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Imaging In Neuro-Ophthalmology

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Abstract

Neuro-ophthalmological pathologies require dedicated imaging studies for proper localisation and appropriate diagnosis. Technical advancements in MRI and CT have facilitated early diagnosis and treatment of various neuro-ophthalmological disorders. This review article has tried to focus on specific imaging techniques and imaging features of these neuro-ophthalmological disorders.

Keywords: Optic nerve; Cranial nerve; Cavernous sinus; MRI

Introduction

Ophthalmologists are the first to examine the patient and recommend specific imaging modality, need of contrast and additional imaging. They should have adequate knowledge of specific imaging sequences required to localise the lesion and should request for the same. Lack of these facts can lead to inadequate and incomplete study causing errors in diagnosis and subsequent delay in treatment.

The various indications for neuro-ophthalmological imaging includes [1]

1. Unilateral / bilateral vision loss (acute or progressive)
2. Efferent / afferent pupillary defects
3. Proptosis
4. Double vision / external ophthalmoplegia
5. Lid abnormalities
6. Oscillopsia (Nystagmus)
7. Ophthalmoscopic abnormalities like papilloedema etc
8. Ocular / orbital trauma

Imaging Modalities and Techniques

Various imaging modalities can be used for imaging in neuro-ophthalmological diseases like X-rays, ultrasound, Doppler, CT scan, Magnetic resonance imaging and catheter angiography. Role of X-rays for orbit is very limited except for visualising foreign bodies and bony abnormalities. Ultrasound and Doppler studies are used for abnormalities of the globe.

Computed tomography (CT)

CT scan plays important role in identifying haemorrhage, calcification, fractures, bony lesions and foreign bodies. It is useful in finding orbital extension of diseases from paranasal sinuses into cavernous sinus or orbital apex. 3D multiplanar reconstructions, maximum intensity projections, CT angiography add value to CT scan. Thin cuts of 0.6 mm with coronal/Sagittal /oblique reconstruction is the protocol followed in our institution.

Magnetic resonance imaging (MRI)

MRI due to its high spatial and temporal resolution, good tissue contrast and multiplanar reconstruction capabilities is preferred choice in neuro-ophthalomology. MRI with contrast is more useful in characterising the diseases and to locate subtle findings. MR angiography is useful to find vascular abnormalities causing secondary ocular symptoms. Advanced techniques like tensor imaging (for optic nerve) and CISS 3D can be used in some pathologies. Our Protocol includes-T1W and T2W axial and Coronal-3 mm slice thickness with 1mm interslice gap, Fat suppressed T1 Coronal and Sagittal pre and post contrast and fat sat T2 coronal apart from routine sequences of brain and spine as indicated.

Catheter angiography

The most common indication is suspicion of carotid cavernous fistula which causes proptosis and chemosis.

The various neuro-ophthalmological disorders can be described according to signs and symptoms as per the indications.

Diseases Causing Visual Problems

Various disorders can cause unilateral or bilateral visual problems like blurring or field defects. Afferent pupillary defects can be seen in pathologies affecting optic nerves and chiasm. Detailed anatomy of the visual pathway is needed for proper localization (Table 1).

Field defect	Localization
Single eye vision loss	Retina or optic nerve
Bitemporal hemianopia	Chiasm
Junctional scotoma (central defect one eye and temporal defect other eye) Incongruous hemianopia	Junction of optic nerve and chiasm
Incongruous hemianopia	Optic tract or lateral geniculate body
Congruous hemianopia	Occipital lobe
Complete homonymous hemianopia	Anywhere in retrochiasmal tract
Superior quantrantanopsia	Temporal or occipital lobe
Inferior quandrantanopsia	Parietal or occipital lobe
Macular sparing hemianopia	Occipital lobe

Table 1: Field defects and its localisation.

Diseases which can cause unilateral visual problem are located in retinal region, optic nerve head and optic nerve. However bilateral visual problems are caused due to pathologies affecting bilateral optic nerves, optic chiasm, retrochiasmal tract and cortex.

Pathologies Affecting Optic Nerve

Lesions affecting optic nerves can be divided as

1. Inflammatory
2. Ischemic
3. Nutritional
4. Infiltrative
5. Neoplastic
6. Compressive neuropathy
7. Drug induced changes

Inflammatory lesions affecting optic nerves

Optic neuritis presents with sudden onset blurring of vision or visual loss which can be unilateral or bilateral and on examination shows relative afferent pupillary defect (RAPD). Investigation of choice is MRI with contrast. MR imaging in acute optic neuritis should include dedicated imaging sequences for orbit, brain and spinal cord along with contrast study.

On imaging, optic nerve appears Hyperintense on T2 and isointense on T1 and may be swollen. The contrast enhancement in acute optic neuritis is very sensitive (94%) and it can affect variable length and region. Longer segment and foraminal region involvement suggests poor initial vision but it does not predict recovery [2]. Inflammatory pathologies which affect optic nerve are predominantly demyelinating diseases like multiple sclerosis (MS) and neuromyelitis optica (NMO).

NMO was initially considered to be spectrum of multiple sclerosis but it is well known now as a different entity. The involvement of brain and spinal cord in both these entities is different. In NMO, lesions in brain are atypical (not like MS) and may involve medulla and hypothalamus, whereas in MS involvement of brain is typical (periventricular involvement). Cord involvement in NMO is seen for more than 3 segments and central part of cord is involved.

however can be bilateral. In 10% cases of NMO, simultaneous spinal cord and optic nerve can be involved [3]. The risk of developing MS by 15 years after onset of acute neuritis is 25% in patients who do not have brain lesions in initial scan and 72% in patients who have 1 or more lesions at initial scan [4] (Figure 1).

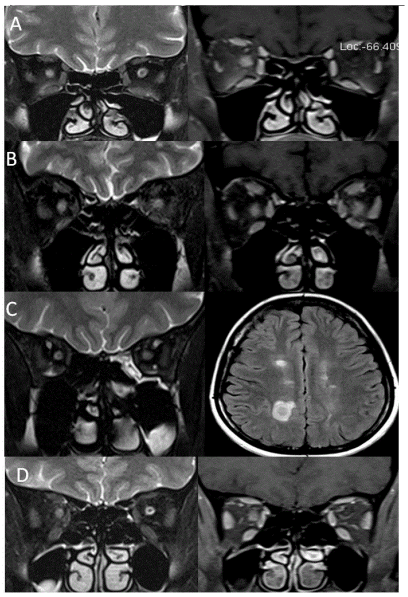

Figure 1 A to D: Patients presenting with acute onset blurring of vision / vision loss. T2 coronal FS and post contrast T1 FS sequence showing enhancing unilateral (RT) optic nerve in A and bilateral in B. C shows bilateral asymmetrical optic neuritis (acute on left side) due to multiple sclerosis. Multiple demyelinating plaques seen on FLAIR. D shows enhancing right optic nerve and atrophy of left optic nerve; combination of acute optic neuritis on right and chronic on left side.

Ischemic optic neuropathy (ION)

Ischemic optic neuropathy can be divided into anterior (AION) and posterior (PION). AION can be further divided into arteritic and non arteritic [5].

Arteritic AION is seen in elderly individuals more than 60 years with systemic signs of myalgia, temporal pain and transient visual loss or diplopia and is commonly associated with giant cell arteritis [6].

Non arteritic AION is seen in individuals more than 50 years with systemic vascular disease like hypertension and diabetes [7]. Differentiation between them can be done fundoscopically by cup to disc ratio in other eye [6].

Role of MRI is primarily to rule out demyelinating diseases and mass lesions. Non arteritic AION does not show any abnormality on T1W or contrast imaging [8]. However posterior ischemic optic neuropathy may show contrast enhancement on MRI [9].

Sometimes acute ischemic optic neuropathy can show diffusion restriction on MRI like acute infarct [10]. Our experience in non arteritic AION shows atherosclerotic changes in ophthalmic segment of internal carotid artery on MR angiogram with hyperintense signal changes in optic nerve on T2W without swelling and contrast enhancement (Figure 2).

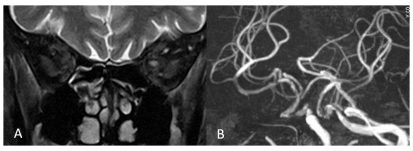

Figure 2 A to B: Patient presenting with unilateral visual loss. A shows bright left optic nerve on FS T2 Coronal image and MR angiogram MIP image in B showing atherosclerotic narrowing of bilateral ophthalmic and supraclinoid segment of ICA. There was no contrast enhancement (not shown) of the optic nerve. Case of non arteritic AION.

Toxic / nutritional optic neuropathy

Common causes of toxic optic neuropathy includes methanol, ethanol, tobacco and drugs like ethambutol, amiodaron, Chloramphenicol, cisplatin etc [11-14]. These toxins affect pappilomacular bundles and cause central and centrocaecal scotoma. Nutritional deficiencies of vitamin B1, B6, B12 cause similar effects like toxic optic neuropathy. We found subtle loss of CSF space surrounding both optic nerves without contrast enhancement on MRI (Figure 3). Differentiating features of various types of optic neuritis is described in Table 2.

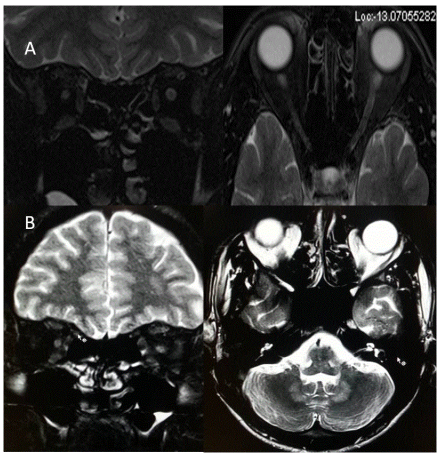

Figure 3A to B: Young patients presenting with bilateral blurring of vision with peripheral field defects. A- Loss of CSF space surrounding right optic nerve (swollen nerve) on FS T2 coronal and axial which was not enhancing on contrast (not shown). It was a case of Vitamin B12 deficiency. B-case of suspected linozolid toxicity with bright bilateral optic nerves with signal changes in bilateral dentate nucleus.

Optic neurltis(NMO)	Optic neuritis (MS)	Nutritional optic neuritis	Ischemlc optic neuritis
Enlarged optic nerve	Enlarged optic nerve	No enlargement (only loss of CSF space with signal changes)	No enlargement (loss of CSF space with signal changes)
Bright contrast enhancement	Bright contrast enhancement	No contrast enhancement	No contrast enhancement enhancement enhancement/mild focal enhancement
No restricted diffusion	No restricted diffusion	No restricted diffusion	Restricted diffusion
No vascular changes	No vascular changes	No vascular changes	Vascular changes seen
Non specific brain lesions	Specific brain lesions	Non specific brain lesions	Non specific brain lesions
Specific cord changes (>3 vertebral involvement and central cord	Non specific cord changes (1 vertebral involvement and posterolateral cord	Specific cord changes (SACD in B12 deficiency)	No cord changes
Non specific field defects	Non specific field defects	Central or centrocaecal scotoma sparing peripheral fields	Non specific field defects
Painful	painful	painless	painless
NMO-neuromyelitis optica, MS-multiple sclerosis, SACD-subacute combined degeneration			

Table 2: Differentiating features of various types of optic neuritis.

Infiltrative lesions of optic nerve

Systemic malignancies like leukemia, lymphoma and multiple myeloma can involve optic nerve as sole manifestation or as a part of systemic disease. It can infiltrate optic nerve causing swelling and enhancement on imaging [15-17]. The optic nerve sheath can be infiltrated causing compression of central retinal artery and vein leading to visual symptoms [18] (Figures 4A and 4B).

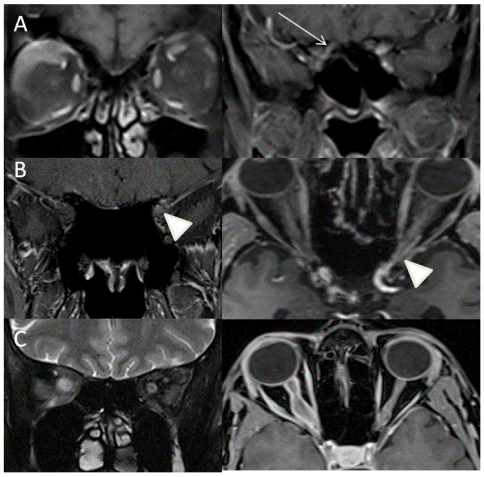

Figure 4A to C: Infilterative pathologies of optic nerve: Case of chronic myeloid leukemia who presented with sudden onset visual blurring showed enhancing leukemic infilteration in right orbit superior portion and at optic strut (thin arrow)as shown in A . Figure B shows enhancing lymphomatous infilteration of left optic nerve at optic foramina (arrow head) This patient presented with sudden onset unilateral blindness. Figure C shows enhancing right side optic nerve sheath meningioma with tram line sign. Patient presented with progressive unilateral visual loss.

Tumors of optic nerve

It can be divided into tumors of optic nerve and optic nerve sheath. Glioma is commonest tumor that affects optic nerve however uncommon tumors include ganglioglioma, medulloepitheliomas and haemangioblastoma. The most common optic nerve sheath tumor is meningioma. Optic nerve tumors commonly present with progressive vision loss with proptosis.

Optic nerve gliomas are unilateral and frequently seen in females and commonly associated with neurofibromatosis type 1. On imaging they are seen as T2 hyperintense enlarged nerve with kinking and enhancement. The symptoms are usually due to compression of central retinal vein.

Optic nerve meningiomas are usually unilateral seen commonly in middle age but can be seen in children and young adults and can be bilateral. On MR imaging, optic nerve is enlarged demonstrating uniform peripheral contrast enhancement with non-enhancing central optic nerve (tram track sign) and can be associated with pneumosinus dilatans [19,20] (Figure 4C).

Compressive pathologies of optic nerve

Optic nerve can be compressed by the mass lesions anywhere along its course. The common lesions that cause optic nerve compression are meningiomas, pituitary adenomas and paranasal sinus lesions. The vision loss is usually gradual. Sometimes vision loss can be rapid in cases of aneurismal compression or in pituitary apoplexy [21] (Figure 5). Some congenital rare causes of progressive visual field defects are described below.

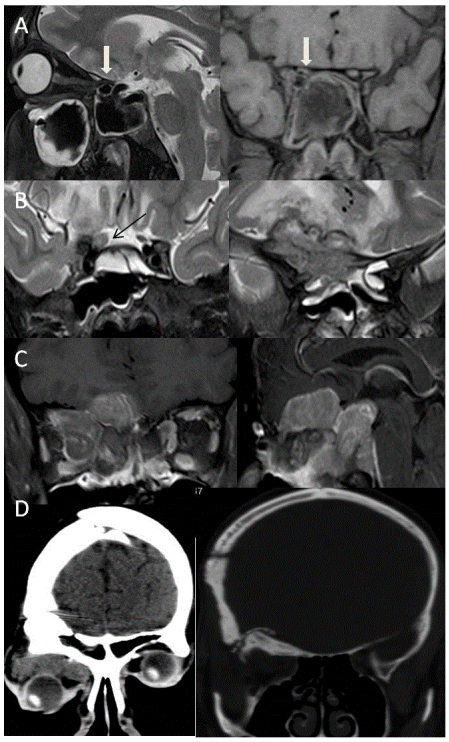

Figure 5A to D: Patients presenting with progressive visual loss. Compressive pathologies of optic nerve: Figure A shows sphenoid sinus mucocele causing compression of optic nerve (thick white arrow). Figure B shows encasing fungal mass on right optic nerve (black thin arrow) with cavernous sinus involvement. A case of invasive pituitary adenoma compressing and encasing right optic nerve in C. Intradiploic meningioma of right frontal bone and greater wing of sphenoid with soft tissue mass in superior orbital portion stretching globe and optic nerve(post operative) as shown in D.

Optic nerve head drusen: Optic disc drusen is uncommon cause of progressive visual field defect seen as small calcified lesion at the optic nerve head on CT scan. It is bilateral in 13% with incidence of 2.4%. They occur due to progressive calcification of mucoproteins and mucopolysaccharides globules [22] (Figure 6A).

Coloboma: Coloboma are developed due to failure of closure of fetal fissure posteriorly with absent choroid and retina at that region. It is seen inferiorly and on nasal side as focal bulges and may present with field defects [23] (Figure 6B).

Staphyloma: Staphyloma are the bulges of sclera on posterior aspect of eyeball with longer and wider globe seen commonly in high myopics and can be the cause of field defects [24] (Figure 6C).

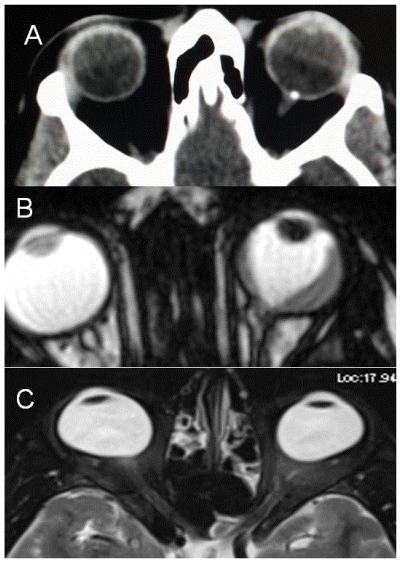

Figure 6A to C: CT axial image showing incidental left optic nerve head drusen in A. B- Patient presented with unilateral nasal field defect. Axial CISS -3D image showing coloboma of left globe involving optic nerve head with retinal detachment. In C patient presented with bitemporal field defects due to bilateral staphyloma.

Lesions of optic chiasm

Optic chiasm can be affected by infectious/inflammatory or neoplastic diseases of basal cisterns .Rarely can be affected by drugs. The presenting symptoms are usually bitemporal hemianopia in compressive etiology and junctional scotoma if the lesion is compressing optic nerve and chiasm junction.

Opto-chiasmatic arachnoiditis: Opto-chiasmatic arachnoiditis can occur due to trauma, infection like tuberculosis, tumors and subarachnoid haemorrhage [25-27]. However most common cause in developing countries is tuberculosis. It is seen commonly in young females and frequently associated with paradoxical response to antitubercular treatment. Clinically it is very important to identify this entity as it causes severe visual loss and thus has to be treated promptly. On imaging there is frequent enhancing exudates surrounding the optic chiasm with extension along the post chiasmal tract (Figure 7).

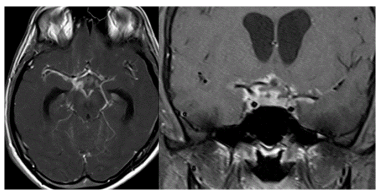

Figure 7: Tubercular exudates surrounding optic chiasm and post chiasmal right optic tract (post contrast T1W).

Toxic opto-chiasmatis: Various substances like ethambutol, isoniazid, methanol and tobacco are toxic to optic nerves and optic chiasm. Toxicity of the anti-tubercular drugs typically present after 2 to 8 months of treatment with dyschromatopsia (especially for blue and yellow colour) as earliest sign of toxicity. It causes central scotomas, but bitemporal and peripheral field defects can also be seen [28-30]. On Imaging, the optic chiasm is swollen with hyperintense signal changes (Figure 8).

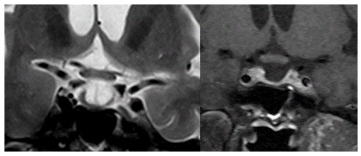

Figure 8: Patient on 2 months of antitubercular treatment presented with blurring of vision, peripheral field defects and difficulty in identifying color contrast. T2W and T1W coronal images shows bulky optic chiasm (ethambutol induced optochiasmatis).

Compressive pathologies of optic chiasm: Optic chiasm can be compressed by large lesions in basal cistern like craniopharyngioma, pituitary adenoma, arachnoid cyst, epidermoid, aneurysms, meningioma etc. The most common clinical feature is bitemporal field defects. Sometimes it can cause junctional scotoma if compression is at the optic nerve/chiasm junction (Figure 9).

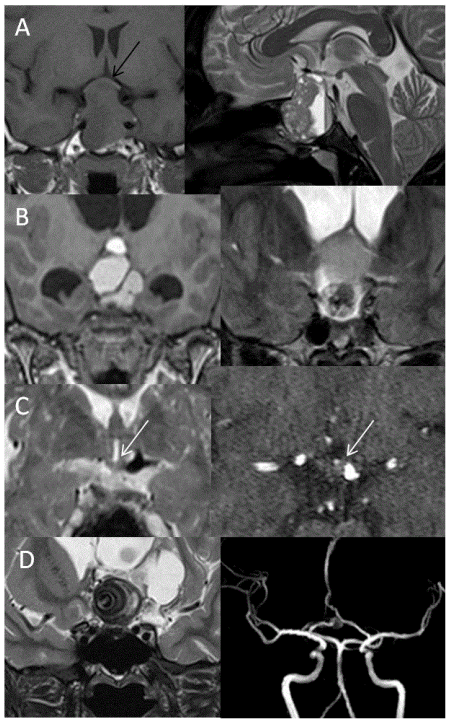

Figure 9A to D: Compressive pathologies of optic chiasm patients presenting with bitemporal field defects (except in C where the presentation was junctional scotoma): Tumor compression of optic chiasm shown in A (pituitary macroadenoma with suprasellar extension) and B (craniopharyngioma). Compression on optic chiasm by ectatic left supraclinoid ICA shown in C and by partially thrombosed ACOM aneurysm shown in D.

Tumors of optic chiasm: The most common tumor of optic chiasm is glioma however few rare tumors like primary chiasmatic germinoma and glioblastoma are also described [31-33]. Different pattern of affection of chiasmal gliomas is described between neurofibromatosis (NF) and non-neurofibromatosis group. Gliomas in NF group, affect optic nerve more commonly (66%) as compared to optic chiasm (62%). On imaging, the chiasm is thickened with preserved contour. In non NF group, chiasmal glioma is most commonly seen with mass like cystic component, variable enhancement, distortion of the contour and extension beyond optic pathway [33] (Figure 10).

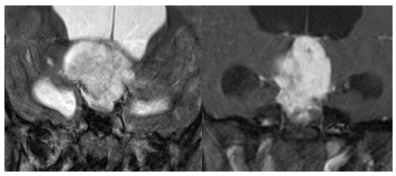

Figure 10: Case of optochiasmatic glioma with progressive bilateral vision loss. Enhancing tumor in suprasellar region.

Cortical lesions causing field defects

Field defects can be variable depending on the site of insult to optic radiation due in brain parenchyma. Various lesions causing field defects are hypoxic injury, arterial/venous infarcts, tumors, granulomas, traumatic contusions etc. Thus depending on site of lesions the field defects can be homonymous hemianopia or quandratanopia (Figure 11).

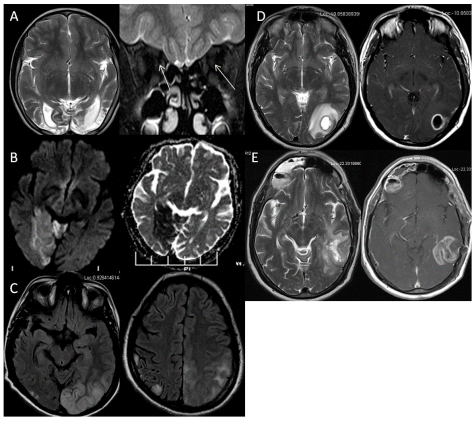

Figure 11A to E: Cortical lesions causing vision loss: A- case of bilateral cortical blindness since birth. Imaging shows bilateral chronic PCA territory infarcts with secondary optic atrophy (white thin arrow). Figure B shows acute homonymous hemianopia in elderly due to acute PCA infarct. Figure C shows a case of MELAS (mitochondrial cytopathy) with multiple cortical infarcts presenting with quandrantanopia. D shows cysticercal cyst in left occipital lobe and E shows high grade glioma with incongruous homonymous hemianopia.

Bilateral visual problem due to raised intracranial pressure

Patients presenting with bilateral gradual visual loss or blurring of vision often shows features of papilloedema on fundoscopy, suggesting raised intracranial pressure. Raised intracranial pressure causes enlargement of the optic nerve sheath as optic nerve sheath communicates with the brain subarachnoid space.

Raised intracranial pressure can be idiopathic or secondary to venous thrombosis/stenosis, dural arteriovenous malformations, tumors, obstructive hydrocephalus, meningitis and trauma. MRI findings which suggest raised intracranial pressure are increased optic nerve sheath diameter, indentation on optic nerve head, vertical tortuosity and kinking of optic nerve, empty sella and widening of foramen ovale. There is direct correlation with raised intracranial pressure and increase in optic nerve sheath diameter. The cut off value of 5.82 mm of optic nerve sheath diameter is considered as best predictor of raised intracranial pressure [34] (Figure 12).

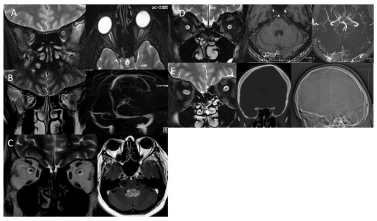

Figure 12A to E: Patients with blurring of vision and papilloedema . Widened CSF space noted in A with indentation at optic nerve head; a case of idiopathic intracranial hypertension. B shows secondary intracranial hypertension due to bilateral transverse sinus stenosis seen on MR venogram. C shows widened CSF space surrounding both optic nerves due to 4th ventricular papilloma. D shows right transverse sinus dural arteriovenous malformation seen in source and MR angiogram with raised pressures. Case of craniosynostosis (fused all sutures) with raised intracranial pressure seen in E.

Diseases causing strabismus and lid abnormalities

Squint and lid abnormalities like ptosis occur due to diseases affecting cranial nerves or muscles. Depending on the pathology, patient may present with only ptosis (as seen in isolated 3rd nerve palsy) or squint due to involvement of one or more extra ocular muscles. Various congenital and acquired pathologies can cause symptoms of ocular movement disorders.

Abnormalities of 3rd, 4th and 6th cranial nerves

Congenital fibrosis of extra-ocular muscles (CFEOM) is a non-progressive congenital restrictive movement disorder of eye, either due to abnormality in innervation of extraocular muscles by 3rd and/or 4th cranial nerves (all or part of nerve) or abnormality in the muscles innervated by these nerves. Depending on clinical features and genetics, it is classified into 3 types. Whether CFEOM is a primary muscle disorder or due to neurogenic cause is debatable. There are few case descriptions of muscle fibrosis on pathology [35,36]. There are other reports favouring neurogenic cause, as there is frequent association of CFEOM with Marcus–Gunn Jaw winking phenomenon [37]. In CFEOM, the third nerve is either normal or may be

hypoplastic (bilaterally or unilaterally) on MR Imaging. The normal diameter of third nerve on CISS 3D was found to be 2.01+/-0.36 mm and abnormal nerve was 1.14+/-0.61 mm suggesting hypoplasia. 3rd nerve hypoplasia can also be seen in congenital third nerve palsy. [38]. Additionally, there are atrophic extraocular muscles with hyperintense signal changes (Figure 13).

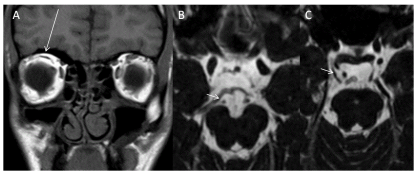

Figure 13A to C: Patient with congenital squint. A-shows atrophic right superior Rectus and levator palpebrae muscle (long arrow). B and C shows hypoplastic right 3rd nerve (short arrow) in axial CISS 3D sequence.

In some cases where there is CFEOM with synergistic divergence, it was found that 3rd nerve is hypoplastic bilaterally with absent 6th nerve on the side of synergistic divergence [39] (Figure 14).

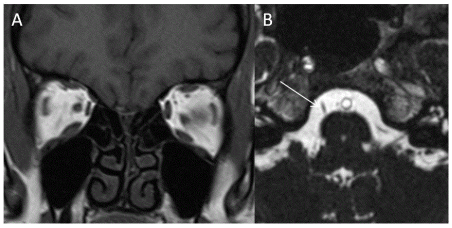

Figure 14A and B: A and B: Patient with congenital squint. A shows atrophic left lateral rectus and B shows absent left 6th nerve (white arrow showing normal right nerve).

In Mobius syndrome, 6th and 7th nerves are hypoplastic and patients present clinically with facial paresis and abduction deficits. In Duane retraction syndrome, 6th nerve is hypoplastic or absent with innervation of lateral rectus by 3rd nerve (miswiring). In CFEOM 3rd, 6th and 4th nerves are known to be hypoplastic with hypoplasia of corresponding extraocular muscles [40]. 3rd, 4th and 6th cranial nerves can be compressed along their course by tumors, vascular lesions like aneurysms and leptomeningeal diseases (Figure 15).

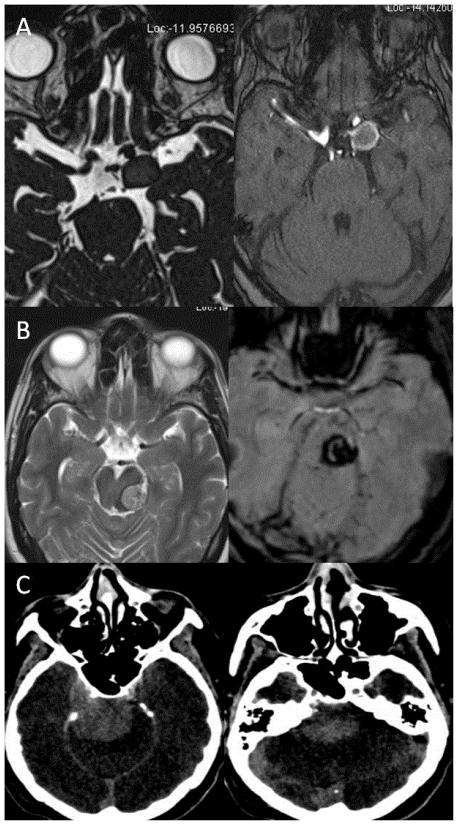

Figure 15A to C: A- Left PCOM aneurysm compressing 3rd nerve(presented with pupil involving ptosis), B shows isolated 4th nerve palsy due to compression by extra-axial cavernoma and C shows clival meningioma causing 6th nerve palsy.

Tumors like schwannoma of 3rd, 4th and 6th cranial nerves are rare and on imaging appears similar to other types of schwannomas. They appears isointense to brain and frequently shows homogenous bright contrast enhancement but can be heterogenous and appears along the course of the involved nerve [41-43] (Figure 16).

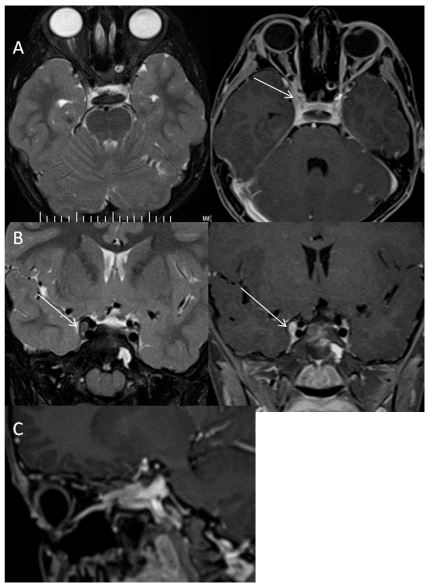

Figure 16A to C: A- axial T2 FS and post contrast T1 axial , B- coronal T2 FS and post contrast T1 and C post contrast T1 sagittal images showing enlarged and enhancing right 3rd nerve (long white arrow) in a child presenting with progressive ptosis due to 3rd nerve palsy.

Tumours and Granulomatous lesions like cysticercal cyst, tuberculomas etc. can involve cranial nerve nuclei, causing ocular movement abnormality (Figure 17). Table 3 shows motility disorder with etiology and localisation of cranial nerve palsies.

Motility disorders	Etiology/localisation
Isolated pupil involving 3rd nerve palsy	Posterior communicating artery aneurysm
Pupil sparing 3rd nerve palsy	Microvascular ischemia of nerve
Weber syndrome (3rd nerve palsy with hemiparesis)	Anterior midbrain
Benedicts syndrome(3rd nerve palsy with contralateral tremors)	Red nucleus and 3rd nerve fascicles
Isolated 4th nerve palsy	Dorsal midbrain or anterior medullary velum
Isolated 6th nerve palsy	Pons (demyelination /ischemia)
Bilateral 6th nerve palsy	Raised intracranial pressure
3rd, 4th and 6th nerve palsy	Cavernous sinus
3rd, 4th and 6th nerve palsy with optic neuropathy	Orbital apex
Internuclear ophthalmoplegia	Median longitudinal fasciculus
Multiple cranial nerve palsies	Subarachnoid space lesions

Table 3: Motility disorders due to cranial nerve palsies and their localisation.

Abnormalities of extraocular muscles (EOM)

Brown syndrome: Brown syndrome is rare abnormality affecting superior oblique tendon and trochlea leading to limited elevation of the affected eye. It can be congenital or acquired. Acquired cases are either due to trauma or surgery, which causes scarring/avulsion of trochlea. When congenital, the superior oblique muscle belly is also atrophic as may be demonstrated by high resolution MRI [44] (Figures 17 and 18).

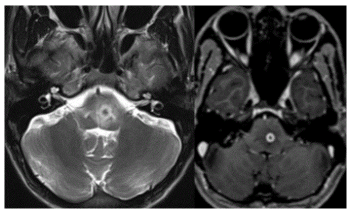

Figure 17: Isolated lateral rectus palsy due to ring enhancing lesions at lower pons.

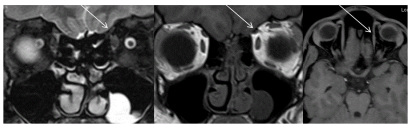

Figure 18: Child with congenital squint. Isolated congenital left superior oblique muscle atrophy (long white arrow) Brown syndrome.

Isolated myositis of EOM: Isolated myositis of extraocular muscle is usually a subgroup of idiopathic orbital inflammatory syndrome. It commonly affects the recti mucles (inferior rectus is least affected). The involvement of EOM in inflammatory diseases is due to its high blood flow and high mitochondria which makes these muscles prone to increased circulating inflammatory cells. On MRI, the affected muscle belly is bulky and swollen with hyperintensity on T1 and T2 weighted images as compared to normal muscle and also shows bright contrast enhancement [45,46] (Figure 19).

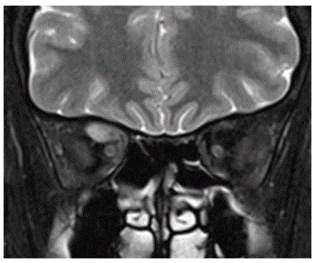

Figure 19: Isolated myositis of right superior rectus muscle which is swollen and hyperintense on FS T2 coronal image and patient presented with ptosis.

Cysticercal cyst affecting EOM: Isolated EOM can be infested with cysticercal cyst. There is fusiform enlargement of the muscle with T2 hyperintense cystic lesion with eccentric scolex within, well demonstrated on MRI [47] (Figure 20).

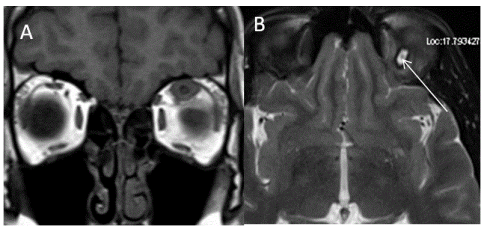

Figure 20: Left superior rectus cysticercal cyst with scolex seen in axial T2 FS image in B (long arrow).

Isolated metastasis to extraocular muscles: Isolated metastasis to EOM muscles is extremely rare. It can be initial presentation of metastases. The most common primary can be from breast and lungs but reports from thyroid and gastric malignancies are also reported. It mostly affects muscle belly with medial rectus being most commonly involved muscle. In 17% cases it can be bilateral [48-50] (Figure 21).

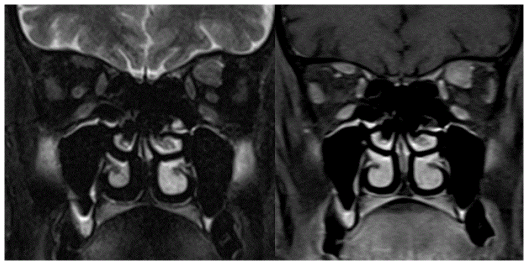

Figure 21: Left superior rectus appears swollen and brightly enhancing. Case Of metastatic adenocarcinoma.

Diseases causing proptosis and external ophthalmoplegia

Proptosis is usually due to enlargement of extraocular muscles, presence of tumor or space occupying lesion in orbit and vascular engorgement. Similarly these patients may present with ophthalmoplegia due to involvement of multiple muscles in the orbital apex or due to involvement of multiple nerves at the apex or cavernous sinus. Patient may present with redness of eyes, chemosis and excessive watering. These patients need dedicated studies concentrating at orbital apex and cavernous sinus.

Orbital psuedotumor: It is an inflammatory non-granulomatous disease process affecting globe, orbit with extraorbital extension. It can manifest as myositis, dacryoadenitis, periscleritis, trochleitis, perineuritis and tumor-like masses. It can present either as mass in orbital apex (focal or diffuse form) or as myositis. It can extend into cavernous sinus, Meckel's cave, middle cranial fossa, petrous apex, pterygopalatine fossa, infratemporal fossa etc. Imaging features depends on the type of affection. It appears as T2 hypointense and T1 isointense to gray matter with bright contrast enhancement. In cases of extraorbital extensions, it can show bright dural enhancement. This disease usually responds well to steroid treatment [51] (Figure 22).

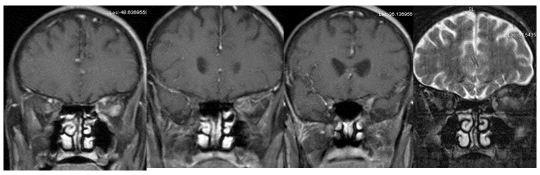

Figure 22: T1 Coronal FS post contrast and T2FS coronal images showing enhancing Bulky left extra ocular muscles with enhancement extending to tendons and orbital apex suggesting orbital pseudotumor. Patient presented with external ophthalmoplegia.

Grave's ophthalmoplegia: Thyroid eye disease manifests with enlarged muscles causing ophthalmoplegia. It usually affects bilateral muscles and sometimes causes optic neuropathy due to crowding of enlarged muscles at the apex. On CT, the muscles appear bulky and enlarged with sparing of tendons and bowing of medial orbital wall [52]. On MRI the muscles appears enlarged with hyperintensity in acute phase. In chronic burnt out phase the muscles undergo fibrosis and does not show stretching on Dynamic MRI [53] (Figure 23).

Figure 23A and B: A-coronal T2 image showing residual pituitary adenoma in a case of acromegaly and B showing bilateral swollen and enhancing extra-ocular muscles on T1 MPR post contrast image. Patient presented with proptosis.

Tolosa hunt syndrome: Tolosa hunt syndrome is important cause of painful ophthalmoplegia showing good response to steroid therapy. The disease is seen affecting cavernous sinus and or orbital apex. On MRI, the affected cavernous sinus is enlarged and shows lateral convexity. The lesions are isointense to muscle and hypointense to fat on T1W and isointense to fat on T2W images. On contrast administration, it shows bright enhancement. On MRI, the important differential diagnosis includes pseudotumor, sarcoidosis, meningioma and lymphoma as all these lesions shows T2 hypointensity [54] (Figure 24A).

Wegeners granulomatosis: It is necrotising granulomatous disease affecting paranasal sinuses, nasal cavity and orbit. Intracranial involvement shows meningeal thickening, which can be seen extending to cavernous sinus. It also shows T2 hypointensity on MR imaging. There can be remote T2 hyperintense signal changes in brainstem and cerebral white matter [55] (Figure 24 B).

Tuberculous meningitis: Tuberculosis can involve cavernous sinus and basal meninges, which appear hypointense on T2 and can be nodular in contour. Bright enhancement is seen on post contrast studies. This picture is identical to sarcoidosis [56] (Figure 24C).

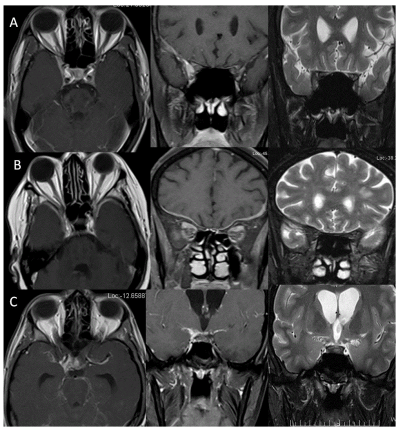

Figure 24A to C: A- Tolosa Hunt syndrome showing contrast enhancement along right anterior Cavernous sinus and orbital apex (patient presented with painful ophthalmoplegia.) B- Wegeners granulomatosis showing dural enhancement along anterior cranial fossa and involving bilateral cavernous sinus region (patient presented with bilteral 3rd nerve palsy) C-Tubercular meningitis involving basal cisterns and right cavernous sinus.

Sarcoidosis: Sarcoidosis shows involvement of dura mater which extends to cavernous sinus. The lesions are dark on T2 with bright contrast enhancement. Other features includes intraprenchymal nodular lesions with edema, thickening of cranial nerves, periventricular lesions and hypothalamic involvement [57].

Fungal infection: Invasive fungal infections are commonly due to mucormycosis and aspergillosis, and are mainly seen in immunocompromised patients. These occur by extension from surrounding paranasal sinus infection with or without bony destruction. They can lead to cavernous sinus thrombosis with wall thickening and narrowing of the cavernous ICA. On MRI, fungal infections are hypointense on T2 and T1 due to ferromagnetic and calcium substance and can show mass-like lesions or dural enhancement [58,59] (Figure 5B).

Hypertrophic pachymeningitis: It is rare disease which causes diffuse dural thickening and can involve cavernous sinus. It is one of the cause of cavernous sinus thrombosis and parenchymal edema [59,60].

Cavernous sinus thrombosis: Thrombosis of cavernous sinus is seen due to infection extending from nasal cavity, paranasal sinuses or face. On MRI, there are filling defects in cavernous sinus due to clot. Indirect signs include enlargement of cavernous sinus with dural enhancement lateral to cavernous sinus extending to tentorium, enlargement of superior ophthalmic vein and exophthalmos [59,61] (Figure 25).

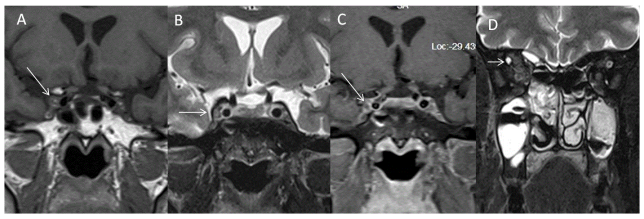

Figure 25A to D: Case of right side cavernous sinus thrombosis secondary to fungal infection of paranasal sinus presenting with chemosis and redness. Subtle T1 hyperintensity in A and T2 hypointensity in B in right side cavernous sinus with filling defect on post contrast T1 Coronal image in C (long arrow). Loss of flow void on T2 coronal in superior ophthalmic vein on right side in D (short white arrow) suggesting thrombus.

Cavernous sinus aneurysm: Cavernous ICA aneurysms cause symptoms due to mass effect, inflammation and rupture into cavernous sinus leading to carotid cavernous fistula. 5% of these are giant aneurysms. On imaging, these appear as flow voids in the large aneurysm on SE sequences and mixed intensity if the aneurysm is partially thrombosed [59,62,63] (Figure 26A).

Carotid cavernous fistula (CCF): CCF is abnormal communication of carotid artery and cavernous sinus. It can be direct or indirect through dural branches of ICA. Most common cause of direct CCF is trauma. On imaging the cavernous sinus is enlarged in size with multiple flow voids and enlarged superior ophthalmic vein. On MR angiography, flow related enhancement can be seen in the cavernous sinus. Associated findings like orbital fat stranding, enlarged veins and muscles with proptosis can be noted in affected eye. 3D FISP sequence shows sensitivity of 83% and specificity of 100% to accurately diagnose shunt flow [59,64] (Figure 26B).

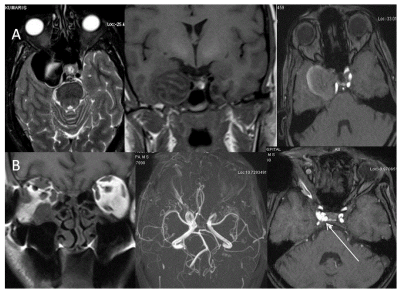

Figure 26A and B: A-Patient presented with external ophthalmoplegia. Large right cavernous segment ICA aneurysm with flow void on T2 B- right side cavernous carotid fistula with congested swollen right extra-ocular muscles with fistula seen on MR angiogram source images (long white arrow). Patient presented progressive chemosis, redness and mild proptosis of the eye.

Tumors affecting cavernous sinus: Schwannomas are frequently seen affecting cavernous sinus, of which 5th nerve schwannoma is most common. Small lesions show bright homogenous contrast enhancement and large tumors are heterogenous. They follow the course of the nerve. These lesions are T1 isointense and T2 hyperintense. Plexiform variety shows fusiform enlargement of the

nerve with heterogenous contrast enhancement. Cavernous sinus haemangiomas are bright on T1 and T2 with progressive intense contrast enhancement and attached to outer wall of cavernous sinus. Meningiomas arise usually from lateral wall of cavernous sinus and is hypo to isotense to gray matter on all sequences and on contrast they show intense contrast enhancement with dural tail sign and narrowing of the ICA. Other tumors that invade cavernous sinus are pituitary adenoma, metastases, nasopharyngeal carcinoma, sphenoid sinus carcinoma, chordoma, chondrosarcoma, lymphoma, leukemia and perineural spread of the nasopharyngeal tumors [59] (Figures 27 and 5C).

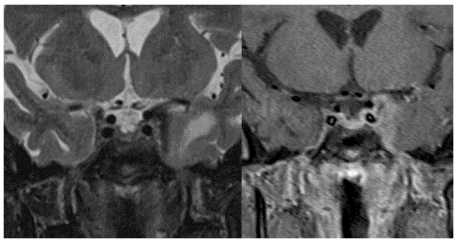

Figure 27: Patient with sudden onset 3rd and 4th cranial nerve palsy. Left side cavernous sinus enhancing lesions with involvement of clinoid process – metastasis from breast carcinoma.

Nystagmus

Nystagmus is oscillating rhythmic movement disorder of the eyes. It can be congenital or acquired. There are different types of acquired nystagmus which can be localised precisely on imaging of brain.(Table 4) So one must be aware of type of nystagmus for proper localisation of the lesion [65] (Figure 28).

Type of nystagmus	Localisation
Sea saw nystagmus	Midbrain / parasellar region
Downbeat nystagmus	Cervicomedullary region
Gaze evoked nystagmus	Vestibular/ cerebellum
Upbeat nystagmus	Pontomedullary junction / cerebellum
Rebound nystagmus	cerebellum
Periodic alternating	Congenital / chiasm
Spasmus nutans	Craniocervical junction
Dissociated nystagmus	Median longitudinal fasciculus

Table 4: Localisation of nystagmus.

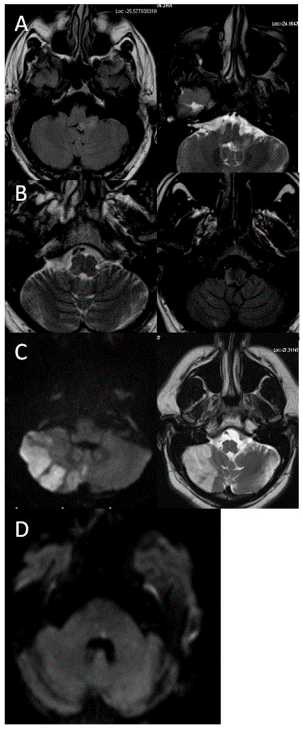

Figure 28A to D: A left inferior cerebellar lesion in patient presenting with sea Saw nystagmus B- right lateral medullary acute infarct in patient with downbeat nystagmus C- Acute right cerebellar infarct in patient of gaze evoked nystagmus D-Patient of internuclear ophthalmoplegia with Small infarct in posterior pons (medial longitudinal fasciculus) Ipsilateral decreased adduction and contralateral abducting Nystagmus.

Trauma

Trauma can cause vision loss either due to injury to globe and optic nerve, or can cause ocular movement disorder due to cranial nerve injury. Muscle entrapment in fracture fragment can also cause restricted eye movements following trauma.

Traumatic optic neuropathy: Trauma to optic nerve can be direct or indirect. Direct trauma to optic nerve can cause avulsion or transection of optic nerve. There can be optic nerve sheath haemorrhage or intra-orbital hematoma. Indirect trauma to optic nerve can be due to fracture of optic strut with fracture fragment causing compression of the optic nerve. Damage to the optic nerve can also be due to shearing/traction stresses at the optic canal as it is fixed to the dural fold [66] (Figure 29).

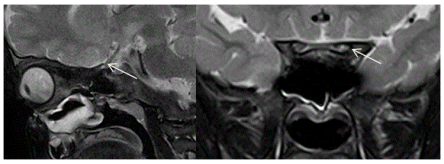

Figure 29: Post traumatic complete unilateral blindness. Traction trauma of left optic nerve at foraminal level (white thin arrow) showing hyperintense signal changes.

Rarely trauma can cause isolated or combined 3rd, 4th or 6th cranial nerve palsy usually due to stretching. 3rd cranial nerve palsy is usually associated with severe head injury, 4th nerve palsy associated with intermediate head injury and 6th nerve palsy associated with minor head injury [67-69] (Figure 30).

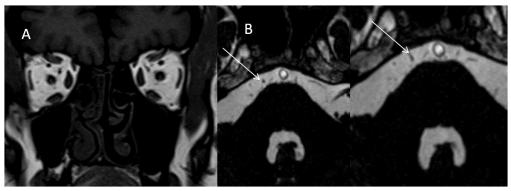

Figure 30A and B: A- T1 coronal showing bilateral lateral rectus atrophy however discontinuity noted in right 6th nerve, post traumatic injury (long arrow in B CISS 3D image).

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