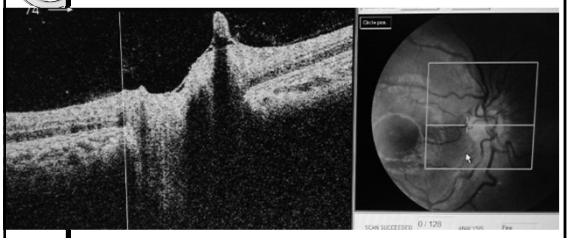
Bilateral Disc Edema

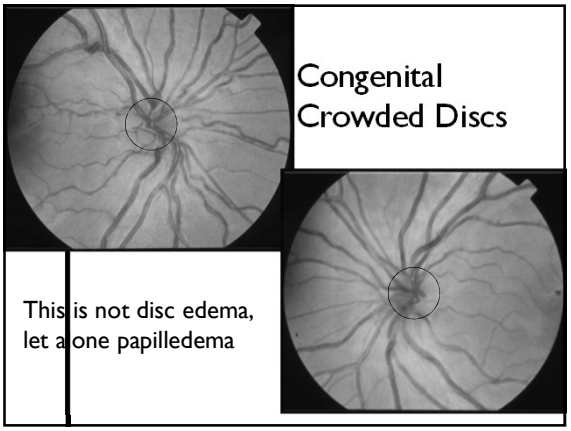
[From benign to fatal]

Dr. Satya Karna

Prominent but not edematous nerve fibre layer RE

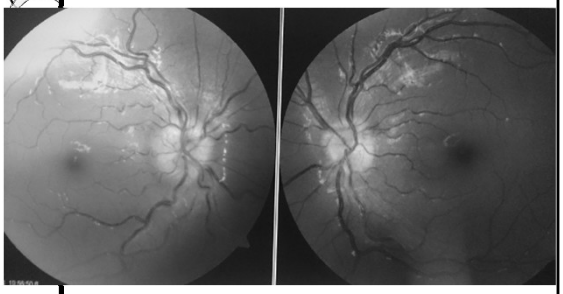


Congenital Crowded Discs

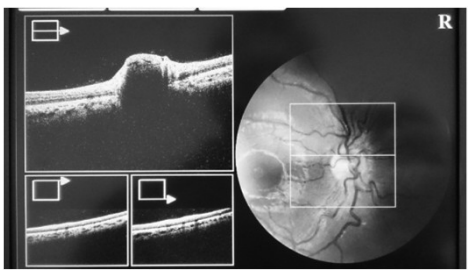


This is not disc edema, let alone papilledema

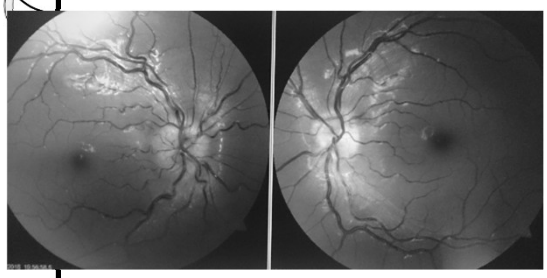
16 yr old girl, MRI, MRV normal



Prominent but not edematous nerve fibre layer RE



Is this disc edema or not?

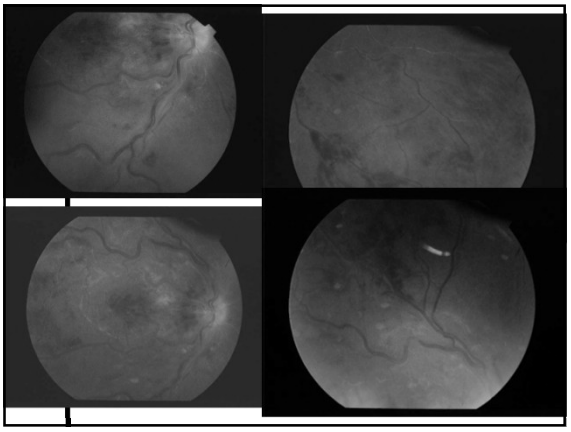


Case 1

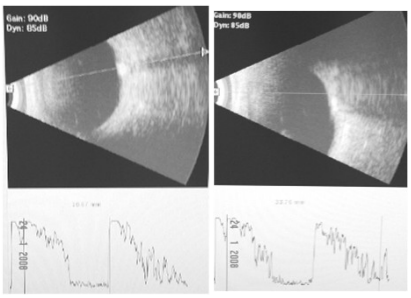
- 24 yrs/Male with sudden DOV RE 4 months ago
- Vision RE -FC 1/2m, LE - 6/6
- Right RAPD 2+ was detected
- Fundus examination :Rt. CRVO with CME and ONH Drusen B/E

Investigations

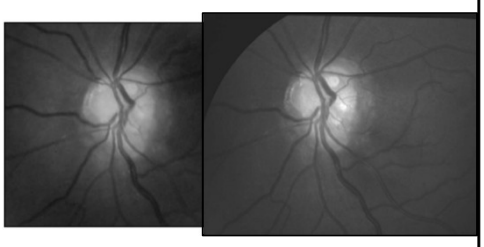
- BP: 160/90 mm Hg, earlier – 180/130 mmHg
- S. Adrenaline , Noradrenaline: WNL
- Thyroid profile: WNL
- ECG: WNL
- RBS: 87 mg/dl
- Lipid Profile: WNL
- Renal doppler - Normal

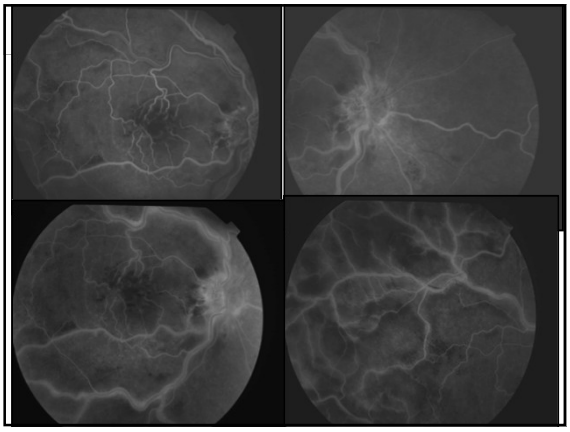


B scan



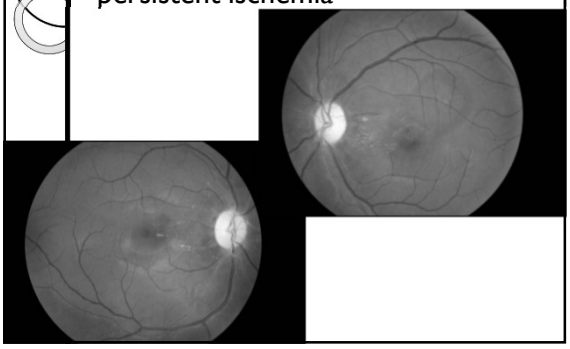
Fundus photo LE





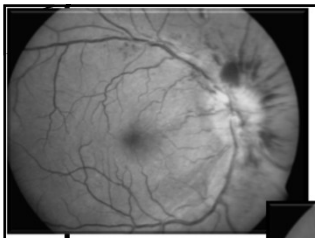
One eye has true disc edema
and
the other a pseudo disc edema!

Hypertensive retinopathy – persistent ischemia



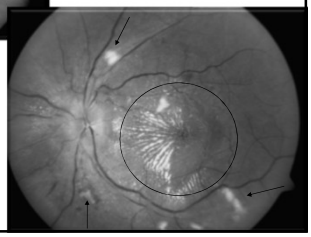
Middle aged male

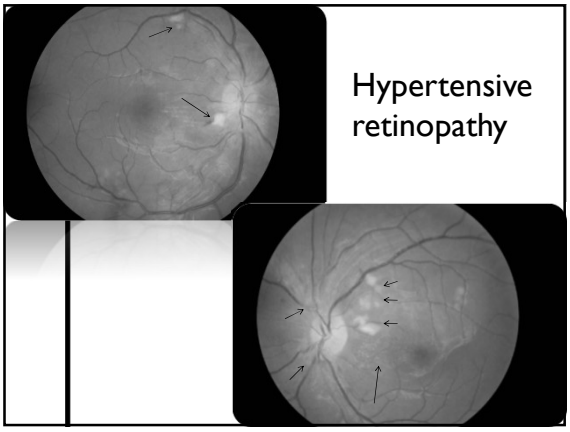
- Vision 6/9 BE
- BP – 180/125 mm Hg
- Persisitent and elevated blood pressure leads to AV nicking, CWS, retinal hemorrhages, disc edema
- Vasoconstriction and choroidal ischemia leads to disc edema
- High risk of stroke, heart attack and death.



HT retinopathy

- Vision RE 6/9, LE 6/60

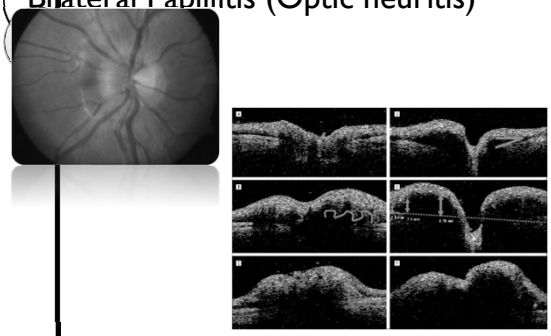




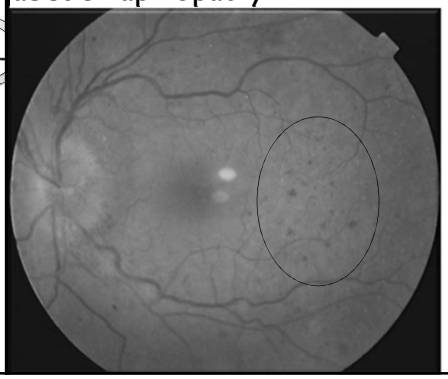
Hypertensive retinopathy

- Bilateral loss of vision in a 12 year old child

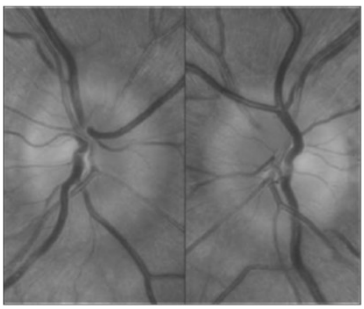
Bilateral Papillitis (Optic neuritis)



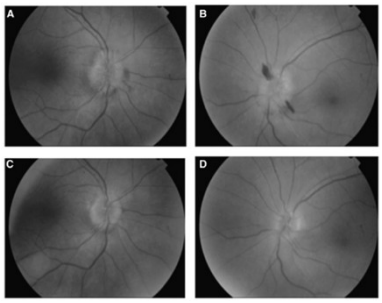
Diabetic Papillopathy



Bilateral papillitis in a child



Diabetic Papillopathy OU



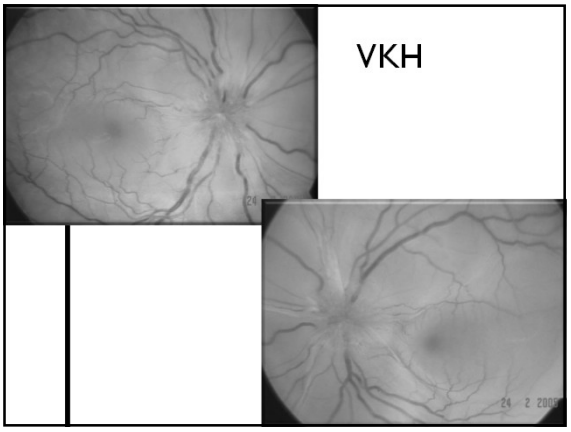
Childhood Optic neuritis

- Loss of vision both eyes
- Headache, sometimes pain on eye movements
- Usually postinfectious or post immunisation
- Steroid responsive and steroid dependant also

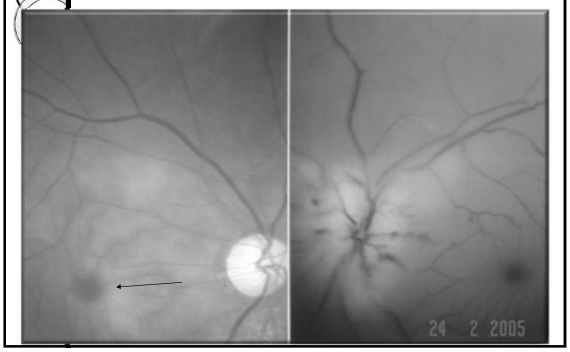
Diabetic papillopathy

- Mild or no visual loss
- Bilateral in 50% cases
- Persistent disc edema
- Good visual outcome
- More common in young patients with insulin dependant diabetes
- Associated diabetic retinopathy

VKH



GCA with AAION and CRAO



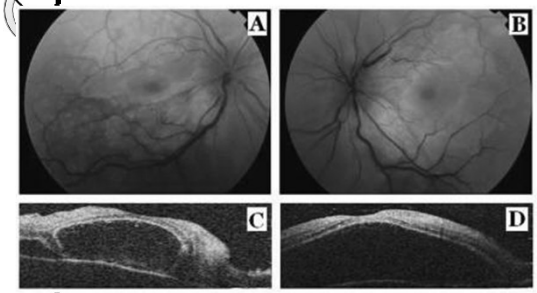
VKH

- Bilateral painful loss of vision
- Disc hyperemia with posterior uveitis, serous retinal detachments
- Anterior uveitis with mutton fat KPs
- Tinnitus, headache, vitiligo, alopecia
- Significant response to steroids, if started on time.

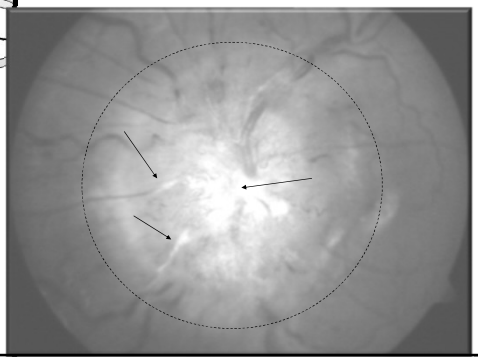
GCA

- Rapid sequential bilateral loss of vision
- AAION, CRAO, Ocular ischemic syndrome
- Pale disc edema
- Headache, jaw claudication, calf pain, fever
- Poor visual prognosis

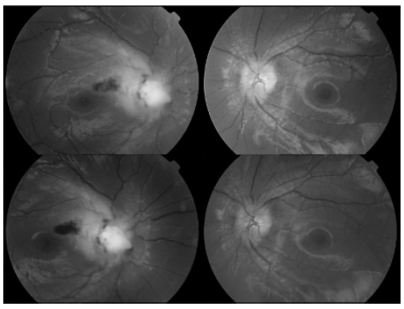
VKH with macular edema



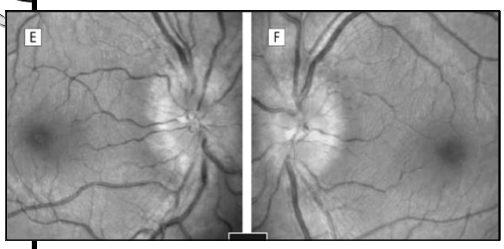
Infiltrative Leukemic optic neuropathy



Infiltrative Optic Neuropathy



Bilateral Chikungunya papillitis



Infiltrative optic neuropathy

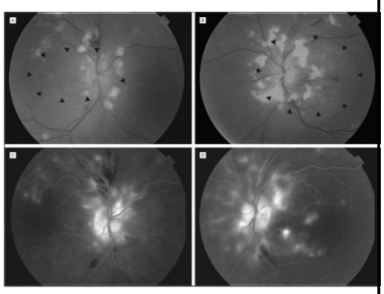
- Sudden loss of vision
- Usually advanced CNS involvement of leukemic disease
- Sarcoidosis – may be optic neuritis, associated with vasculitis and candle wax drippings.

25 year old male

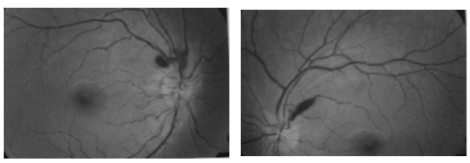
- Sudden, painless, blurring of vision OU
- Vn 6/24 OU
- Pupils sluggish OU
- central scotomas

Infectious optic neuropathy

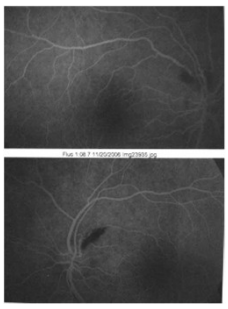
- HIV
- Dengue
- Hepatitis vaccination
- Chikungunya



bilateral hyperemic discs with flame shaped hemorrhages



FFA shows normal retinal and choroidal fluorescence



Investigations in bilateral disc edema

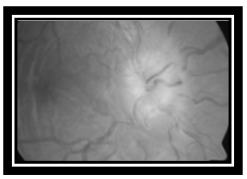
- OCT ONH
- Ultrasound for Drusen
- CT scan for Drusen
- MRI for papillitis, VKH, infiltrative optic neuropathy
- Temporal artery biopsy in AAION

Nutritional Optic Neuropathy

- Poor appetite
- Thinly built
- Vit B12 deficiency (187-1059 pg/ml)
- Hb – 12 gm%
- Occasional alcohol and smoking habits
- Vision recovered very well over 3 months with intramuscular and oral multivitamins and improved diet

Evaluate

- Detailed history
- Check for RAPD
- BCVA
- Color vision
- Study morphology
- BP check
- Visual fields
- OCT
- Fundus Photo



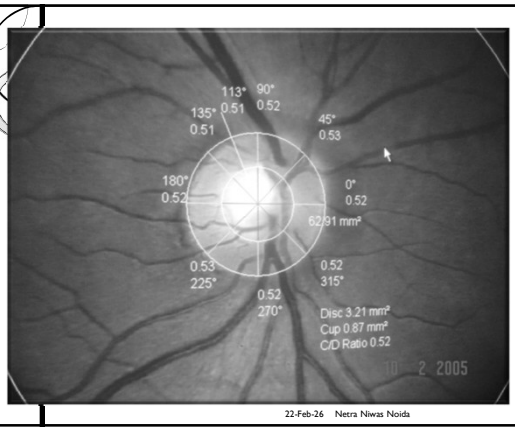
Investigations in bilateral disc edema

- Blood Pressure
- VFA for AION, papillopathy, papillitis, nutritional optic neuropathy
- CBC, FBS, PPBS, ESR, CRP, B12 levels
- HIV, Dengue, Hepatitis serology
- FFA for Diabetes, HT, GCA, VKH

10 Signs of True Disc Edema

5 Mechanical Signs
- Loss of cup
- Elevation of disc
- Blurring of margins
- Nerve Fiber Layer Edema
- Folds:
  - Retinal (Paton's)
  - Choroidal

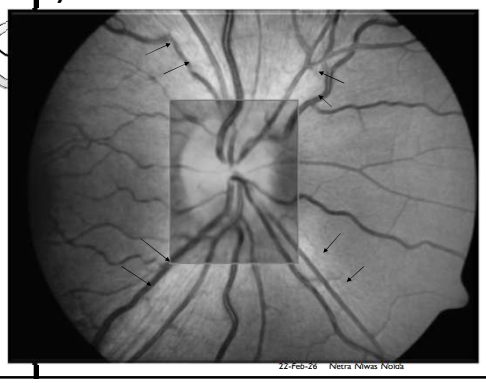
5 Vascular Signs
- Hyperemia of disc Venous congestion
  - Vein dilation
  - Vein tortuosity
- Peripapillary hemorrhages
- NFL infarcts (CWS)
- Exudates



Don't Use the Term Papilledema Unless You Mean It

- Bilateral Disc Edema (usually)
- Clinically indicated by other signs and symptoms of increased ICP
- Laboratory confirmation (like MRI and LP)
- Papilledema requires urgent workup

Early Disc Edema

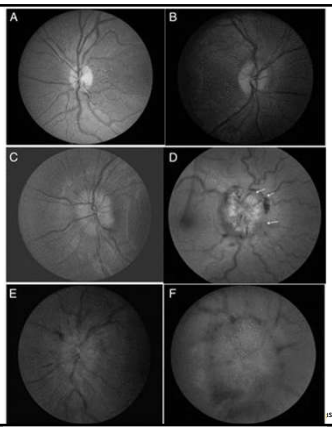


Frisen grades of papilledema

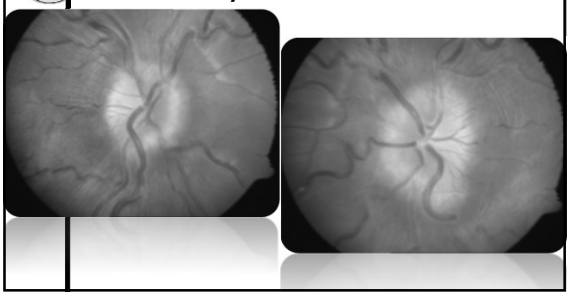
- Grade 0. There is no halo of obscuration of the peripapillary nerve fiber layer.
- Grade 1. There is a C-shaped halo of retinal nerve fiber layer edema obscuring the peripapillary retina.
- Grade 2. The halo is now circumferential; there is no temporal gap and no vessel obscuration.
- Grade 3. Major vessels are obscured by edema as they leave the disc.
- Grade 4. Major vessels are now obscured by edema on the optic disc.
- Grade 5. All vessels are at least partially obscured by edema.

Aetiology of Disc Edema (Bilateral)

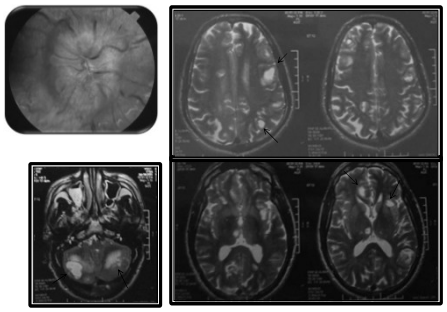
- Diabetic Papillopathy
- Papilledema
- Pseudopapilledema
- Hypertensive Retinopathy



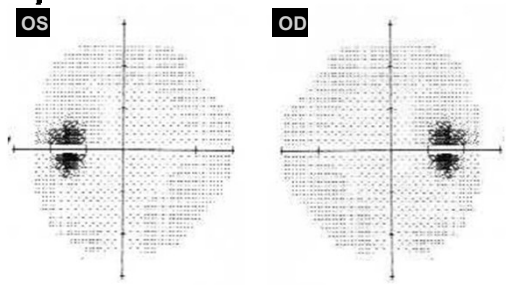
Papilledema
Grade 2-3 Right eye,
Grade 3 Left eye

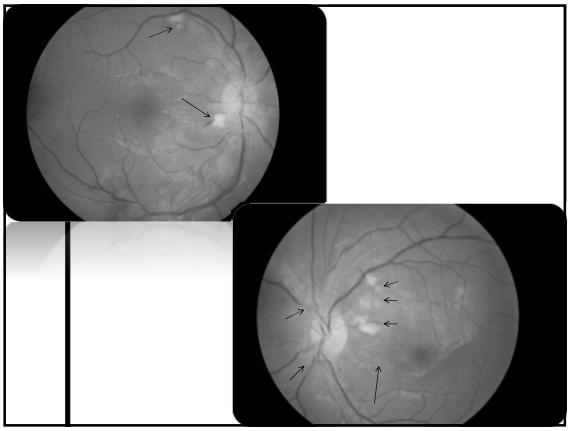


Papilledema with Multiple cerebral and cerebellar metastasis

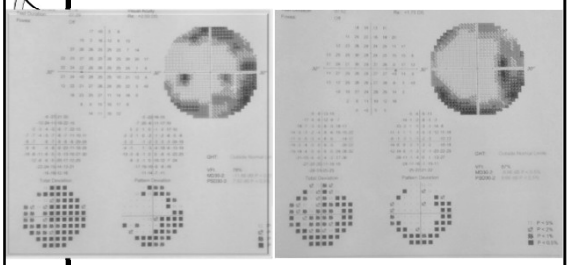


Enlarged Blind Spots



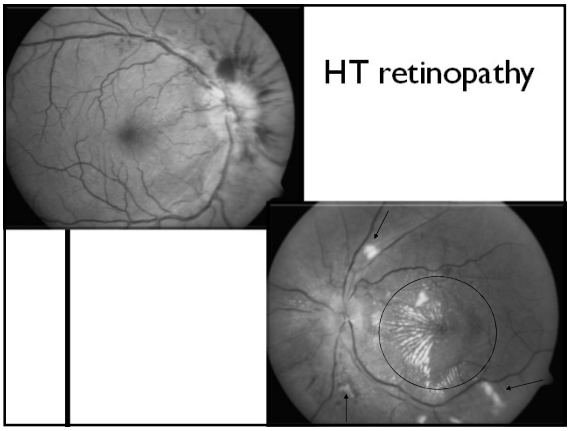


Persistent papilledema leads to constricted visual fields



22-Feb-26 Netra Niwas Noida

HT retinopathy



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CATCH THE CORRECT SIGNS IN TIME IN NEUROOPHTHALMOLOGY

Some diseases of the brain presenting with visual symptoms are life threatening and need urgent management.

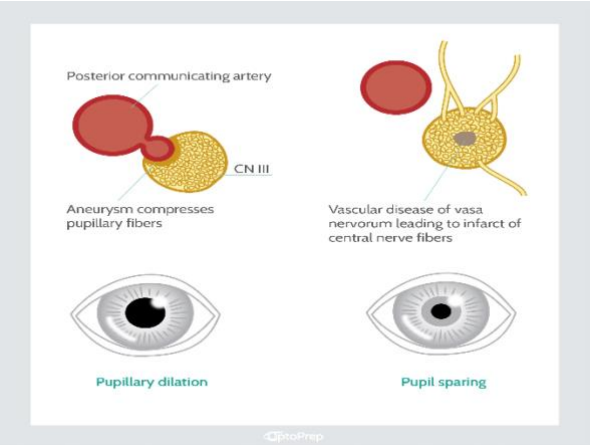
When to panic immediately:

- Sudden vision loss (painful or painless)
- Transient monocular vision loss
- Acute diplopia with pupil involvement
- Bilateral disc edema
- Visual field respecting vertical meridian
- Horner's syndrome + pain

Pupil-Involving Third Cranial Nerve Palsy: Think the Worst First

"A pupil-involving third cranial nerve palsy is one of the true emergencies in neuro-ophthalmology," That's because it may signal an aneurysm, which could rupture and lead to a subarachnoid hemorrhage—a potentially fatal event.

It may have varied presentations, including ptosis, diplopia, ocular movement disturbances, and pupillary abnormalities. The classic "rule of the pupil" states that when aneurysms compress the oculomotor nerve, the iris sphincter will be impaired, leading to a sluggishly reactive or dilated pupil. If the pupil is entirely spared in the setting of a complete ophthalmoplegia and ptosis, the oculomotor nerve palsy is usually due to a local infarction.



Painful 3rd Nerve Palsy + Dilated Pupil

Rule out aneurysm → risk of Subarachnoid Haemorrhage

Pearl

Parasympathetic fibers are superficial → compressed first.

Immediate CTA/MRA.

Managing Third Cranial Nerve Palsy

If the diagnosis is an aneurysm, the patient is immediately admitted for neurosurgical clipping.

Anterior Ischemic Optic Neuropathy: High Stakes and Quick Decisions

Encountering a patient with signs of anterior ische-mic optic neuropathy tests the mettle of any ophthalmologist. The clinician must be able to quickly determine whether the patient has arteritic

anterior ischemic optic neuropathy (AAION) or nonarteritic anterior ischemic optic neuropathy (NAION).

The key findings that point to a diagnosis of AAION:

- Severe vision loss to counting fingers or worse
- Chalky white swelling of the optic nerve (compare with hyperemic swelling in NAION)
- Evidence of retinal ischemia (i.e., cotton-wool spots or retinal artery occlusion)
- Abnormalities in perfusion of the choroid on fluorescein angiogram
- Abnormal exam of the temporal artery under the skin of the scalp

Arteritic AION

Due to Giant Cell Arteritis

Red flags

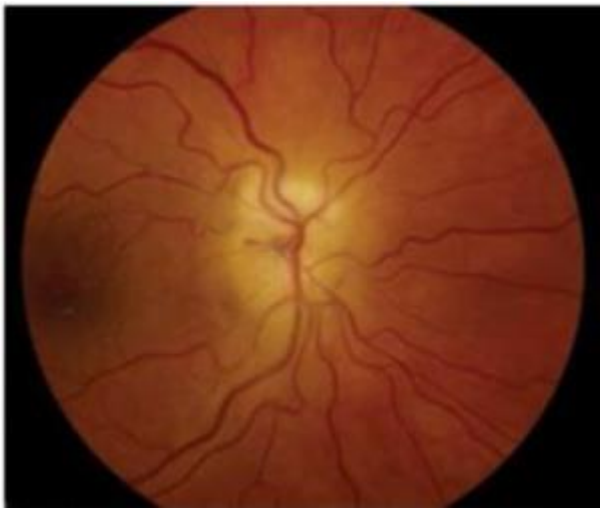
Age >50

Headache

Jaw claudication

Pale swollen disc

Fellow eye can go blind within days.



Action

Start IV steroids immediately.

Do NOT wait for biopsy.

Pituitary Adenoma: Recognize the Visual Field Patterns

The good news is that pituitary adenomas do not grow quickly, and they're usually benign .The patient may come with headache, hormonal changes, or visual disturbance.

Usually they are asymptomatic but they can disturb vision and even lead to blindness by putting pressure on the chiasm or other parts of the visual system in that area, such as the optic nerve and optic tracts.

Visual field testing is essential. “Automated perimetry is a must,” It is also important to assess visual acuity, pupils, and optic disc appearance.

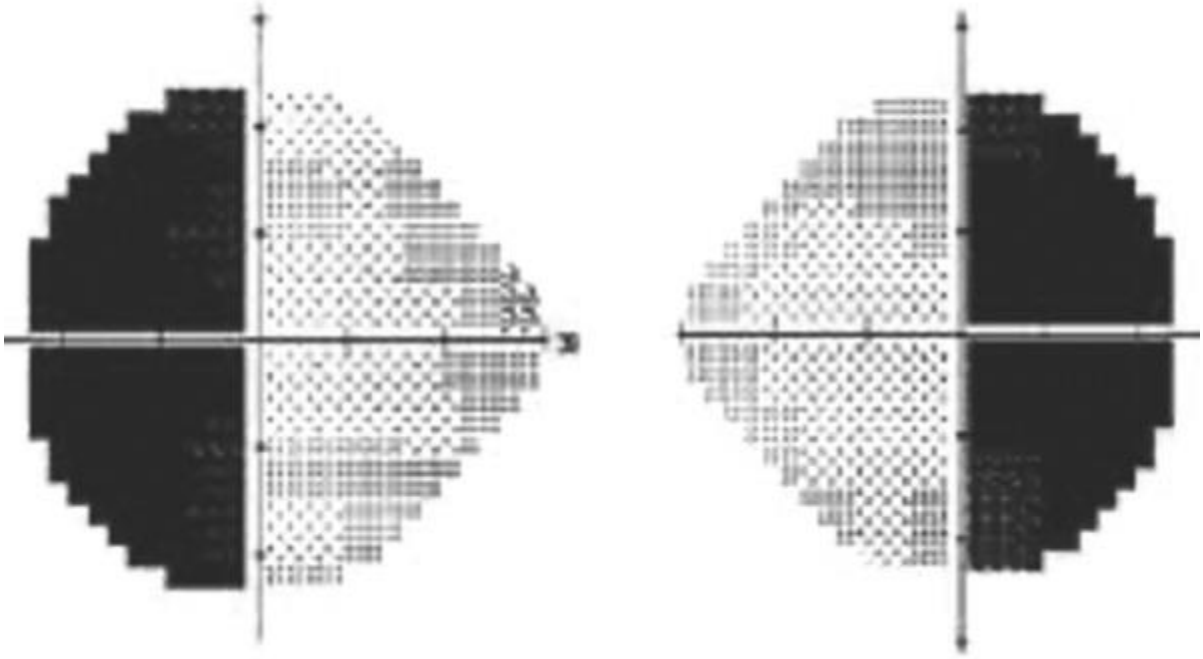
Bitemporal defect is a classic finding “But remember that pituitary tumors can present in more ways than just the classic bitemporal visual field defect,” he said. For example, in a nonclassic visual field, one side may look bad, while the other side shows only a mild defect. “It’s still a bitemporal field defect, but not complete. The asymmetry can throw people off. In some cases, one might even see a unilateral temporal defect if the compression is occurring at the distal optic nerve just before the chiasm.

Another variation of visual field sometimes seen in pituitary adenoma is the so-called “junctional scotoma,” or anterior chiasmal syndrome. In this situation, there is central loss of vision due to optic nerve compression in one eye and temporal loss in the other eye due to chiasmal compression.

The visual field does not cross the vertical midline (referred to as respecting the vertical midline). If you see a visual field which respects the vertical midline, it is worth arranging some imaging of the brain, such as a coherence tomography (CT) or magnetic resonance imaging (MRI) scan.

Managing Pituitary Adenoma

If the ophthalmologist discovers the tumor, the next stop is referral to a neurosurgeon. Not all pituitary adenomas need to be removed; medication, radiation, and watchful waiting are other management options.



A visual field defect in the temporal aspect of both eyes which does not cross the vertical midline, highly suggestive of a non-glaucomatous lesion and suggests a lesion posterior to the eye,

Transient Monocular Vision Loss

Amaurosis Fugax = Retinal TIA

Common cause:

Carotid Artery Stenosis

Warning sign of Ischemic Stroke

⏳ Highest stroke risk: first 48 hours.

Action

Carotid Doppler + antiplatelet + stroke workup.

Painful Vision Loss

Typical Optic Neuritis

Often associated with Multiple Sclerosis

Clues

Age 20–45

Pain on eye movement

RAPD

Red desaturation

Central scotoma

Catch Early

MRI brain/orbit are done- Identify MS risk lesions

⚠️ **Red Flags for Atypical ON**

Think beyond MS:

Bilateral severe loss

No pain

Poor steroid response

Consider:

Neuromyelitis Optica

MOG Antibody Disease

Case I

• A 19 years old female presented with complaint of drooping of left lid for 6 months

O/E

• Vision – normal in both eyes

• Ocular motility

• Minimal restriction in movement on adduction, elevation

• Abduction and depression were normal

• Partial ptosis on left side, on depression, elevation,adduction of left eye, there was retraction of left lid

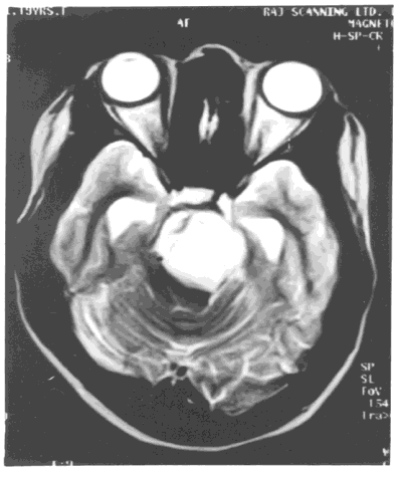
• Fundus, field normal

• Rest of the Neurologic examination normal

Clinical diagnosis of primary aberrant regeneration of 3rd nerve palsy

On investigation

• MRI –extra – axial lesion anterior to mid brain and pons. Hypointense on T1 and hyperintense T2 mainly on left side, compression of brainstem, superiorly interface between



brainstem and tumour lost Epidermoid in prepontine and

pre-mesencephalic cistern

• Per-operatively – 3rd nerve was found to be engulfed by the lesion while 4th was stretched and thinned

Epidermoid in pre-mesencephalic & prepontine cistern presenting as primary aberrant regeneration of 3rd nerve palsy

CASE 2

A 30-YEAR-OLD FEMALE PRESENTED WITH COMPLAINS OF PAINLESS PROGRESSIVE DECREASED VISION IN LEFT EYE FOR LAST 2-3 WEEKS WITH HEADACHE

SHE HAD SIMILAR RECURRENT ATTACKS FOR LAST 6 MONTHS

FIELD EXAMINATION SHOWED TEMPORAL HEMIANOPIA IN RE AND TEMPORAL HEMIANOPIA WITH CENTRAL SCOTOMA IN LE

IMAGING SHOWED **PITUATRY APOPLEXY**

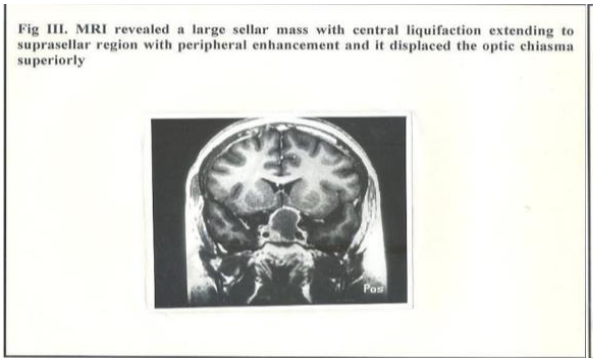


Fig III. MRI revealed a large sellar mass with central liquifaction extending to suprasellar region with peripheral enhancement and it displaced the optic chiasma superiorly

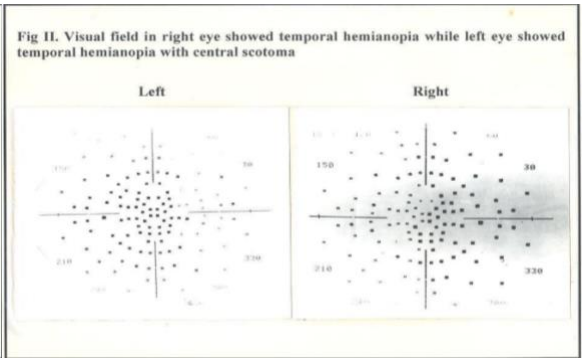
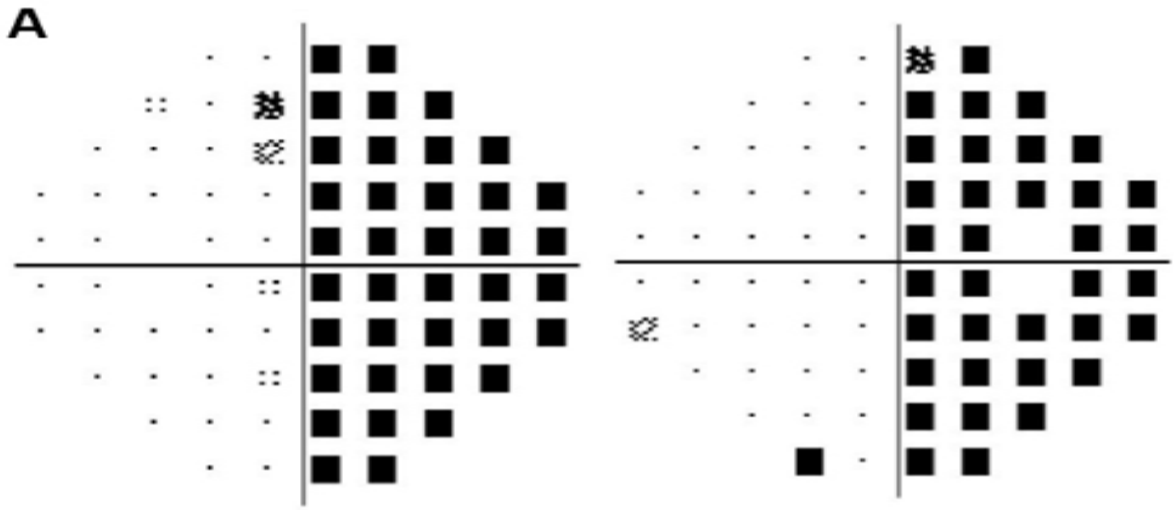


Fig II. Visual field in right eye showed temporal hemianopia while left eye showed temporal hemianopia with central scotoma

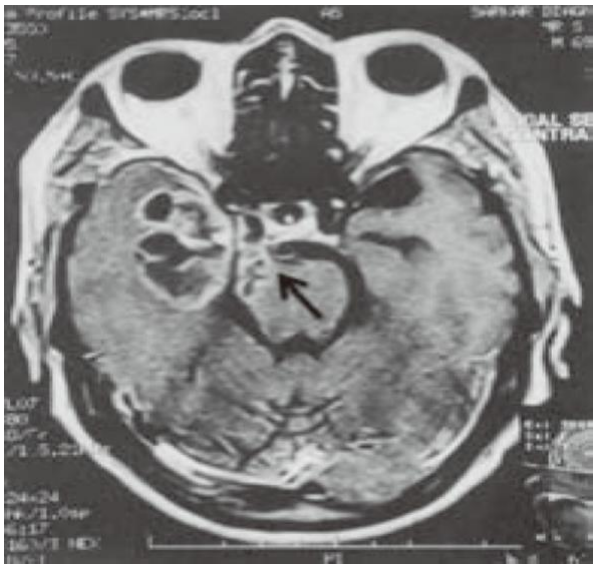
CASE 3

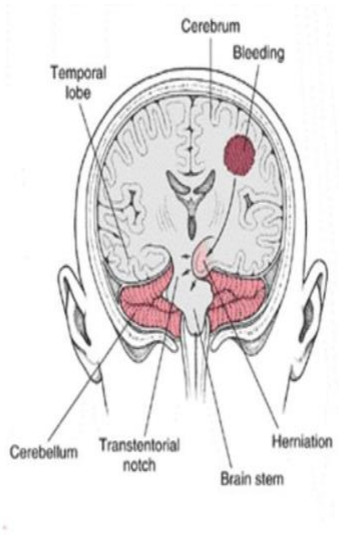
A 56-year-old diabetic man presented wit H/O dull pain in left eye with drooping of left eyelid for 2-4 weeks

O/E painful complete 3rd nerve palsy was found with mild weakness in right Lower leg



MRI – Mass in anteromedial temporal lobe extending to adjacent CS also extending posteriorly and compression left cerebral peduncle





The 5 Golden Neuro-Ophthalmology Rules

Painful vision loss → demyelination vs inflammatory

Elderly sudden loss → treat as GCA first

3rd nerve + pupil → aneurysm until proven otherwise

TMVL → stroke warning

Papilledema → image before LP

Unexpected cause and less known sign in Neuro ophthalmology

Dr Kumudini Sharma

Ocular Stroke in young requires evaluation of the heart, blood vessels of head and neck, blood work up. It is critical to identify the likely pathogenesis to prevent stroke recurrence. Common vascular causes in young are cervical arterial dissection, fibromuscular dysplasia, inflammatory vasculopathies, antiphospholipid antibodies syndrome, large artery atherosclerosis. Heart is another common source of embolism to retinal blood vessel. Retinal vasculitis secondary to TB, syphilis, Behchet and other viral etiology can cause occlusion of blood vessel but tubercular vasculitis of cervical ICA has not been reported as a cause of embolism to retinal artery as in our case.

Transverse Venous Sinus thrombosis is rare and potentially life threatening form of cerebral venous sinus thrombosis and patient presents with headache, seizure, vision changes. While serious but most patients make full recovery with prompt diagnosis and treatment. Our second case had transverse sinus thrombosis following severe dehydration. Along with headache he had pain on right side of neck along sternocleidomastoid muscle as the main symptom due to extension of thrombus in Internal Jugular Vein. So in case of Papilledema with pain in the neck one should suspect cerebral venous sinus thrombosis.

CASE 1

A 30-year-old female non diabetic and non-hypertensive presented with complaint of infero nasal field defect left eye for last 2 months. Prior to this she used to get transient blurring of vision in left eye lasting for less than one minute. On examination visual acuity was normal in both eyes, ocular motility was full and painless. Pupil in left eye had slight sluggish reaction to light. Fundus in left eye showed mild temporal pallor of disc while right eye fundus was normal. Perimetry showed normal field right eye with inferonasal field constriction to left eye with corresponding RNFL loss in left eye on OCT. Systemic examination was normal except enlarged

submandibular lymph nodes on left side which were non tender, firm, mobile .Clinical diagnosis of super temporal branch retinal artery occlusion in left eye was made . Haemogram, biochemistry, Homocysteine, coagulation and vasculitis profile were normal. Echo and ECG were also normal. To rule out Takaysu arteritis carotid Doppler was performed which revealed diffuse thickening of left CCA and left ICA, velocity of blood flow was low. CT scan cranial was normal. CT angiogram of neck and upper thorax showed mild wall thickening with severe luminal narrowing of left CCA and proximal left ICA along with infective aneurysm of descending aorta with hypo dense lobulated mass in right upper lobe with mild plural thickening. Similar lesion was seen in left lower lobe of lungs. CT angio also showed enlarged neck lymph nodes. Submandibular lymph nodes biopsy revealed caseating tubercles suggestive of granulomatous tubercular lymphadenitis with left CCA and ICA arteritis with pulmonary tuberculosis and mycotic aneurysm of descending aorta.

Patient was given a course of Anti tubercular treatment for one year. Post treatment Carotid Doppler and CTA showed normal wall and lumen of left CCA and ICA with significant resolution of cervical lymph nodes. This case has retinal artery embolism from a very unexpected cause. In work up of a case of ocular stroke along with cranial blood vessels, neck vessels including arch of aorta should be evaluated to look for the source of evaluation.

CASE II:

Case of a thirty-five-year-old hypertensive male patient presented with headache with pain in neck in the right side along the sternocleidomastoid muscle for last 1 week. Prior to this he had severe diarrhoea and vomiting and was admitted in hospital with diagnosis of severe dehydration. On examination his vision was normal in both eyes. Ocular motility and pupils were normal, while fundus showed bilateral Papilloedema and hemorrhages in both eyes. Perimetry was normal .Systemic examination was normal except tenderness along the right SCM muscle.

A diagnosis of Benign Intracranial hypertension in both eyes was made. On neuroimaging there was a thrombus in right transverse sinus extending to sigmoid sinus. Patient was treated with Diamox and an anticoagulant. The patient was very much troubled by pain in his neck, the cause of this symptom was extension of thrombus in internal jugular vein. In Walsh and Hoyt's Clinical Neuro Ophthalmology it is mentioned that papilledema with pain in neck suggest thrombosis of TVS extending IJV. Hence, in a case of Papilledema with pain in neck one should suspect venous sinus thrombosis as cause of it and ask for MRI with MRV.

Tips and tricks to solve Neuro-Ophthalmology puzzles

Dr Priyadarshini Mishra

1. Mastering the History:

The history is the most critical piece of the puzzle; if it doesn't match the exam, one or both must be re-evaluated.

Speed of Onset: Sudden, painful loss = inflammatory/vascular (e.g., optic neuritis, GCA). Slow, painless loss = tumor/compression.

Associated Symptoms:

- Headaches (worse in the morning?).
- pulsatile tinnitus or limb weakness.
- Transient visual obscuration.
- Diurnal variation in diplopia.
- Pain Factors: Pain with eye movement is a classic sign of optic neuritis, but if pain lasts longer than 3 months, alternative diagnoses should be considered.
- Any history of malignancy also deserves special attention. A new neuro ophthalmic deficit in a patient with a recent history of malignancy should be attributed to either the cancer or its treatments until proven otherwise

2. Essential Diagnostic "Tricks"

- Laterality: In practice, patients are sometimes unable to distinguish whether one eye or both eyes are involved: since the temporal visual field is larger than the nasal, patients with homonymous visual field defects often localize vision loss to the eye with the temporal field cut.
- Visual Acuity
- Colour saturation.
- Relative Afferent Pupillary Defect (RAPD).

- Horner’s Syndrome: Small pupil + Ptosis + Worse in dim light.
- 3rd Nerve Palsy: Large pupil (fixed) + Down-and-out eye + Ptosis.
- Adie’s Tonic Pupil: Dilated pupil + Poor light response + Good near response.
- Fatigue Testing (Myasthenia): If ptosis is present, have the patient look up for 2 minutes to see if it worsens, or use ice packs (ice test) to see if it improves.
- The "Double Vision" Check: Always ask if the double vision goes away when one eye is covered. If it persists, it's monocular (usually ocular/corneal); if it goes away, it's binocular (neurological).
- Bielschowsky Head Tilt Test: If vertical diplopia worsens when the patient tilts their head toward the side of the higher eye, it confirms a 4th nerve (trochlear) palsy.
- Mirror Test for Malingering: To check for nonorganic vision loss, hold a mirror in front of the "blind" eye and rock it; a seeing eye will unconsciously follow the moving reflection.
- Orbicularis Strength: In patients with drooping lids (ptosis), test the strength of the eyelid closure. Weakness here strongly suggests Myasthenia Gravis.
- Light-Near Dissociation: If pupils react poorly to light, check their reaction to a near object. A better reaction to "near" can help diagnose Argyll Robertson pupil or Adie’s tonic pupil.
- Ocular movements: Vertical vs. Horizontal: As a rule of thumb, horizontal eye movement disorders typically point to the pons, while vertical disorders point to the midbrain.
- Torsion Check: To differentiate a CN IV palsy from a skew deviation, look for eye rotation (torsion). Skew deviation often involves incyclotorsion of the higher eye, whereas CN IV palsy usually shows excyclotorsion.

3. Visual Field Interpretation

Visual Field Testing: Get a formal visual field test the same day for any unexplained vision complaint; it can localize problems to specific parts of the brain like the occipital lobe.

- Confrontation test
- “finger-to-nose” perimetry
- Neuro-ophthalmology lesions typically cause field defects that respects the vertical meridian - Chiasmal or postchaismal lesion
- Does not respect vertical meridian- Retinal or optic nerve diseases

Anatomical Localization:

Optic Nerve: Unilateral blindness/central scotoma.

Optic nerve ring scotoma connect up with blind spot at 15 degrees from fixation. Retinal ring scotoma of retinitis pigmentosa start approximately 30 degrees from fixation

Chiasm: Bitemporal hemianopia (classically pituitary tumor).

Behind the Chiasm (Tract/Radiations): Contralateral homonymous hemianopia.

"Pie in the sky" (upper quadrant) = Temporal lobe. "Pie on the floor" (lower quadrant) = Parietal lobe.

Occipital lobe:

- Marked congruity
- Macular sparing
- Very small homonymous hemianopic scotoma
- Monocular loss of unpaired temporal crescent (no counterpart in shorter nasal field) represent anterior occipital lobe.
- Quadrantic defects that stop sharply at horizontal meridian. The upper and lower occipital branches are supplied by distinctive vascular branches.

4. Other accessory Tools

- Optical Coherence Tomography (OCT): Use this to distinguish between real papilledema (swelling from brain pressure) and pseudopapilledema (like optic disc drusen).
- FFA in GCA

5. Essential Imaging and Red Flags

When to Image: New diplopia, new ptosis, unexplained vision loss, or any optic disc swelling (papilledema) needs immediate neuroimaging (MRI/MRV).

Spontaneous Venous Pulsations (SVPs): If present on fundus exam, it is highly unlikely the patient has increased intracranial pressure.

In general, trauma cases excluded, MRI remains the strongly preferred imaging modality for patients with visual complaints. You should always order intravenous gadolinium contrast unless the patient has renal failure. New onset right cranial nerve 6 palsy" is a much more helpful description to the radiologist than "double vision.

Patients with rapid onset of symptoms or known hypercoaguable disorders or who do not fit the typical pseudotumor cerebri demographic (reproductive-age, overweight women) also require a magnetic resonance venography to rule out a dural venous sinus thrombosis that could be missed on an MRI brain.

Optic Neuritis- Diagnostic dilemmas

Optic neuritis is an inflammatory disorder of the optic nerve and causes significant visual impairment. Optic neuritis (ON) has over 60 distinct causes, only one of which is multiple sclerosis (MS). This poses a challenge for its prompt and accurate diagnosis It can be demyelinating, post-viral, infectious, traumatic or due to systemic illness. It can involve any part of the optic nerve and includes neuro-retinitis, papillitis and retrobulbar neuritis.

ON is characterized by sudden onset vision loss typically unilateral and can present with periocular pain or pain on eye movement, impaired colour or motion perception. Typical clinical signs include reduced visual acuity, reduced colour vision and contrast sensitivity, relative afferent pupillary defect (RAPD). The recovery phase typically follows a similar pattern, with pain improving before visual function.

Although optic neuritis is a clinical diagnosis, ancillary investigations like optical coherence tomography (OCT), magnetic resonance imaging (MRI), visual evoked potential (VEP), cerebrospinal fluid analysis, blood biomarkers are important particularly in atypical optic neuritis. The interpretation of relevant investigations depends on their timing.

Here, we describe our stepwise diagnostic approach to ON, with illustrative cases, which mirror the clinical scenarios where clinicians must reach the correct diagnosis and classification of definite ON and atypical ON. We provide advice on avoiding pitfalls in interpreting tests, sharing clinical pearls in MS and other neuroinflammatory conditions. We highlight OCT as an important and very sensitive test that helps to make a correct diagnosis.