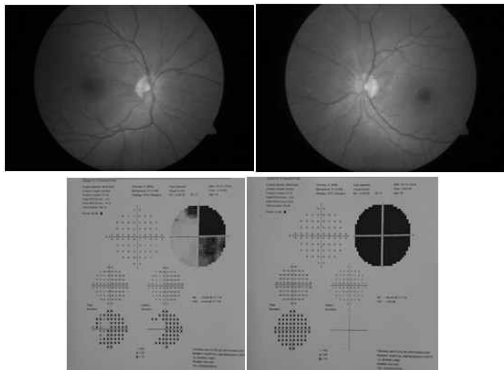


Our Patient Can't See: Is it a Neuro-ophthalmic Disorder? (Neurological Causes of Acute Vision Disturbance)

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Diagnostic Case Scenarios

Descriptive Case 1: A 19-year-old lady presented to the Neuro-Ophthalmology Services of Bombay Hospital and Medical Research Centre with Right Occipito-frontal headache with vertigo. On examination, she had Right Eye: 6/9, N6, Normal Colour Vision and Left Eye: Finger Counting 2 metre, N36 with a severely affected Colour Vision. Fundus Examination showed bilateral optic neuritis (Left More than Right). MRI Brain showed Demyelinating lesion and she was treated for **Multiple Sclerosis**.

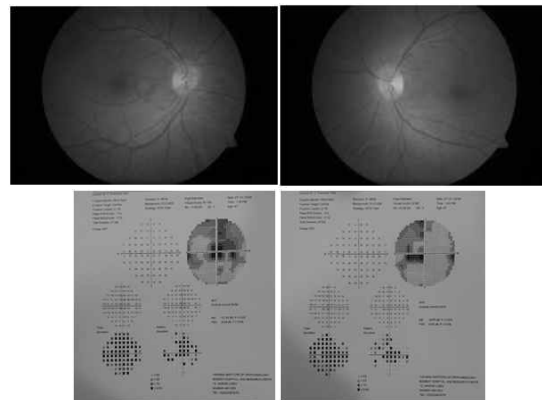


Figs. 1-4: Fundus Pictures and Visual Fields of a 19-year-old girl with bilateral optic neuritis showing a severe affection of the central 10 degree field in left eye more than right eye.

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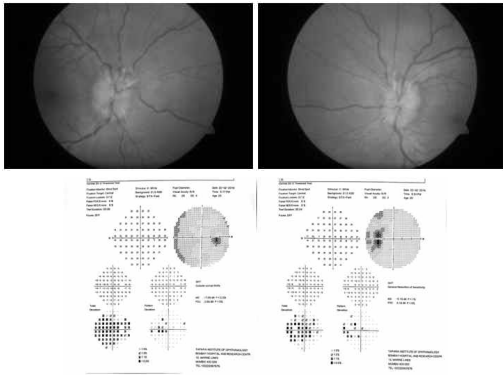
Descriptive Case 2: A 41-year-old gentleman presented to the Neuro-Ophthalmology Services of Bombay Hospital and Medical Research Centre with sudden onset bilateral painless loss of vision. He was on treatment of Koch's Lymphadenitis. MRI Brain + Orbits done was Normal.

On examination, Right Eye had a vision of Finger Counting 3 metre, N36 and Left Eye: 6/60, N36. His Colour Vision was severely affected in both eyes. Fundus examination showed mild optic nerve oedema in both eyes with a central scotoma in the visual field. He was diagnosed to have Ethambutol induced Optic Neuropathy and the only intervention required was cessation of Ethambutol to regain vision.



Figs. 5-8: Fundus Pictures and Visual Field of a 41-year-old gentleman with Ethambutol induced Optic Neuropathy with bilateral central scotomas.

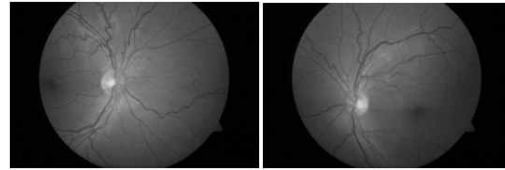
Descriptive Case 3: A 25-year-old gentleman presented to the Neuro-Ophthalmology Services of Bombay Hospital and Medical Research Centre with double vision and squinting of eyes for 10 days. On examination, he had a vision of Right Eye: 6/6, N6 and Left Eye: 6/6, N6 and Normal Colour Vision in both eyes. Fundus Examination showed bilateral severe papilloedema with an enlarged blind spot on visual field analysis. His MRI Brain Scan revealed a Thalamic Space Occupying Lesion for which he required Neuro-Surgery.



Figs. 9-12: Fundus Pictures and Visual Field of a 25-year-old gentleman with a Thalamic SOL causing bilateral Papilloedema and enlarged blind spot in left eye

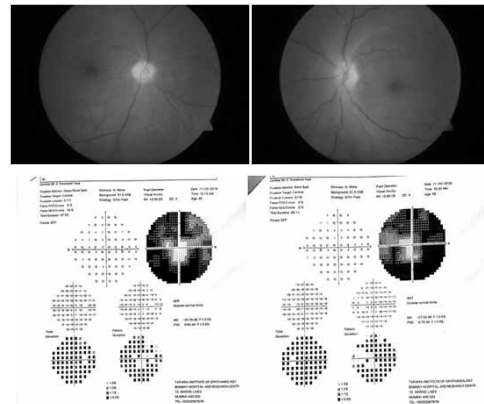
Descriptive Case 4: A 17-year-old gentleman presented with decrease of vision - Left Eye > Right Eye - 6 days. He was on AKT including Ethambutol for 8 months but had Vision Loss Progressing despite stopping AKT. He had a vision of Right Eye: Finger Counting 2 Feet and Left Eye: Finger Counting Close to Face. Fundus Examination showed both eyes mild optic nerve hyperaemia with nerve fibre layer oedema and tortuous blood vessels characteristic of Leber's Hereditary

Optic Neuropathy. He tested positive for Mutation G11778A on Mitochondrial Gene ND4. He was started on Idebenone orally and regained back useful vision to complete his studies and have start his job.



Figs. 13,14: Fundus Pictures of a 17-year-old gentleman with Ethambutol Induced Leber's Hereditary Optic Neuropathy

Descriptive Case 5: A 45-year-old gentleman presented with painless decrease of vision - first right eye and then left eye over one week. He had Hansen's Disease on treatment for 5 months. His Vision was Right Eye: 6/18, N10, Normal Colour Vision and Left Eye: 6/18, N 12, Normal Colour Vision. Fundus Examination showed bilateral Pallid Disc Oedema (Left Eye More than Right Eye) with a visual field affection. He was diagnosed to have Ischaemic Optic Neuropathy related to leprosy.



Figs. 15-18: Fundus Pictures and Visual Fields of a 45-year-old gentleman with Ischaemic Optic Neuropathy related to leprosy.

Neuro-ophthalmic Emergencies or Neurological causes of acute vision loss include¹

Arteritic Anterior Ischaemic Optic Neuropathy

- Most patients with temporal arteritis are over the age of 60 years.
- When present, jaw claudication and neck pain are strongly suggestive of GCA.
- Other symptoms of GCA include headache, scalp tenderness, myalgia (polymyalgia rheumatica), and constitutional symptoms such as fever, malaise, weight loss, or anorexia.

Occipital Infarct

- Patients with occipital infarcts are often misdiagnosed as having an intraocular or functional problem, especially if the visual fields have not been adequately tested.
- However, with occipital infarction, the pupillary light reaction is normal, as is the fundus and ocular motility examination.
- In addition, these patients will not typically have headaches.

Transient Monocular Vision Loss

- Transient monocular visual loss (TMVL) and amaurosis fugax are used interchangeably to describe painless, transient vision loss in one eye, attributed to ischaemia or vascular insufficiency and lasting for several seconds to a few minutes.
- Patients describe their vision loss as a shade or curtain coming down over

their eye.

- TMVL usually occurs in people older than 50 who have atherosclerotic risk factors such as diabetes, hypertension, and hyperlipidaemia.
- Also, coronary artery or peripheral vascular disease is often present, especially carotid artery stenosis.
- Frequently, patients may have had similar previous episodes or other cerebral transient ischaemic attacks (TIAs).
- Owing to the transient nature, vision returns as the retina, optic nerve, or choroid are reperfused

Methanol Optic Neuropathy

- Methanol poisoning can initially present with nausea and vomiting.
- Within 1 to 2 days, patients develop abdominal pain, headaches, confusion, drowsiness, blurred vision, respiratory distress, and central nervous system depression.
- If the diagnosis goes unrecognised, death from coma and respiratory depression can occur.

Pituitary Apoplexy

- Pituitary apoplexy is the result of infarction and/or haemorrhage into a pituitary tumour.
- The resulting haemorrhagic expansion of the pituitary tumour can cause mass effect on adjacent neural structures.
- Pituitary apoplexy should be considered whenever there is sudden blindness and/or ophthalmoplegia associated with headache.

Take Home Message

1. Demyelinating Disorders present as Optic Neuritis with acute or sub-acute loss of vision and colour vision with a central scotoma on visual field analysis
2. Ethambutol (and at times Isoniazid and Linezolid) are anti-tuberculosis agents that cause optic neuropathy which presents as sub-acute loss of vision and colour vision with a central scotoma on visual field analysis which is reversible on cessation of the drug.
3. Space Occupying Lesions cause Papilloedema which causes headache, diplopia and an enlargement of blind spot on visual field analysis.
4. Leber's Hereditary Optic Neuropathy can be induced in susceptible men by certain agents.² Treatment involves use of a novel agent Idebenone which reverses blindness partially.
5. Leprosy can cause ischaemic optic neuropathy in susceptible patients.
6. Neurological emergencies include Arteritic Anterior Ischaemic Optic Neuropathy, Occipital Infarct, Transient Monocular Visual Loss,

Methanol Optic Neuropathy, Pituitary Apoplexy

7. Perhaps 2 of the easiest ways to assess the functioning of the optic nerve apart from vision assessment include assessment of pupils and looking for red colour desaturation.

Acknowledgments

All pictures used in the presentation are of patients (whose identities are not disclosed) who are treated by the Corresponding Author in conjunction with General Physicians, Neurologists and Neuro-Surgeons and Other Appropriate Specialists as required / deemed necessary for control of the disease and the corresponding author whole-heartedly acknowledges the help and guidance of all those general physicians / specialists for these and the other patients - past and future.

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2. Sadun AA, Morgia CL, Carelli V. Leber's Hereditary Optic Neuropathy. Current Treatment Options in Neurology 2010

Alcohol and cancer

Alcohol is causally linked to upper aerodigestive tract cancers (oral cavity, pharynx, larynx, oesophagus) and those of the colon, liver, and female breast.

For example, a three times increase in the recommended alcohol limit raises the risk of oesophageal cancer eight times, leading to estimates that three-quarters of oesophageal cancers are due to high alcohol consumption.

Longer life expectancy means greater cumulative exposure, and as more populations become affluent, alcohol consumption grows.

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