).

(C) Coronal scan showing the calcified phlebolith (arrow).
superior ophthalmic vein and other draining veins (Figure 16). Post-contrast MRI and CT are complementary in the diagnosis of dural CCFs. However, for confirmation, proper tying and planning management, selective cerebral arteriography is required [22]. MRA (Figure 17) can demonstrate the enlarged draining veins due to arterialized flow.

Figure 16: Caroticocavernous fistula
T2W axial image (Fig A) showing enlarged right cavernous sinus with tortuous flow voids (double arrows). T2W axial (Fig 16B) and T2W coronal (Fig C) images show dilated right superior ophthalmic vein (curved arrow).

Figure 17: Magnetic Resonance Angiography.
MRA MIP images (Figures A and B) showing dilated and tortuous right SOV (thick arrow). Thin arrows show the normal ICAs on both sides. Note that no other venous structures are visualized as MRA depicts only high velocity flow.

Lymphoproliferative and leukemic disorders
Although orbital involvement in lymphoma may not be associated with systemic disease, leukemic involvement is always associated with systemic disease. Leukemic infiltration can involve the globe (uvea, choroid, and retina), optic nerve or orbital soft tissue including fat, lacrimal gland and extraocular muscles (Figure 18). MRI can show these as localized or diffuse enhancing masses which are isointense to muscle on T1W images and mildly hyperintense on T2W images [23]. Lymphoma can involve any part of the orbit. However, lacrimal gland, anterior and superior orbit, conjunctiva (salmon patch appearance) and lids are commonly involved. The lesions may be diffuse and poorly defined or well-circumscribed and isolated extraconal and/or intracranial masses. Infiltration of the extraocular muscles with diffuse enlargement can occur. A feature of lymphoma is its tendency to mould itself around orbital structures, such as the globe, optic nerve or the orbital wall without causing indentation or erosion. Aggressive, malignant lymphoma, however, can demonstrate osseous destruction. The lesions are usually homogenous and isointense to extraocular muscles on T1W and T2W images and show moderate enhancement (Figure 19).

Figure 18: Leukemic deposits in a 27-year-old male with acute leukemia (A) T2 coronal image showing bilateral hypointense lacrimal gland deposits (arrows) and diffuse enlargement of right inferior (arrow head) and left medial rectus (double arrows). Fat-suppressed post-gadolinium T1W coronal image (B) showing minimal enhancing deposits (arrowhead and arrows) and normal enhancement of unaffected right medial rectus and left inferior rectus (open arrows)

Figure 19: Lymphoma.
T2W axial image (A) showing diffuse enlargement of the left lateral rectus muscle (thick arrow), which is iso to hypointense and ‘moulds’ itself along the globe (thin arrow). T1W fat-suppressed, post-contrast axial image (B) in another patient with diffuse large B cell lymphoma, showing focal enlargement and enhancement of left prechiasmatic optic nerve (arrow in B)
MRI cannot reliably differentiate lymphoma from pseudotumor of the orbit, often requiring biopsy correlation.

**Optic nerve lesions**

**Optic Nerve Sheath Meningiomas (Onsm):** Meningiomas surround the optic nerve, and thus the caliber of the nerve itself is attenuated within the surrounding tumor (Figure 20). This is in contrast to optic nerve gliomas, where the nerve itself appears expanded [24]. Three distinct growth patterns include tubular enlargement, fusiform or spindle shaped pattern, and globular exophytic growth patterns [25]. Meningiomas may be iso to hypointense on T1-weighted images and isointense or even hypointense on T2-weighted images. Moderate to marked enhancement is seen on post-gadolinium images with central nonenhancing nerve, producing the ‘tram-track’ sign on axial images and ’doughnut’ configuration on coronal images [26]. The tram-track sign is non-specific and can be seen in pseudotumor, periocular neuritis, sarcoidosis, leukemia, lymphoma, metastases, periocular hemorrhage, and Erdheim-Chester disease. Meningiomas can calcify and sensitivity of MRI in detecting the calcification is lower than CT. Hyperostosis or thickening and sclerosis in surrounding bones and erosions are also better appreciated on CT.

![Figure 20: Optic nerve sheath meningioma](image)

(A) T1W axial image showing iso to minimally hyperintense fusiform optic nerve sheath mass (arrowheads). Optic nerve is seen through the lesion.

(B) Post-contrast T1W image shows moderate homogenous enhancement (arrowheads).

**Optic Nerve Glioma:** Optic nerve gliomas (figure 21) cause diffuse thickening of the nerve and are iso to hypointense on T1W images and hyperintense on T2W images [27]. Gliomas enhance variably and complete lack of enhancement can also occur. Although they are low grade tumours, they can also enhance brightly due to increased vascularity. MRI can depict the extent of involvement clearly as even the intracanalicular and intracranial portions of the optic nerve, optic chiasm are well imaged.

![Figure 21: Optic nerve glioma in a 12-year-old child with neurofibromatosis.](image)

T2W coronal (Fig A), T2W axial (Fig B) and fat-suppressed post-gadolinium axial image (Fig C) showing fusiform enlargement with heterogeneous enhancement of both optic nerves and chiasm (thick arrows). Retrobulbar portion of the right optic nerve shows involvement (single thin arrow) while retrobulbar portion of the left optic nerve is uninvolved (thin double arrows).

**Optic Neuritis:** In most cases, if rigorous clinical criteria are applied, MRI is not required to diagnose and distinguish typical optic neuritis from other common optic neuropathies [28]. The goal of imaging is look for demyelinating plaques in the brain which may be seen as round, ovoid or flame shaped white matter hyperintensities, best seen on Flair images.

In the acute stage, the affected optic nerve may be thickened, hyperintense on STIR images and show enhancement on fat-suppressed post-gadolinium images (figure 22). Enhancement indicates a blood–optic nerve barrier breakdown.

![Figure 22: Optic neuritis.](image)

STIR coronal images (Fig A) and fat-suppressed post-gadolinium axial images (Fig B) showing thickened, hyperintense enhancing right optic nerve (single arrow). Compare with normal left optic nerve which does not enhance and shows the normal perioptic CSF space (double arrows).

**OPTIC PERINEURITIS:** Fat-suppressed plain MRI images, especially the STIR images can show thickening of the optic nerve sheath (figure 23) and irregular linear strands in the intraconal fat, indicating inflammation or edema. Fat-suppressed gadolinium enhanced MRI typically shows characteristic enhancement of the optic nerve sheath (“tramtrack” on axial views and “doughnut” on coronal views). In most cases, the optic nerve does not enhance [29]. CT does not provide sufficient resolution to distinguish perineural enhancement from the intraneural enhancement seen in optic neuritis.

![Figure 23: Optic perineuritis.](image)

Fat-suppressed post-gadolinium axial image showing ‘tram track’ enhancement of the perineural sheath (arrows) (perineuritis). Stranding and enhancement of retrobulbar fat (*), enhancement of medial rectus muscle (arrow head) and preseptal soft tissue (thin double arrows) are seen indicating multicompartment involvement in idiopathic orbital inflammation.
Peripheral nerve sheath tumors

**ORBITAL SCHWANNOMA:** They are seen as well defined extraconal (Figure 24) or less commonly intraconal lesions arising from the sensory nerves of the orbit. They are isointense to muscle on T1W and mildly hyperintense on T2W images. Schwanommas show an early wide area of enhancement unlike haemangiomas which start enhancement in one portion [18].

Figure 24: Orbital Schwannoma.

T2W coronal (Fig A) and axial (Fig B) images show a large T2W hyperintense supero-medial extraconal lesion (arrow) displacing the medial rectus (thin arrow) and optic nerve (curved arrow). Post- gadolinium axial images (Fig C) show moderate heterogeneous enhancement.

**Orbital apex and cavernous sinus lesions**

The orbital apex and cavernous sinus can be affected by contiguous spread of pathology from the adjacent structures/ sinuses. The causes can be inflammatory, iatrogenic/traumatic, infectious, vascular or neoplastic.

**Tolosa-Hunt Syndrome:** Tolosa-Hunt syndrome is recurrent painful ophthalmoplegia caused by non-specific inflammation of the anterior portion of the cavernous sinus or superior orbital fissure (SOF) (Figure 25), which is responsive to steroid therapy. In two thirds of the patients conventional CT scan can be normal whereas MRI shows the abnormality [30]. Enhancing soft tissue mass isointense with gray matter on T1W and T2W images is seen in the cavernous sinus with convex bulging of the contour of the lateral border of the involved sinus [31]. Carotid artery involvement and extension to the orbit/ SOF can also be seen. These changes show resolution with steroid administration. These features are not specific and can be seen in granulomatous inflammation like tuberculosis and sarcoidosis, meningioma, lymphoma and hence is a diagnosis of exclusion.

**Diseases affecting the lacrimal glands**

Dacryoadenitis, pseudotumor, lymphoma and pleomorphic adenoma are some of the more common pathologies encountered.

Figure 25: Tolosa-Hunt Syndrome in a patient with painful ophthalmoplegia.

Fat-suppressed post-contrast T1W axial (Fig A) and coronal (Fig B) images show abnormal enhancement in right orbital apex (arrow) and cavernous sinus (arrowhead). Compare with normal left side (thin arrow).

Lacrimal gland may also be affected in conditions like Sjogren’s, tuberculosis and sarcoidosis. The superotemporal location of the lesion in the orbit indicates lacrimal gland involvement (figure 26). Benign tumors like pleomorphic adenoma are well defined and do not show bony erosion. Bone erosion indicate aggressive tumor. Imaging is useful in determining the extent and characteristics of the lesion but clinical features and biopsy form the mainstay of diagnosis and management.

Figure 26: Lacrimal gland lesion.

Post-contrast T1W fat-suppressed axial (Fig A) and coronal (Fig B) images showing enlarged, homogeneously enhancing right lacrimal gland (double arrows). Compare with the normal left lacrimal gland (arrow). Biopsy revealed granulomatous inflammation.

**Developmental anomalies**

**Dermoid Cysts:** Presence of cyst with fat intensity and calcification favour diagnosis of dermoid cyst. Fat intensity is bright on T1W and intermediate to bright on T2W images and gets suppressed on fat saturated images (figure 27). Associated bony erosion is better picked up on CT scan but MRI is more useful in determining if intracranial communication is present and determining its extent.

Figure 27: Dermoid cyst

Well defined ovoid lesion at the left medial canthal region, which is hyperintense on T1W (Fig A) and T2W (Fig B) images with complete suppression of the signal on fat saturated image (C). There is no intracranial extension.
Cephalocele: Orbital cephalocele (Figure 28) is a rare congenital abnormality in which the meninges, CSF and brain herniate through a bone defect or through a natural anatomic opening. Microphthalmos, colobomas and anophthalmos are associated ocular anomalies. MR imaging is the study of choice to illustrate cephalocele; CT scanning in the sagittal and coronal plane identifies the bony defect.

Figure 28: Orbital cephalocele.

T2W coronal image (Fig A) shows herniation of the frontal lobe and meninges into the medial aspect of right orbit (arrow in A). Coronal reformed CT image (Fig B) shows the bony defect in fovea ethmoidalis (arrow in B)

K. Miscellaneous

Orbital Metastases: Metastasis can occur to the orbit (muscle, bone, fat) and adnexal structures.

Breast, lung, prostate, malignant melanomas in adults and sarcomas and embryonal tumors in children are the important source of metastases. MRI features are nonspecific.

Bony Lesions: Bony lesions like osteoclastoma and Ewing’s sarcoma are better evaluated by CT.

Foreign Body: Wooden foreign body can be missed on CT as it can be isodense to orbital fat or muscle. MRI is the imaging modality of choice when a wooden foreign body is suspected and metallic foreign bodies have been excluded on CT. Depending on the water content, the wooden foreign body may appear hypo to hyperintense [32] with surrounding inflammation and granulation tissue seen as soft tissue thickening and fat stranding (Figure 29).

Conclusion

Imaging in orbital pathology is indispensable. The choice of appropriate MR imaging sequences and the need for contrast studies, dynamic imaging is decided by the radiologist based on the clinical impression by the ophthalmologist. Subsequently the ophthalmologist can best manage the patient if he/she can interpret the MRI looking for details relevant for management including the surgical approach. We hope that after reading this article, the ophthalmologist when encountered by a patient with orbital pathology, feels more motivated to look at the patient’s MRI, meaningfully interpret it and plan appropriate management. Discussion with the radiologist along with the clinical information would be ideal in arriving at the most probable differential and management.

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References


