

# Magnetic resonance imaging of the orbit, Part 1: Basic principles and radiological approach

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Magnetic resonance imaging (MRI) is an eloquent, noninvasive, cross-sectional imaging modality that offers superior tissue characterization of orbital pathologies. The ophthalmologist needs to be aware of the advantages of MRI and its step-wise interpretation in liaison with a radiologist to optimize patient outcomes. In this review, we discuss the basic principles of MRI, some of the commonly used sequences and protocols, the anatomy of the orbit on MRI, and an approach to radiological interpretation.

**Key words:** Eye, magnetic resonance imaging, MRI, orbit, sequence, tumor

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Modern medicine has been revolutionized by the ability to noninvasively identify, diagnose, and follow up lesions with high precision. It is essential for the ophthalmologist to harness the advances in radiology so as to use the appropriate modality and be capable of competent image interpretation to optimize patient outcomes. In this review, we discuss the basic principles of magnetic resonance imaging (MRI), some of the commonly used sequences and protocols, and the technique of radiological interpretation.

## Basic Physics of MRI

MRI is an elegant technology, and its clinical application is better appreciated by having at least a basic understanding of the basics of its underlying physics. The acquisition of an MR image can be simplified into three processes based on its very name: *magnetic* (the inherent electromagnetic activity of atomic nuclei), *resonance* (the behavior of these nuclei in an external magnetic field and the method of manipulating them), and *imaging* (manipulation of these nuclei resulting in an observable signal to give every point in the sampled tissue a unique signal and location).<sup>[1]</sup> The near-ubiquitous nature of hydrogen atoms in biological molecules makes it ideal for clinical MR imaging.<sup>[1,2]</sup>

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The nucleus of the hydrogen atom (<sup>1</sup>H) is essentially a single spinning proton. This spin gives it a magnetic field, in essence, making it a small bar magnet with a north and south pole. In the native state, tissues have no net magnetization since the <sup>1</sup>H nuclei axes are randomly aligned in orientation resulting in the cancellation of polarities. However, when an external magnetic field (termed B<sub>0</sub>) is applied, the magnetic axes of the nuclei align with the magnetic axis of B<sub>0</sub>. Just over half of them align in parallel with the north-south axis of B<sub>0</sub> while just under half align anti-parallel to it since the latter is a higher energy state. This results in the net magnetization of the tissues. This newly induced magnetic field is the key underlying basis of MRI. In order to get a detectable magnetic signal, the inducing external magnetic field needs to be extremely strong, usually anywhere between 1.5 and 3 tesla (T) for clinical machines (the earth's magnetic field is 25–65 microtesla by comparison). Superconducting magnets maintained at extremely low temperatures (-270°C) generate these required higher field strengths.

In order to identify the location and type of tissue that lies in the magnetized region/zone/part, the field is manipulated using pulses of radiofrequency (RF) waves. These are non-ionizing electromagnetic waves, similar to light. When the RF pulse is applied, it excites the spinning protons and causes them to flip by a certain angle. This can be done only if the RF frequency is in *resonance* with the spinning frequency of the protons.<sup>[3]</sup> The

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resonant frequency of the hydrogen proton in a molecule is influenced by the neighboring atoms linked to it. Hence, the

hydrogen atom in a fat molecule resonates differently than the same atom in a water molecule due to changes in its milieu. This forms the basis for the superior soft-tissue differentiation on MRI.

**Table 1: MRI: Advantages and Disadvantages**

**Advantages**

- Improved lesion detection, delineation, differentiation from normal tissues
- Improved lesion characterization
- No ionizing radiation
  - Can be performed in pregnancy (2nd trimester onwards) and young children
  - More repeatability

**Disadvantages**

- Longer scan times
- More expensive
- Machine construction may induce claustrophobia
- Noisier due to RF pulse generation

**Table 2: MRI Hardware**

**Magnetic Strength: 1.5 vs 3T:**

- 1.5T provides adequate diagnostic quality images
- 3T affords a faster scan and a higher resolution

**Coils:**

- RF pulses are applied from the magnetic bore within which the patient lies. A coil is akin to a conformable antenna that receives the signal back from the tissues. For the orbit, two types of coils are available:
  - Head coil: most commonly used for general cranial imaging, usually adequate for orbital lesions.
  - Surface coils: pad-like structure, can be placed over the orbits for high-resolution imaging, particularly for globe lesions.

**Precautions:**

- Avoid makeup (eyeliner, etc.) these contain magnetic elements and may distort images
- The individual must close the eyes during the examination with no eye movements (eye movements result in artifacts)

The advantages and disadvantages of MRI, as well as specifics of MRI hardware and scanning, are detailed in Tables 1 and 2.

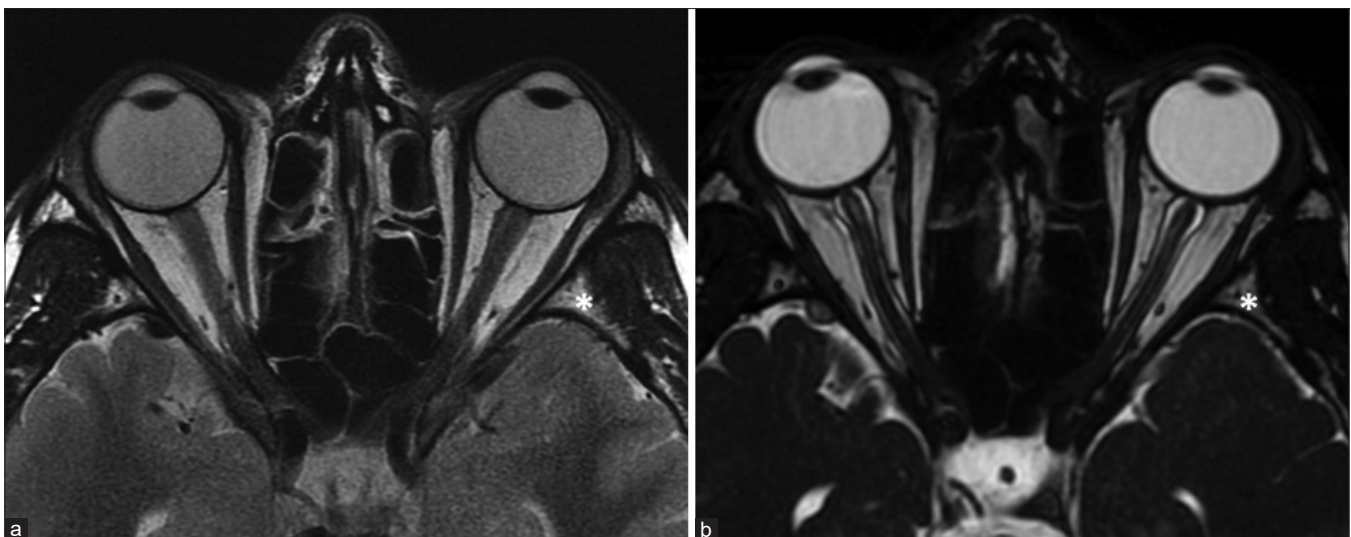
## MRI Sequences

Unlike CT, MRI consists not of one scan but a series of “sequences.” Each MRI sequence is designed to manipulate the magnetization and achieve amplification or suppression of a particular tissue characteristic. Hence, it is said to be “weighted” to the specific tissue characteristic. The appearance of a lesion, specifically its brightness (generally referred to as signal intensity), may vary on each sequence depending on the particular amplified or suppressed features of the tissue. In contradistinction, in CT, the lesion has a fixed appearance or “density” measured in Hounsfield units. A lesion can thus be described as iso-, hypo-, or hyperintense relative to recognizable normal reference structures, including fluid (CSF, vitreous, etc.) or soft tissue structures like extraocular muscles in that particular sequence.

Using varying appearances on each sequence, we can create a reasonably accurate individual signature for each type of lesion that may provide a clue to its nature and diagnosis, if not narrowing the differentials. The following are the commonly used MRI sequences with a mention of their utility.

### T2 weighted images (T2)

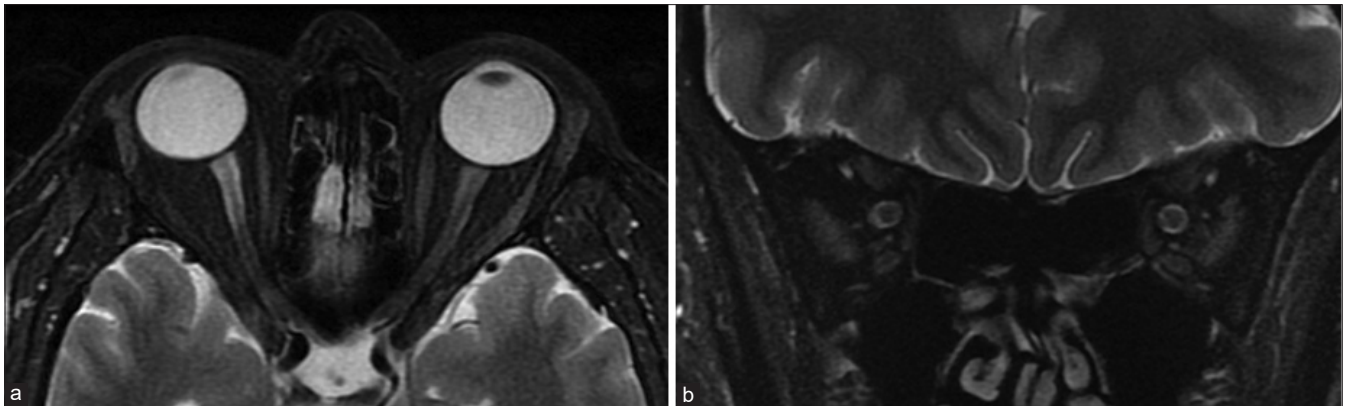
The T2-weighted sequence highlights the tissues with a longer time to realign their magnetic vector (i.e. their individual atomic north-south pole direction) in line with the external magnetic field ( $B_0$ ) after being tipped by the RF pulse (its T2 relaxation time). Water has this prolonged T2 relaxation time and hence stands out on this image as a bright object. T2 is thus weighted towards the character of water, and hence fluids such as aqueous, vitreous, and cerebrospinal fluid (CSF) will therefore appear characteristically hyperintense on this



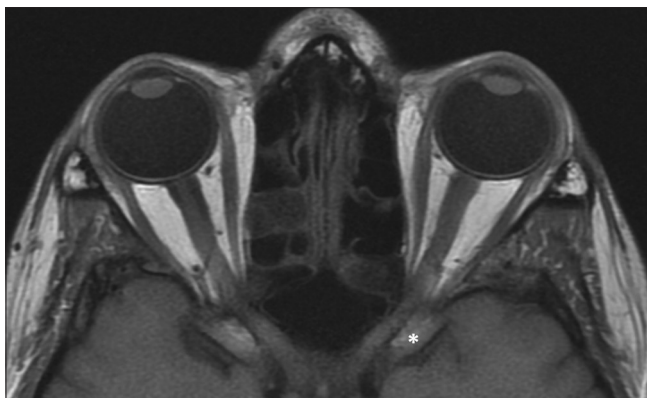
**Figure 1: T2 weighted image.** On T2 (a), fluids such as the aqueous and vitreous humor and CSF appear hyperintense. Fat (orbital, subcutaneous, etc.) and fatty marrow, such as in the greater wing of sphenoid (\*), also appear hyperintense relative to soft tissue. Extraocular and the masticatory muscles are hypointense compared to isointense neural structures such as the optic nerve or brain parenchyma. Volumetric heavily T2 weighted sequences (b) accentuate fluid signal intensity at the cost of intrinsic soft tissue T2 contrast details. Thus, fluid-filled structures such as the eye globe, the perioptic CSF spaces, or any cystic pathology are better seen owing to the high spatial resolution and better fluid-soft tissue contrast differentiation

**Table 3: T2 hypointense lesions and possible underlying mechanisms**

Lesions	Possible Mechanism of T2 hypointensity
Bony or completely calcified lesions	Lack of mobile protons that can be manipulated to obtain the signal
Fibrotic lesions	Increased collagen content with reduced interstitial fluid
Lymphomas and other small blue round cell tumors	Dense cellular lesions with decreased interstitial space
Meningiomas	Either fibrotic or densely cellular lesions
Fungal infections	Hyphae with minerals that create magnetic susceptibility effects causing hypointensity
Hemorrhagic lesions (acute and chronic stages)	Secondary to magnetic susceptibility effects of iron in degrading hemoglobin
Melanomas	Melanin bonding with minerals and propensity to hemorrhage both give it hypointensity
Lesions with proteinaceous content such as mucocoeles or degenerated vitreous	Immobilization of protons by viscous content
Granulomatous diseases like tuberculosis, Tolosa-Hunt syndrome, and sarcoidosis	Dense pyogenic material with inflammatory cells with reduced water content in Tuberculosis. Increased lymphoplasmacytic infiltration and fibrosis in Tolosa-Hunt and sarcoidosis
Intraocular bands, membranes, or detachments	Relatively low water content against the background of the hyperintense water-rich vitreous
Focal T2 hypointensities within a T2 hyperintense lesion	Calcifications in meningiomas, phleboliths in slow-flow venous malformations, areas of hemorrhage



**Figure 2:** T2 weighted fat-saturated image (T2FS). Pathological lesions that are most commonly T2 hyperintense are difficult to differentiate within the similarly T2 hyperintense orbital fat background. T2FS images (a) are basically T2 images with suppression of the bright signal from fat-containing structures such as fatty tissues or bony marrow. This improves the contrast of the T2 hyperintense lesion which now stands out against a dark background. In neuro-ophthalmology, coronal T2FS images (b) are particularly useful for evaluating the optic nerves



**Figure 3:** T1 weighted image (T1). Fluids such as the aqueous and vitreous humor and cerebrospinal fluid appear hypointense. Orbital and subcutaneous fat along with the fatty marrow such as in the anterior clinoid processes (\*) appear hyperintense relative to soft tissue. Muscles are hypointense compared to adjacent optic nerve or brain parenchyma but not as hypointense as fluid filled structures

image [Fig. 1]. However, fat will also appear hyperintense as it has a comparable T2 relaxation time. Normal soft tissues are described as isointense. Despite being fluid, fast-flowing blood within the arteries and larger veins do not remain stationary enough to give off a resonant signal and hence appear as “flow voids” (a distinct type of hypointensity due to blood flow that recognizably conforms to a vessel shape).

Most pathological lesions have altered fluid homeostasis leading to increased water content. The increased fluid content explains why they tend to stand out as T2 hyperintense lesions relative to normal tissue (but not as hyperintense as water itself). Therefore, the T2 images should ideally be the first image analyzed for the *identification* of a lesion. Subsequently, the *characterization* of a lesion based on its water content can be done. When a lesion has low water content, by virtue of many mechanisms, it appears T2 hypointense. This is an important finding that helps narrow the differentials [Table 3] and should prompt analysis of other sequences to ascertain the type of tissue in question.



T2 allows for further characterization of a lesion apart from its water content. *Fluid-fluid levels* in vitreous hemorrhage, venolymphatic malformations, and aneurysmal bone cysts are secondary to the dependent layering of the proteinaceous or hemorrhagic component of fluid within these lesions and appear as an anteroposterior horizontal layering of two different signal intensities. Within lesions, areas of *cystic degeneration or necrosis* appear significantly more hyperintense, almost similar to that of adjacent CSF or aqueous or vitreous humor, and allow further characterization of the lesion. T2 is also advantageous for detecting intra- or perilesional *flow voids* that may indicate a significant vascular network of blood vessels such as in arteriovenous malformations (AVMs) and highly vascular tumors.<sup>[4]</sup>

**Fat suppressed T2 weighted images (T2 FS)**

A problem with T2 is that the orbital fat also appears hyperintense, often causing difficulties in delineating lesions. A solution is to add additional RF pulses to the T2 sequence that selectively delays (saturates) the resonant signal from the fat protons. The end result is the T2 fat-saturated image (shortened colloquially to T2 fat-sat) which has all the properties of the native T2 but with the nulling of the fat signal, i.e., the background orbital and subcutaneous fatty tissue now appears hypointense [Fig. 2].

**Table 4: T1 hyperintense lesions**

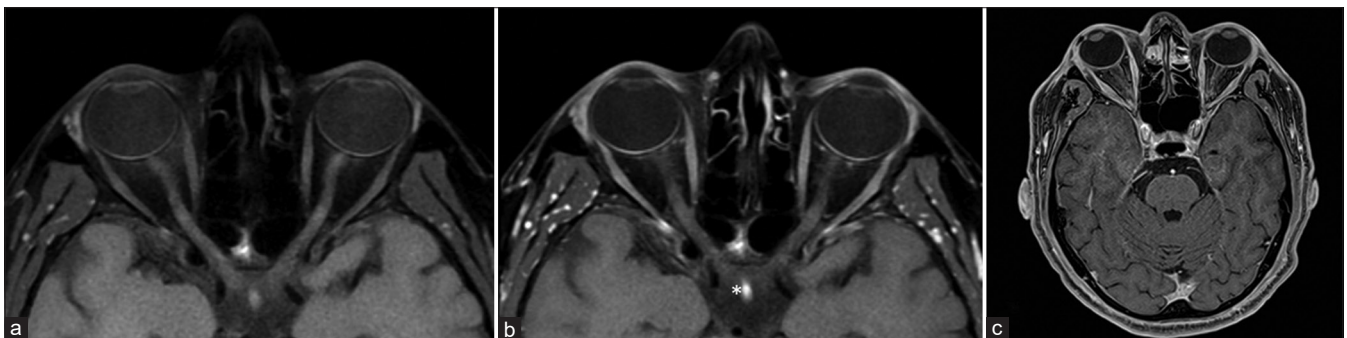
Substance	Tissues/Lesions
<b>Lipid</b>	Orbital fat Oils such as SF6 Lipomas, dermoids
<b>Methemoglobin</b> (subacute stage of degenerating hemoglobin)	Subacute hemorrhage within lesions
<b>Proteinaceous content</b>	Degenerated vitreous, mucocoeles, fluid within cysts
<b>Melanin</b>	Melanoma and other melanomatous lesions
<b>Mineralization</b>	Calcium or manganese with organic content- distinct from calcification/bone with little/no organic content which will appear hypointense

Thus, T2FS allows for improved detection and better delineation of T2 bright (hyperintense) lesions against this dark background. Further, secondary effects of lesions, including interstitial edema in infections and inflammation and infiltration in malignant lesions, appear as hyperintense streaking along the fibrovascular septae of otherwise normal signal fat described as *fat stranding*. In addition, since the normal fatty bone marrow signal is also nulled, *marrow edema or infiltration* is also made apparent as abnormal bone marrow hyperintensity. *Peri or parosteal lesions*, as in Tolosa Hunt syndrome or meningiomas, can also be better delineated from the adjacent bone (which has fatty marrow). In particular, T2FS coronal images are best suited for examining the optic nerves to assess optic nerve hyperintensity in optic neuritis or demonstration of the optic atrophy with the prominence of the perioptic nerve sheath.<sup>[4,5]</sup>

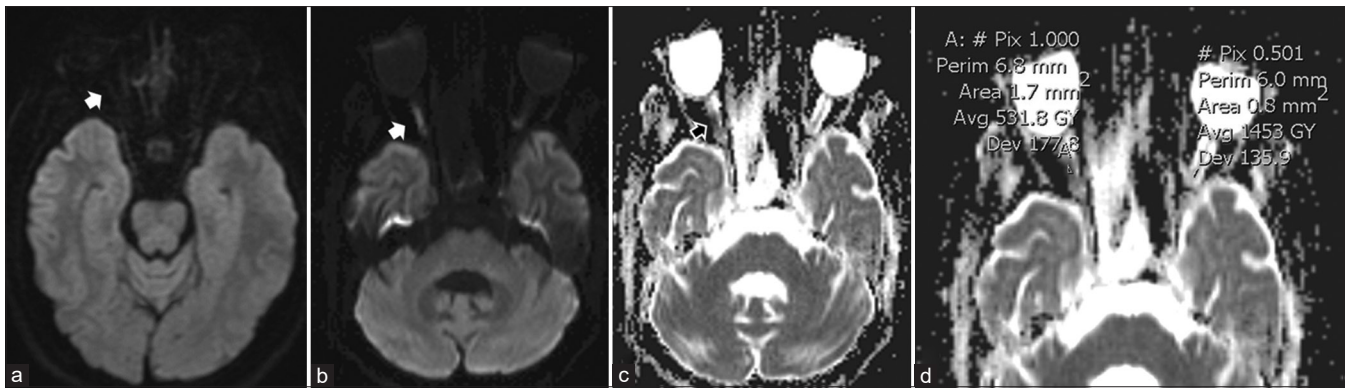
**T1 weighted image (T1)**

The T1 image is based on the relative ease with which the absorbed energy of the RF pulse is given off toward its surroundings, i.e. T1 relaxation time. Soft tissues have an intermediate signal intensity and appear isointense. Fat relaxes or gives up its energy relatively quickly; hence, it has high signal intensity (hyperintense) compared to water, which retains the energy for a longer time and thus appears hypointense. Therefore, most lesions are T1 hypointense since they have higher water content. From this description, it becomes apparent that the T1 is remarkably similar to a CT with a few exceptions: fat is hyperintense (hypodense on CT), and the cortical bone (lacks fatty marrow unlike cancellous bone) is hypointense on T1 (densely hyperdense on CT) [Fig. 3].

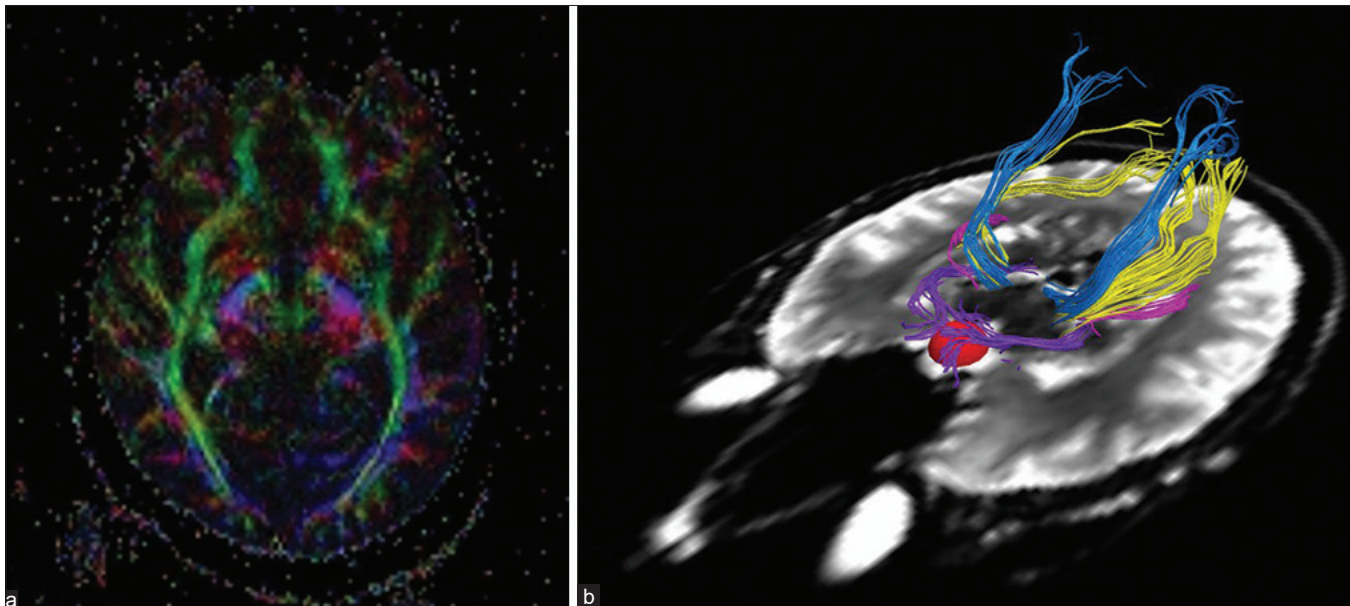
The T1 image serves three primary purposes: analysis of anatomical relations, lesion characterization based on signal intensity, and contrast enhancement. The anatomical details are not only relevant to the diagnosis, but also for surgical planning. The T1 is ideal for evaluating the relationship of a lesion to the surroundings as they may be well outlined by the hyperintense fat.<sup>[6]</sup> After identifying a lesion on T2, the appearance should always be correlated with the signal intensity on T1 for better tissue characterization. Because most lesions have increased water content, T1 hypointense signal



**Figure 4:** T1 weighted fat-saturated image (T1FS). Non-contrast T1FS images (a) are similar to T1 except that the bright signal of fat is suppressed. This sequence is useful for two reasons: to confirm if a T1 hyperintense lesion (such as dermoids or teratomas) contains fat (they should also get suppressed on T1FS) and as a comparison for post-contrast images (b). Note normal enhancing structures such as the EOMs, lacrimal glands, chorioretinal layers, pituitary stalk (\*), etc. For the orbit, most images are acquired in a 2D sequence, i.e., the thickness of each slice is high, which does not allow for reconstruction in any plane. Hence, in most cases, a 3D sequence (c) of orbits and the brain is also obtained in submillimeter thickness that allows for reconstruction in any plane. This is useful to diagnose smaller lesions and to evaluate the relationship to other structures with multiplanar reconstructions



**Figure 5:** Diffusion-weighted image (DWI). DWI is used to look for diffusion restriction of water molecules. Normally, (a) freely diffusing fluids (such as the aqueous/vitreous humor and CSF) appear hypointense and are said to show diffusion facilitation. Cerebral parenchyma and the optic nerves show isointense signal (arrow in a). When Brownian motion of the water molecules is restricted, as in cytotoxic edema, hypercellular tumors, or thick pus in abscesses, these areas appear bright and are said to be diffusion restricted. In this particular case (b-d), the right optic nerve (arrow in b) shows diffusion restriction on DWI. Apparent Diffusion Coefficient (ADC) maps (c) is a calculated image that usually shows signal intensity opposite to the DWI image (arrow in c). Here, the diffusion restricting optic nerve appears hypointense on ADC, confirming that it is not artifactual. ADC maps can also be used to quantify the degree of diffusion restriction by calculating ADC values [labeled as average (Avg) in d] that may help in prognostication

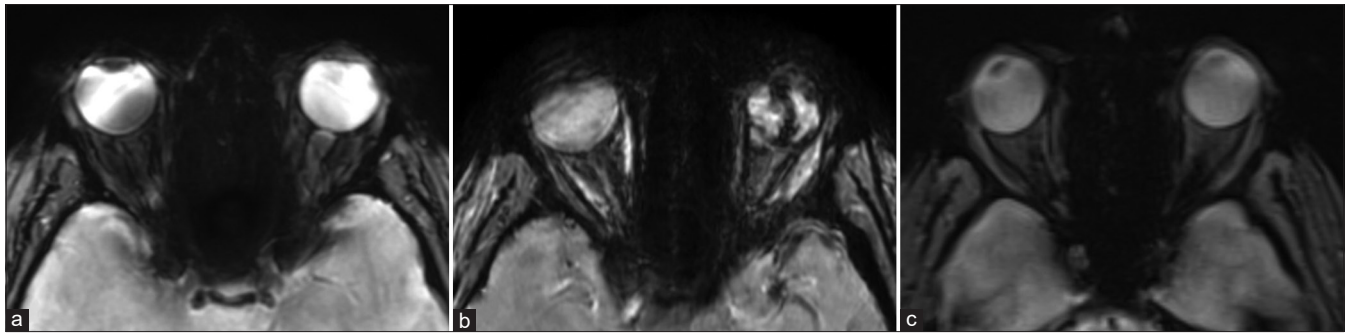


**Figure 6:** Diffusion tractography (DTI). DWI, when acquired with encoding for a higher number of molecular movement directions, allows for the generation of tractography maps. Because they are longitudinal structures, axons within the brain allow preferential movement of interstitial water molecules parallel to them, while limiting the movement perpendicular to them. Thus, diffusion facilitation, in a particular direction, indirectly indicates the white matter tracts. The white matter tracts may be mapped out by placing regions of interest (ROIs) between two structures in the brain. The source images of the DTI maps (a) are color-coded with the red, blue, and green colors indicating craniocaudal, mediolateral, and anteroposterior directions, respectively. Tractography (b) is the graphical representation of white matter tracts connecting two or more ROIs, in this case, the optic tracts

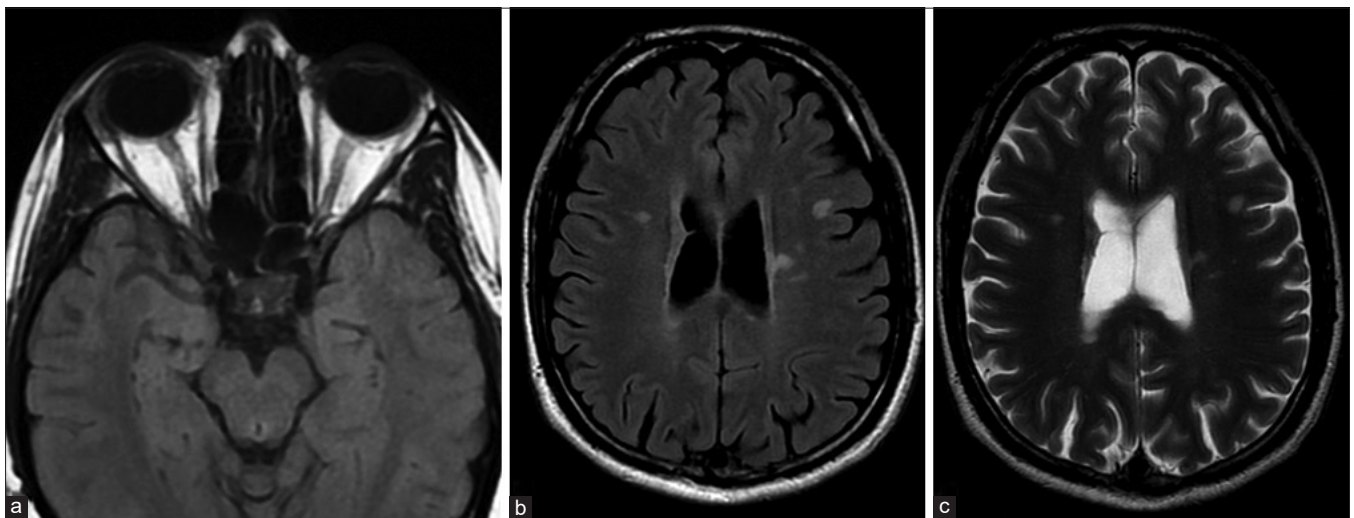
intensity is a non-specific finding. However, only five types of lesions/tissues are T1 hyperintense [Table 4].<sup>[7]</sup> An additional sixth category constitutes artificially introduced paramagnetic substances such as gadolinium-based contrast agents, which also appear hyperintense on T1W. Fat suppressed T1 sequences (T1 FS) aid in the differentiation of fat and other T1 hyperintense substances as fat loses its hyperintense signal and becomes hypointense after suppression. Similar to T2 hypointensity, the presence of T1 hyperintensity is an important finding that helps narrow the differential diagnosis.

#### Pre- and post-contrast (PC) T1 fat-saturated images (T1FS-PC)

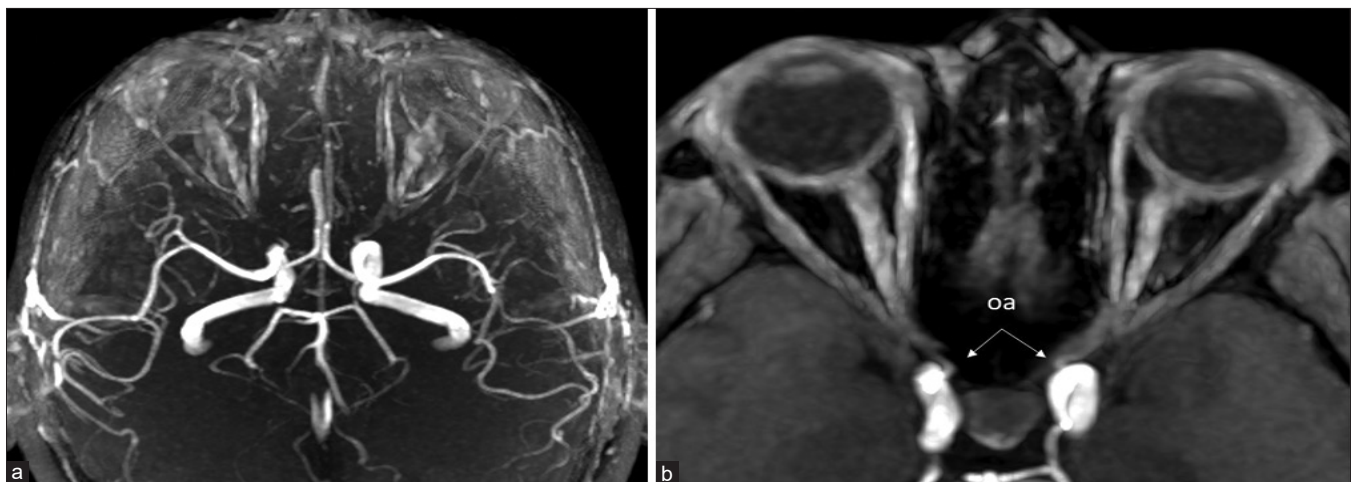
Gadolinium is a paramagnetic substance that appears bright on T1. Gadolinium-based contrast agents distribute in the vascular and interstitial spaces, particularly in areas with increased vascularity secondary to inflammation, angiogenesis, or altered homeostasis. Lesions that take up gadolinium contrast become T1 hyperintense, i.e., appear enhanced on post-contrast T1 and serves as a means of characterizing a lesion's vascularity.<sup>[8]</sup>



**Figure 7:** Susceptibility weighted image (SWI). SWI is a sequence that exaggerates the magnetic susceptibility of various substances against a background of T2 weighting of tissues. Thrombosed structures or hemorrhages comprise iron-containing deoxyhemoglobin, appear more prominent, and hence are said to "bloom" or show "susceptibility." The orbits usually do not show susceptibility except those caused by artifacts from the air in the paranasal sinuses and immobile protons in bones (a). In a case of persistent fetal vasculature with microphthalmos (b), the thrombosed persistent hyaloid artery shows blooming on susceptibility, appearing more prominent than on other sequences. Gradient recalled echo (GRE) (c) sequences are similar to SWI but an older technique and less sensitive than the SWI. These may be the only available sequences in some older MRI machines



**Figure 8:** T2 weighted FLAIR (FLuid Attenuated Inversion Recovery). This is a T2 weighted image with an additional inversion recovery (IR) pulse that negates the signal of free water or free water like substances. This results in an appearance similar to a T2 image except for the hypointense signal of the freely moving fluids, including aqueous and vitreous humor and the watery lens (a). A similar effect is seen in the ventricles of the brain. In multiple sclerosis (b), the demyelinating lesions are seen to a better extent than on T2 weighted image (c) – due to background CSF signal suppression and accentuation of the parenchymal lesions in the FLAIR image

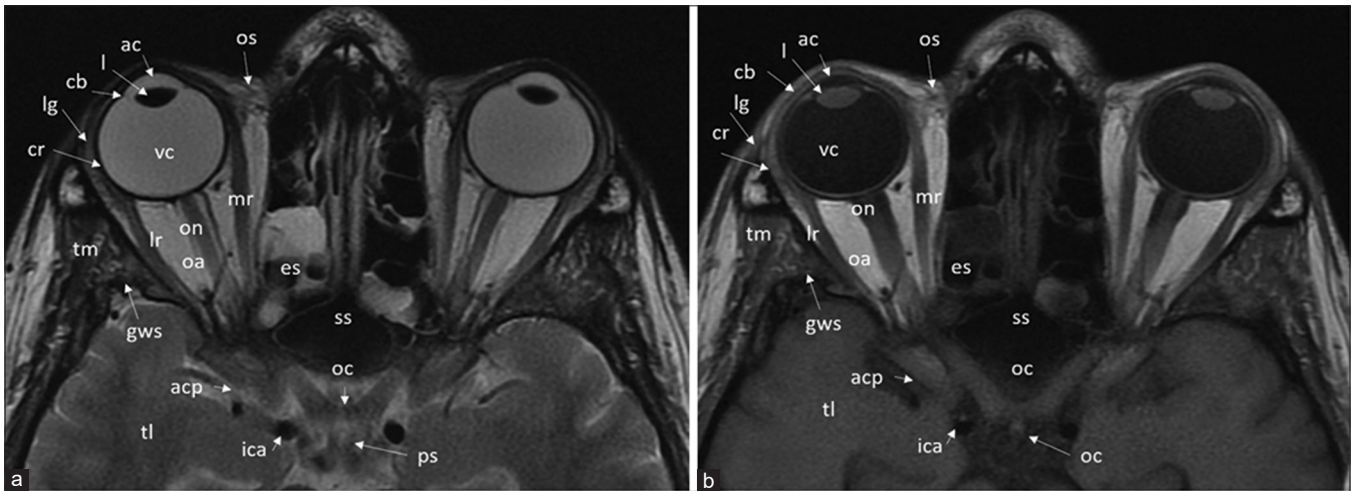


**Figure 9:** MR Angiography. This is a T1 based image with pulse signals that tag protons in flowing blood. Thus, vascular structures stand out as strongly hyperintense structures. Entire cerebral vasculature is represented in (a) along with the origins of the ophthalmic arteries (OA) from the internal carotid arteries in (b)

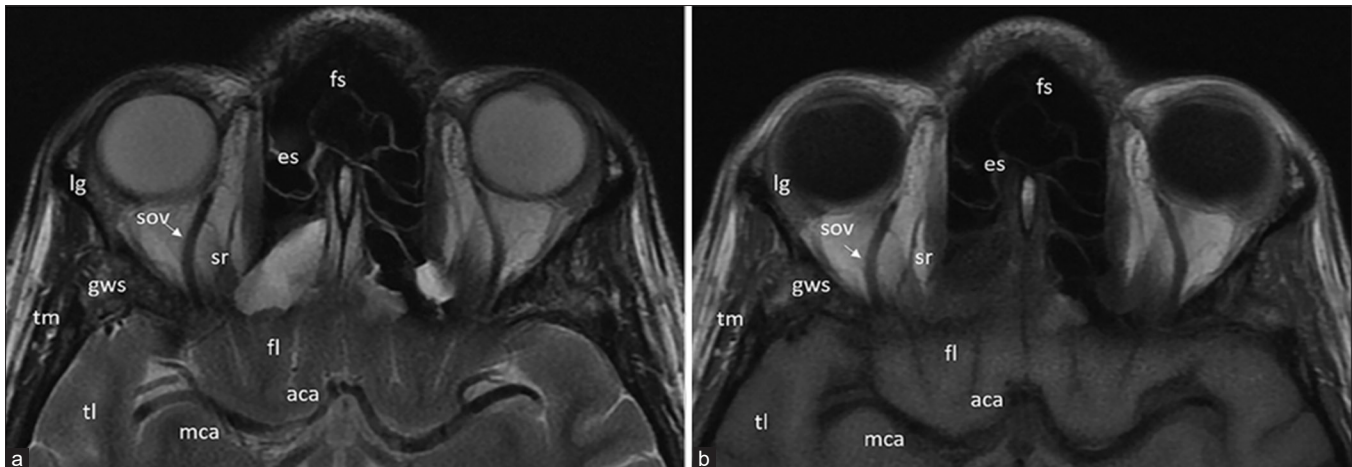




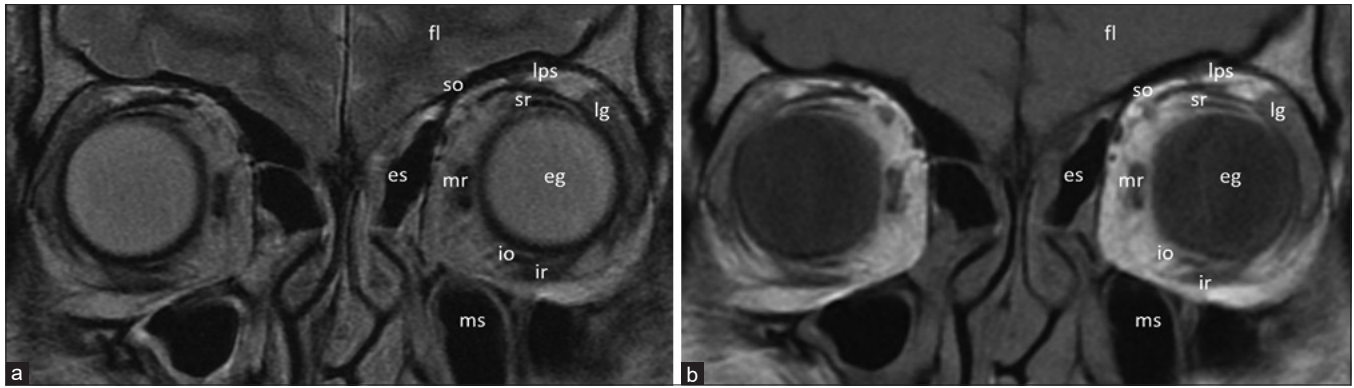
**Figure 10:** Orbital axial anatomy in inferior sections at the level of the inferior recti. Structures seen in T2 (a) and T1 (b) weighted images are inferior rectus (IR), temporalis muscle (TM), greater wing of sphenoid (GWS), temporal lobe (TL), sphenoid (SS), and ethmoid (ES) sinuses, inferior ophthalmic vein (IOV), and nasolacrimal duct (NLD)



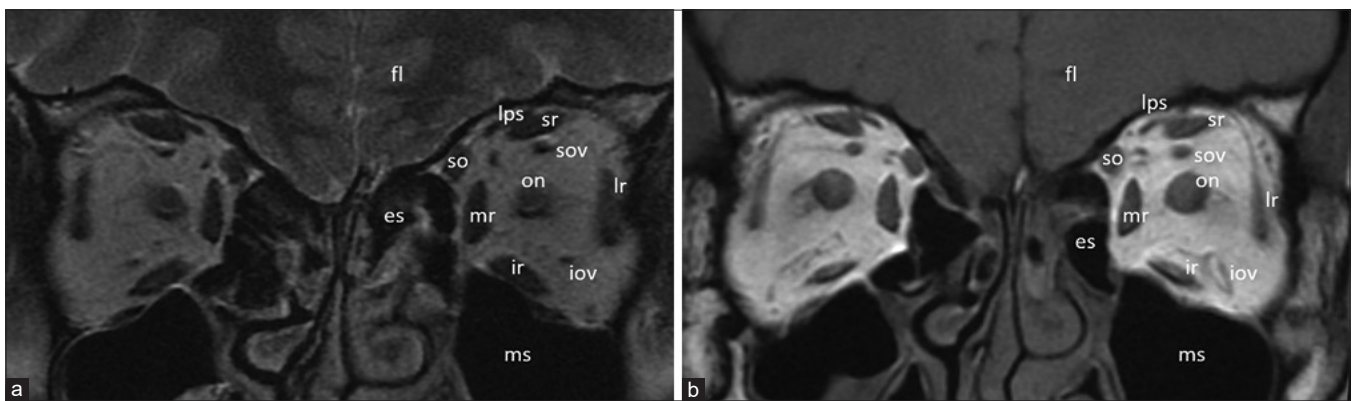
**Figure 11:** Orbital axial anatomy in the midsection at the level of the eye globes. Structures visualized in T2 (a) and T1 (b) weighted images are the orbital septum (OS), anterior chamber (AC), lens (L), ciliary body (CB), lacrimal gland (LG), chorioretinal layers (CR), vitreous cavity (VC), lateral (LR) and medial (MR) recti, optic nerve-sheath complex (ON), ophthalmic artery (OA) flow voids, ethmoid (ES) and sphenoid (SS) sinuses, optic chiasm (OC), pituitary stalk (PS), internal carotid artery (ICA), anterior clinoid process (ACP), greater wing of sphenoid (GWS), temporal lobe (TL) and temporalis muscle (TM)



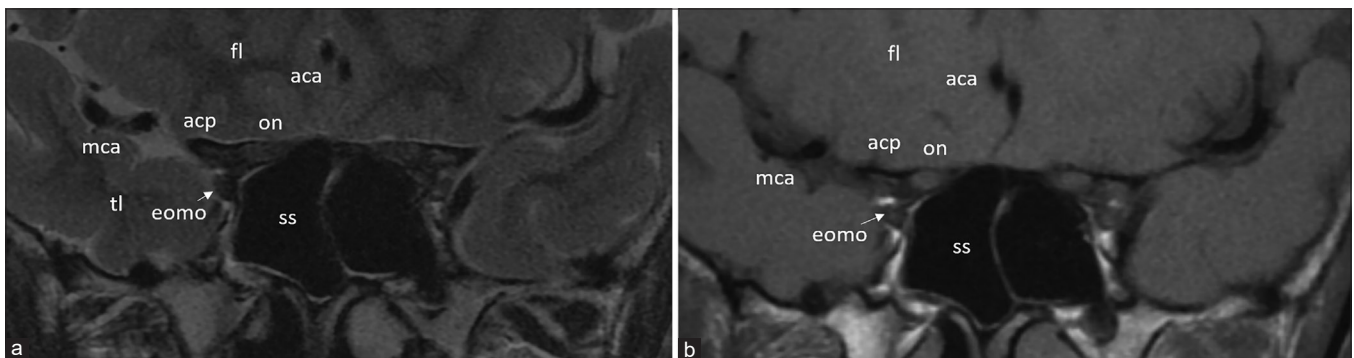
**Figure 12:** Orbital axial anatomy in superior sections at the level of the superior ophthalmic veins (SOV). Structures visualized in T2 (a) and T1 (b) weighted images are the superior (SR) rectus, superior ophthalmic vein (SOV), lacrimal gland (LG), greater wing of sphenoid (GWS), frontal (FL), and temporal lobes (TL), flow voids of the middle (MCA) and anterior (ACA) cerebral arteries, temporalis muscle (TM), and the frontal (FS) and ethmoid (ES) sinuses



**Figure 13:** Orbital coronal anatomy in anterior sections at the level of the eye globes (EG). Structures visualized in T2 (a) and T1 (b) weighted images are the four recti [medial (MR), lateral (LR), superior (SR), inferior (IR)], superior (SO), and inferior (IO) obliques, levator palpebrae superioris (LPS), ethmoid (ES) and maxillary (MS) sinuses, lacrimal gland (LG), and frontal lobe (FL)



**Figure 14:** Orbital coronal anatomy in mid-sections. Structures visualized in T2 (a) and T1 (b) weighted images are the four recti [medial (MR), lateral (LR), superior (SR), inferior (IR)], superior oblique (SO), frontal lobes (FL), optic nerve sheath complex (ON), superior (SOV) and inferior (IOV) ophthalmic veins, and ethmoid (ES) and maxillary (MS) sinuses



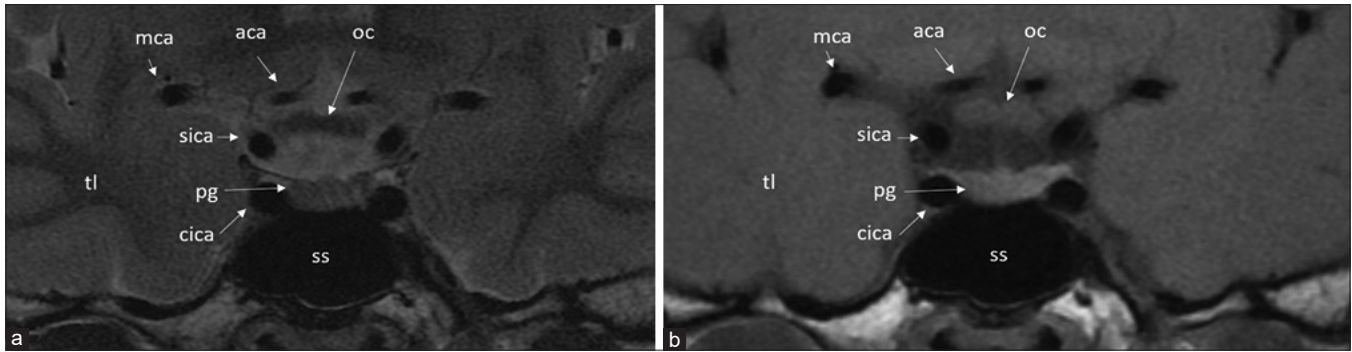
**Figure 15:** Orbital coronal anatomy in posterior sections at the level of the optic nerve canal/orbital apex. Structures visualized in T2 (a) and T1 (b) weighted images are the canalicular segment of the optic nerve (ON), EOM origins (EOMO), anterior clinoid process (ACP), frontal (FL), and temporal (TL) lobes, the flow voids of the middle (MCA) and anterior (ACA) cerebral arteries, and sphenoid sinus (SS)

Recall that the most voluminous content of the orbit is fat, which already appears hyperintense on T1. The enhancement would be difficult to be appreciated against the background of T1 hyperintensity. Hence, a fat-saturated T1 (T1FS) sequence (similar to T2FS) is used to suppress this background fat signal so that only the contrast-enhanced lesion stands out.<sup>[5]</sup> The sequence is always performed before and after contrast administration to allow comparability for even subtle enhancement. Within the orbit, the EOMs, lacrimal gland, and

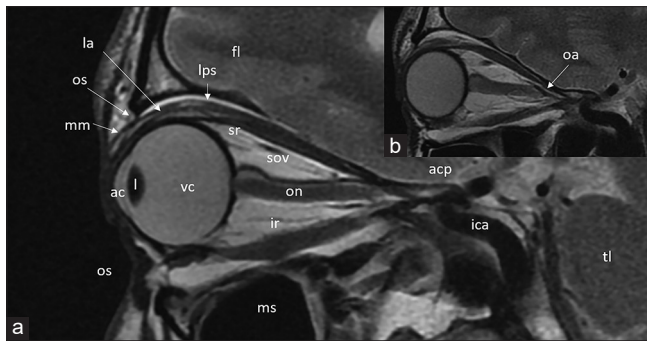
the chorioretinal structures typically show enhancement since they are relatively vascular [Fig. 4]. The optic nerves being a direct extension of the brain have an intact blood-brain barrier and lack contrast enhancement.

Abnormal lesion enhancement can be described by three characteristics: *homogeneity, intensity, and pattern*. Lesions may show either homogenous or heterogenous enhancement to a mild, moderate, or intense degree.





**Figure 16:** Post-orbital coronal anatomy at the level of the optic chiasm (OC). Structures visualized in T2 (a) and T1 (b) weighted images are the flow voids of the supraclinoid (SICA) and cavernous (CICA) internal carotid arteries, middle (MCA) and anterior (ACA) cerebral arteries, pituitary gland (PG), sphenoid sinus (SS), and temporal lobe (TL)



**Figure 17:** Sagittal orbital anatomy (a). In oblique sections in parallel with the optic nerve sheath complex (ON), structures visualized are the levator palpebrae superioris (LPS), levator aponeurosis (LA), orbital septum (OS), Muller's muscle (MM), anterior chamber (AC), lens (L), vitreous cavity (VC), superior (SR) and inferior (IR) recti, superior ophthalmic vein (SOV), maxillary sinus (MS), anterior clinoid process (ACP), the flow voids of the internal carotid artery (ICA), temporal lobe (TL) and maxillary sinus (MS). In a slightly more paramedian section to the optic nerve (inset, b), the ophthalmic artery (OA) flow void can be visualized

Poorly vascular lesions such as lipomas, lymphangiomas, or benign cystic lesions may be *non-enhancing*. Some lesions, such as abscesses and neurocysticercosis, may show a *ring enhancement* pattern. Areas of *non-enhancement within enhancing lesions* may represent cystic areas (usually well-defined margins) or necrosis (more irregular margins). Some lesions such as hemangiomas may show gradually *increasing intensity of enhancement* over successive sequences.<sup>[4,9]</sup>

### Diffusion-Weighted Imaging (DWI)

Diffusion-weighted imaging (DWI) is a special type of sequence that exploits the property of random Brownian motion of water molecules within the tissue's interstitial spaces. The sequence acquisition consists of two parts: a  $B_0$  image, the baseline image (with only the external magnetic field applied), and the  $B_{1000}$  image (which has an additional RF pulse applied for 1000 ms and tracks the motion of water hydrogen protons). Subtracting the images from these two acquisitions results in the "diffusion-weighted image"—a map of the degree of freedom of random movement of water molecules within a tissue [Fig. 5].<sup>[10]</sup>

The regions that allow total unrestricted motion of molecules within them (such as the chambers of the eye, the

CSF in the subarachnoid spaces) or areas of cystic degeneration will appear hypointense on DWI. Tissues such as the optic nerves and brain parenchyma normally show a mild limitation of molecular movement within their intercellular spaces and hence appear isointense on DWI. Edematous tissue with its increased interstitial space will appear more hypointense than the normal counterparts elsewhere and termed "facilitated diffusion."

Lesions that appear hyperintense on DWI are said to show "diffusion restriction," which is always a pathological finding.<sup>[11]</sup> The mechanism for restricted diffusion of water may be varied; however, for e.g. in abscesses, the motion of water molecules is limited by the viscosity of the fluid and dense inflammatory infiltrate. In ischemia or severe demyelinating optic neuritis, cytotoxic edema results in cellular swelling and decreased intercellular spaces with cellular swelling and resultant diffusion restriction. In highly cellular tumors, such as lymphomas, reduced interstitial spaces result in similar diffusion restriction. An additional calculated map called the apparent diffusion coefficient (ADC) map, which is similar to an inverted color image, is also provided. True diffusion restriction will appear hypointense on ADC maps and hyperintense on DWI. Advanced applications based on diffusion include quantitative analysis of the degree of diffusion restriction, which allows differentiation between benign and malignant lesions.<sup>[12]</sup>

Diffusion tensor imaging (DTI) is a variant of DWI that allows for tracking water molecules along white matter tracts resulting in tractography maps [Fig. 6]. DTI allows for 3D reconstructions of the fiber tracts and quantitative evaluation of the integrity of white matter tracts, including the optic nerves and radiation.<sup>[13]</sup> They have an additional prognostic value in the assessment of neuro-ophthalmic pathologies.<sup>[14,15]</sup>

### Susceptibility Weighted Imaging (SWI)

Susceptibility weighted imaging (SWI) [Fig. 7] and its precursor, the Gradient Recalled Echo (GRE) sequence, are techniques that exaggerate the magnetic susceptibility effects of certain types of substances.<sup>[16]</sup> In the simple diagnostic context, SWI is useful to detect susceptibility from minerals such as iron in heme and calcium in calcifications. Thus, hemorrhagic areas or calcification will appear slightly enlarged and darker compared to other sequences and are said to show "blooming" or "susceptibility foci." SWI is superior to GRE and provides

**Table 5: Standard Orbit MRI protocol****Plain Scan:**

- Axial: T1, T2, T2FS
- Optional: Volumetric highly T2WI (DRIVE, FIESTA, SPACE)
- Sagittal (along the course of the optic nerve): T2FS,
- Coronal: T1, T2, T2FS/STIR
- Other basic sequences: DWI, SWI

**Vascular Lesion**

- TOF and Contrast-enhanced MR Angiography

**If IV Contrast required:**

- Pre-contrast: T1FS Axials±Coronals±Perfusion imaging
- Post-contrast: T1FS in all 3 planes+3D T1FS including brain

**Brain screening**

- FLAIR Axials for demyelinating lesions
- T2 Sag, 3D T1PC Axials to assess for extension

**Table 6: Radiological approach to the orbit**

- **Knowledge** of normal anatomy, including anatomical variants, age differences, imaging blind spots, and normal measurements is crucial.
- **Detection** of an abnormality: asymmetries, use T2FS, PC-T1FS.
- **Compartmental localization** of the abnormality and probable structure of origin.
- **Characterization** of the lesion with clinical correlation leading to a probable diagnosis and differential possibilities.
- **Assessment of the extent** of the lesion, including extra orbital extension and mass effect.

additional valuable information in characterizing lesions such as retinoblastomas, optic nerve sheath meningiomas, venolymphatic malformations, etc. Thrombosis within the orbital vessels in vascular diseases will also be well demonstrated.

**T2 Weighted FLuid-Attenuated Inversion Recovery (T2 FLAIR)**

Screening of the brain is of utmost importance in the evaluation of neuro-ophthalmologic conditions. As in orbit, a T2 weighted image is most suited for screening of the cerebral parenchyma, brainstem, and cerebellum. However, the brain is bathed in hyperintense CSF, making lesions less easy to detect. Similar to fat saturation for orbit imaging, FLAIR is a T2 weighted image with suppression of signal from the fluid. Thus, the CSF and aqueous and vitreous humor appear hypointense on FLAIR [Fig. 8]. This allows for better detection and delineation of cerebral parenchymal abnormalities such as mass lesions, demyelinating plaques, or perilesional edema.<sup>[1]</sup> FLAIR sequence can also be utilized to confirm the presence of cystic changes, which will appear hyperintense on T2WI but hypointense on FLAIR due to suppression of the signal. The presence of a significant abnormality on FLAIR should alert the attending radiologist to extend the study to include a complete evaluation of the brain and spine with or without contrast.<sup>[5]</sup>

**MR Angiography (MRA)**

Magnetic resonance angiography (MRA) is a general term used for a range of non-contrast and contrast-based sequences that enable visualization of the blood vessels. MRA is useful

in screening for intracranial aneurysms in ophthalmoplegic conditions, assessing orbital and intracranial vascular malformations, including carotid-cavernous fistulae (CCF), central retinal arterial and venous occlusions, and in the evaluation of amaurosis fugax.<sup>[17]</sup> Non-contrast techniques, most commonly the time-of-flight (TOF) sequence [Fig. 9], are widely used as they are entirely noninvasive and simple to perform.<sup>[18]</sup> TOF MRA utilizes higher velocity flow related signals that are usually found in the arterial tree. Anomalous signal within a venous structure such as cavernous sinuses or ophthalmic veins thus enables detection of shunting arteriovenous lesions. More advanced “4D” techniques such as Time Resolved MRA sequences are capable of multiphase acquisitions. This results in multiple snapshots that capture the flow of blood from the arterial to venous ends of vascular malformations such as AVMs and CCFs. This allows for delineation of angioarchitecture almost reaching the temporal resolution of digital subtraction angiography.<sup>[19]</sup>

**Perfusion weighted imaging (PWI)**

Unlike contrast enhancement, which represents a combination of vascularity and interstitial fluid seepage, PWI focuses solely on the vascularity aspect. The assessment of perfusion on MRI can be performed using non-contrast techniques, such as arterial spin labeling (ASL), and contrast techniques, such as dynamic susceptibility contrast (DSC) and dynamic contrast enhancement (DCE). DSC imaging is T2 based and is invaluable in the assessment of vascularity and the grading of tumors, while DCE imaging is T1 based and can demonstrate flow patterns in slow flow venous malformations or enhancement patterns in cavernous hemangiomas.<sup>[20]</sup>

In conclusion, a standard protocol for MRI of the orbit would include thin sections (usually 2–3 mm) of the above sequences, acquired in the axial, coronal, or sagittal planes. An ideal protocol for MR imaging of the orbit is provided in Table 5.

**Radiological Approach to Orbital Disease**

A radiological approach to the orbit should consist of a systematic series of steps [Table 6]. A basic depiction of anatomical structures within the orbit on T1 and T2 weighted images is illustrated in Figs. 10-17. While obvious lesions are usually easily detectable, it is the subtle lesions that are often missed. A few general rules of thumb may aid in reducing this false-negative rate. In the head and neck, bilateral symmetry is an essential clue to the **detection** of lesions. Any asymmetric structure or enlargement must be viewed with suspicion and examined on other sequences, views, or modalities to rule out a significant pathology. Contrast-enhanced scans are particularly advantageous for picking up small lesions by merely looking for asymmetric contrast enhancement. Certain lesions may only be diagnosed or identified on a few specialized sequences such as DWI or SWI.

A key approach to the radiological diagnosis of lesions is to narrow the differentials by **localizing** the lesion to a region or compartment of the orbit and ascertaining the structure of origin. Lesions may either be localized to a single compartment or maybe multispatial.<sup>[8]</sup>

**Characterization** of the lesion consists of analyzing a series of imaging features. Characterize the type of tissue—

does it consist of soft tissue? Are there areas of fluid (cystic degeneration/necrosis), fat, or blood? Assess the vascularity based on the presence of flow voids and with perfusion imaging when available. Grade the degree of contrast enhancement into mild, moderate, or intense—this serves as a surrogate marker of not just lesional vascularity but also the “leakiness” or permeability of its microvascular bed allowing seepage of gadolinium contrast medium into its interstitium. Structural characterization is based on its internal architecture (whether homogenous or heterogeneous) with a solid, cystic, or variegated pattern. Look for the shape of the lesion and its margins (whether well-defined, ill-defined, infiltrative), which will help assess whether it is an aggressive or benign growth pattern. Finally, evaluate the extent of a lesion and any possible invasion of critical structures by inspecting the paranasal sinuses, maxillofacial and intracranial regions. Look for mass effect in terms of displacement or compression of adjacent structures.

The combination of patient’s age, clinical history, and examination findings, anatomical compartmentalization, and lesion characterization helps narrow down the differential diagnoses. Effective teamwork may positively affect patient safety and clinical outcome. It is a safe practice to communicate with the radiologist for optimal imaging evaluation and not miss additional significant findings that may impact management or change a diagnosis. We hope that this review would serve as a useful primer to enable the ophthalmologist to utilize this elegant imaging modality more frequently and to its fullest potential. In part two of this article, we discuss the MR imaging features of the specific orbital lesions.

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#### Conflicts of interest

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