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Indian Journal of Clinical and Experimental Ophthalmology

Journal homepage: www.ijceo.org

Case Series

Optic nerve head: A diagnostic Saga!

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ARTICLE INFO

Article history:
Received 19-01-2024
Accepted 18-04-2024
Available online 30-09-2024

Keywords:
Optic neuropathy
Optic nerve head edema
Optic disc hyperemia
Optic disc cupping

ABSTRACT

Optic nerve head assessment can be a window to diagnosing a plethora of diseases. It can provide us with a hint into glaucoma as well as many non glaucomatous optic neuropathies associated with neurological conditions like space occupying lesion, raised intracranial tension, demyelination, & systemic diseases like uncontrolled hypertension, diabetes, vascular auto- dysregulation, as well as toxic dietary influences & deficiencies, which can have serious consequences in terms of not just a threat to vision, but also systemic morbidity & mortality.

1. Methodology & Case Description

This is a retrospective case series of ten patients who presented at a tertiary eye care, with clinical features suggestive of an Optic neuropathy. The patients were evaluated & diagnosed on the basis of clinical judgement & investigations.

2. Case 1

A 43-year-old female patient presented with complaint of dimness of vision left eye since 1 month. BCVa RE- 6/6, LE- 6/60, RE-disc edema along nasal margin, LE - temporal pallor. Visual fields - LE unequivocal & RE total loss of right half, respecting vertical meridian. Suspecting Foster Kennedy syndrome, patient was further investigated & found to have a bilateral frontal space occupying lesion, for which she underwent bifrontal craniectomy with supraorbital osteotomy & excision of SOL which was adherent to optic chiasma. Biopsy confirmed olfactory groove sarcoidosis.

3. Case 2

61 year old female, diabetic & hypertensive, presented with complain of decreasing vision left eye since 10 days. BCVa, RE-6/9 & LE- 6/36p, RAPD in LE , IOP 19 mmHg BE, .75 & .85 cupping RE & LE respectively with prominent disc pallor. LE showed very advanced visual field defect, but RE field clinched the diagnosis of neurological fields. Investigations revealed pituitary microadenoma compressing left optic nerve & crossing fibres of right eye along optic chiasma, for which she underwent surgery with a successful removal of the tumor.

4. Case 3

A 53 year old male, hypertensive, presented with complaint of decreased vision. BE had shallow anterior chamber, occludable angles, BCVa RE-6/12p, LE-6/9, IOP 18 & 19 mmHg respectively & CCT of 490 microns BE, RE C:D- . 7 & LE C:D -.5 prominent temporal pallor of both optic nerve heads, left homonymous hemianopia. Field defects appeared to break through to the opposite side with a questionable superior arcuate form. He gave a history of left hemeperesis 4 years back with CT brain showing

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https://doi.org/10.18231/j.ijceo.2024.104

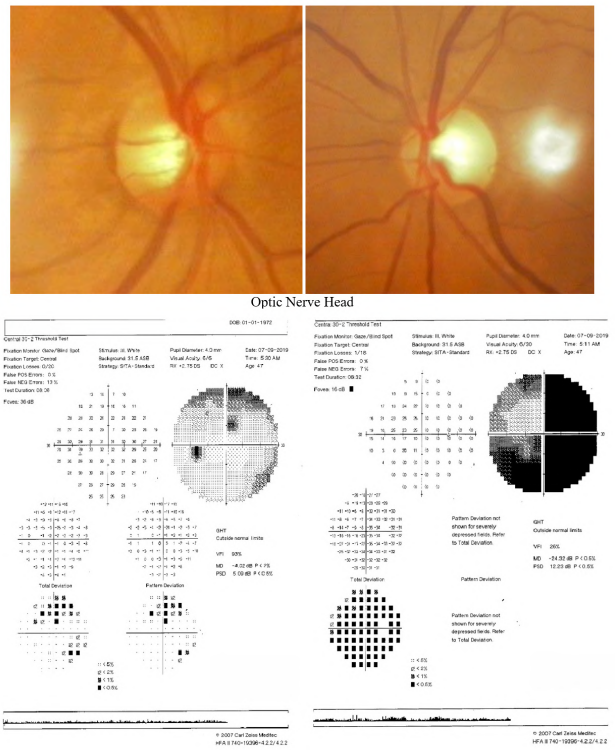

Figure 1: Case 1: Olfactory groove sarcoidosis

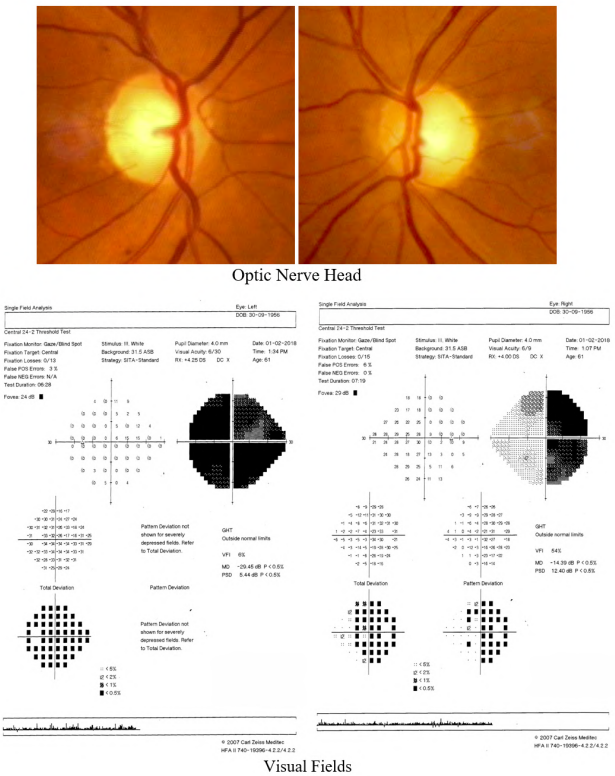

Figure 2: Case 2: Pituitary microadenoma

retrochiasmatic right hemorrhagic infarct.

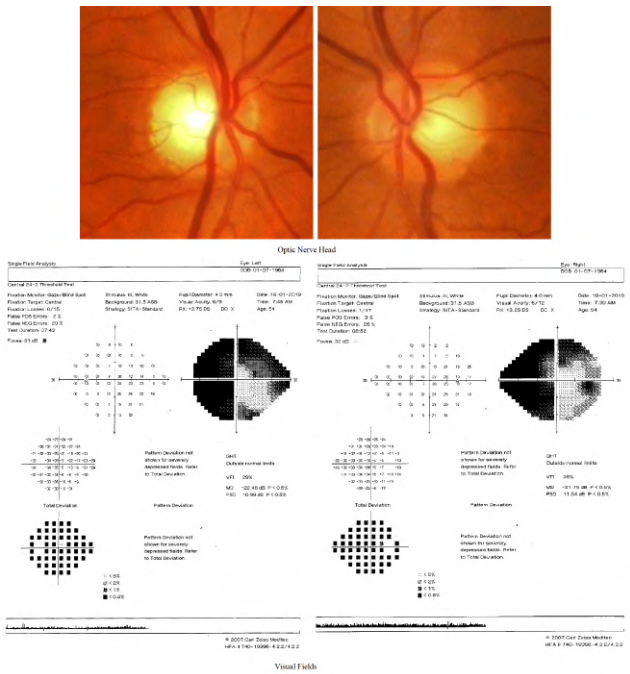

Figure 3: Case 3: Retrochiasmatic right hemorrhagic infarct

5. Case 4

A 24-year-old female patient presented with complaint of dimness of vision both eyes since 1 year, tinnitus & some hearing loss. BCVa 6/60 OU, IOP 17 mmHg, normal ODs with .3 cupping, open angles. She had been diagnosed as a malingerer elsewhere, in view of all normal findings, including a normal color vision test. We did Visual field test, which revealed a central, para central field defect. Suspecting some form of optic neuropathy, a VEP was advised, which showed bilateral prolonged P-100 latencies, confirming an optic neuropathy of a non-glaucomatous type, for which she is being further investigated.

6. Case 5

A 53 year old male patient presented with complaints of progressive dimness of vision both eyes & headache since few months , but a sudden & gross dimness of vision RE , followed soon in LE 20 days back. His Vn both eyes was CF 1 Feet, IOP-15 & 11 mmHg RE, LE respectively, open angles & C:D .75 RE, .3 LE , with disc pallor, almost total color vision defect ,marked field defects both eyes. After an intensive questioning & history taking, he revealed heavy alcohol intake daily since many years. A diagnosis of toxic optic neuropathy/atrophy, was made.

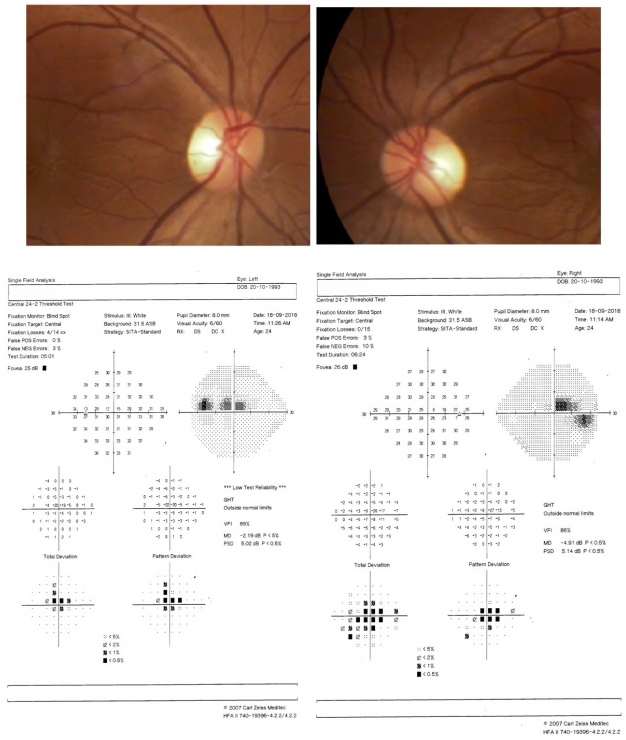

Figure 4: Case 4: Non glaucomatous optic neuropathy

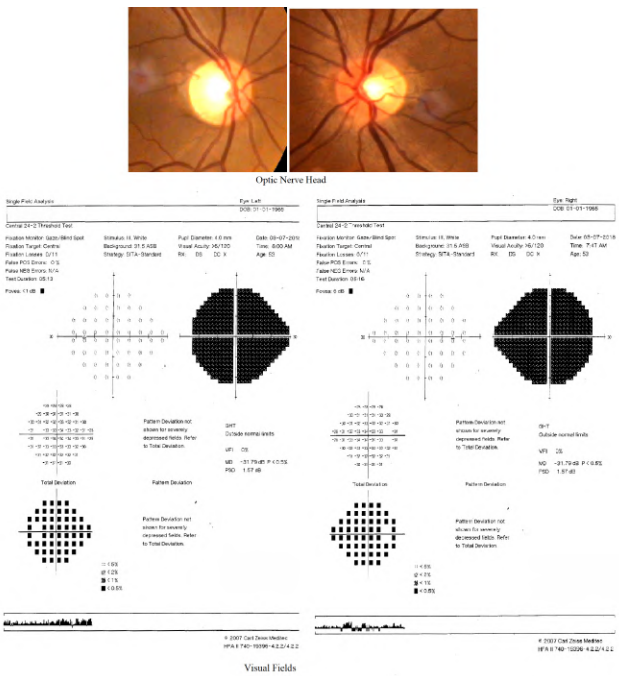

Figure 5: Case 5: Toxic optic neuropathy/atrophy

7. Case 6

A 50 year old diabetic patient presented with complaint of discharge in left eye. His BCVA BE was 6/5, N-6, LE showed RAPD, IOP OU-19/ 20 mmHG respectively, RE optic disc normal, LE showed disc edema . Color vision BE- normal, Visual fields RE- Normal, LE showed early/ relative inferior altitudinal field defect, uncontrolled blood sugar levels. A diagnosis of non arteritic anterior ischemic optic neuropathy [NA-AION] was made.

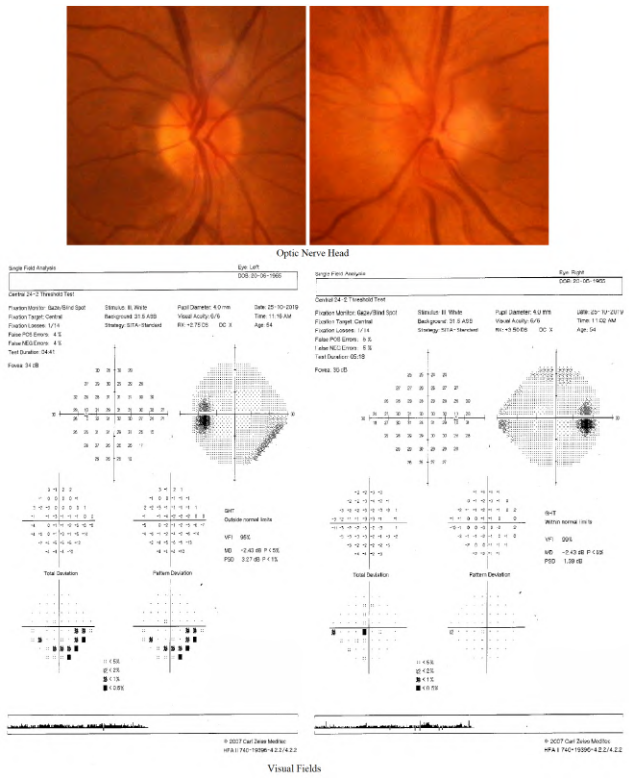

Figure 6: Case 6: Non arteriticanterior ischemic optic neuropathy

8. Case 7

A 55 year old male patient presented with complains of sudden & gross dimness of vision RE followed by some dimness of vision LE since 1 year. BCVA RE 6/60, LE 6/18,IOP- 17 mmHg .RE- Disc gliosis with pallor, LE disc - temporal pallor, with both eyes cupping not clear. RE showed gross field defect, LE altitudinal field defect. ESR -22,borderline hyperlipidemia, CRP normal, other features like jaw claudication were absent & patient denied temporal artery biopsy . A diagnosis of Arteritic anterior ischemic optic neuropathy was made [A-AION].

9. Case 8

A 42 year-old female patient came for a regular glass prescription. BCVa 6/6, N/6, uneventful ocular exam, except

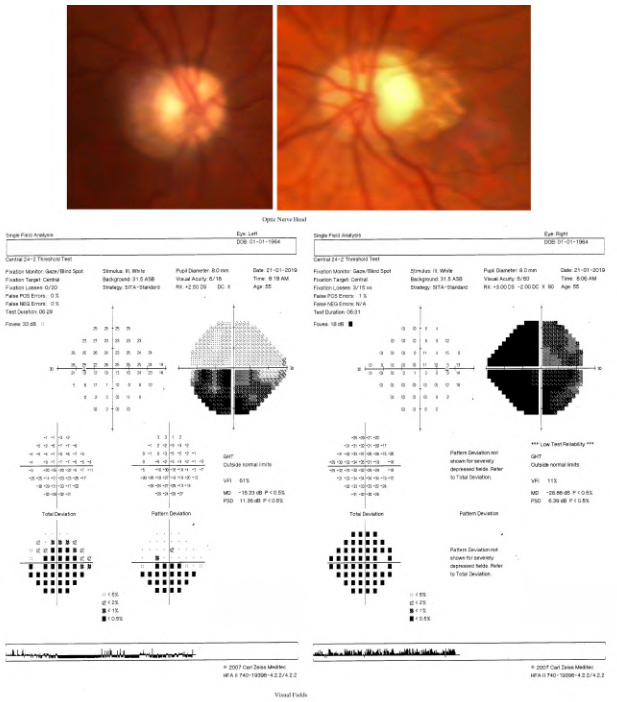

Figure 7: Case 7: Arteritic anterior ischemic optic neuropathy

a bilateral disc edema. On questioning she told that she was a patient of Benign intracranial hypertension under long term diuretic treatment. Visual fields showed a classic enlargement of blind spot with peripheral constriction of field.

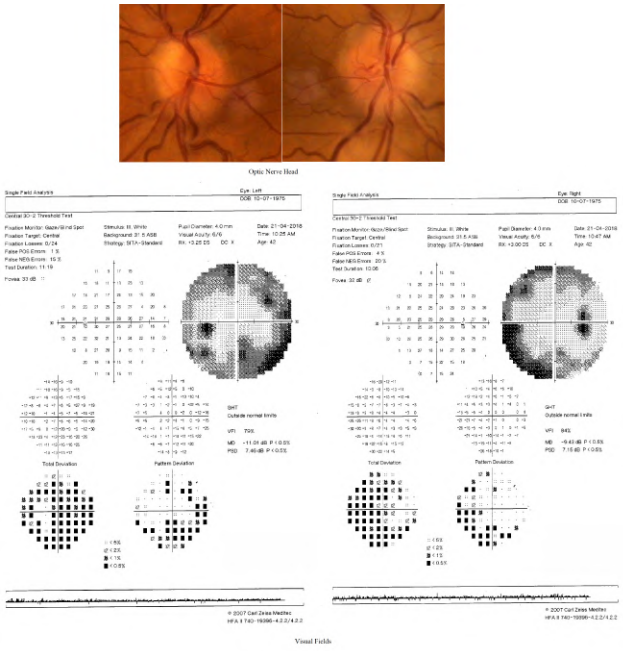

Figure 8: Case 8: Papilledema benign intracranial hypertension

10. Case 9

A 49 year old female patient presented with complain of pain right eye with decreased vision since few days. BCVA RE 2/60, RAPD, total color vision loss, & disc hyperemic & edematous. LE normal with BCVA 6/6. Visual fields LE-normal, while RE showed a gross irregular field defect, Borderline neutrophilia. MRI brain & spinal chord did not show MS plaques. Optic neuritis was diagnosed & treated with high dose systemic steroids, with vision & fields returning to normal.

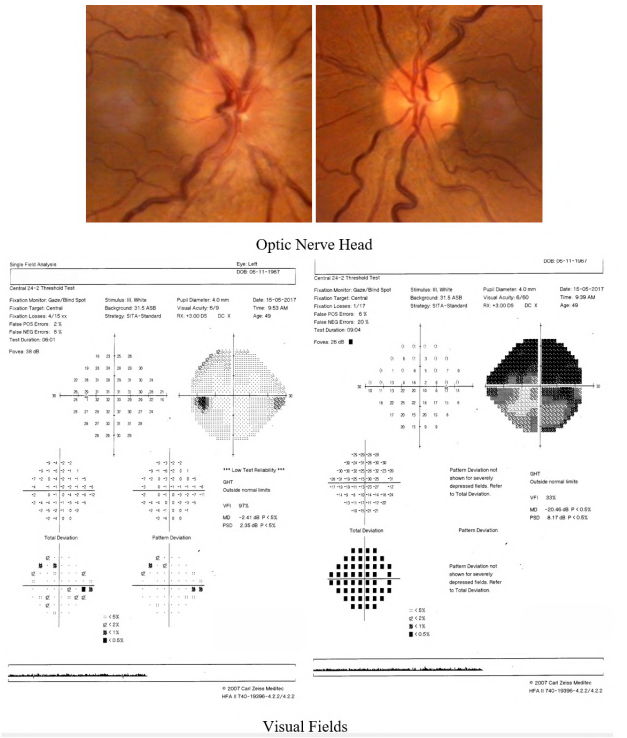

Figure 9: Case 9: Optic neuritis

11. Case 10

A 40-year-old male patient presented with complaint of gradual & gross dimness of vision since last 15 years. Vision BE 1/60, IOP 18 mmHg, defective color vision. Optic discs showed disc pallor & peripapillary telangiectasis & Visual fields showed irregular constriction. MRI plain & contrast Brain & orbit - T2 hyperintense signal seen in optic nerve & chiasma. VEP & CSF analysis - normal. Suspecting Leber's hereditary optic neuropathy, a genetic study was conducted for mitochondrial gene variation evaluation, which showed a homoplasmic missense variation in the MT-ND4 gene, mutations of which have been reported to be associated with LHON.

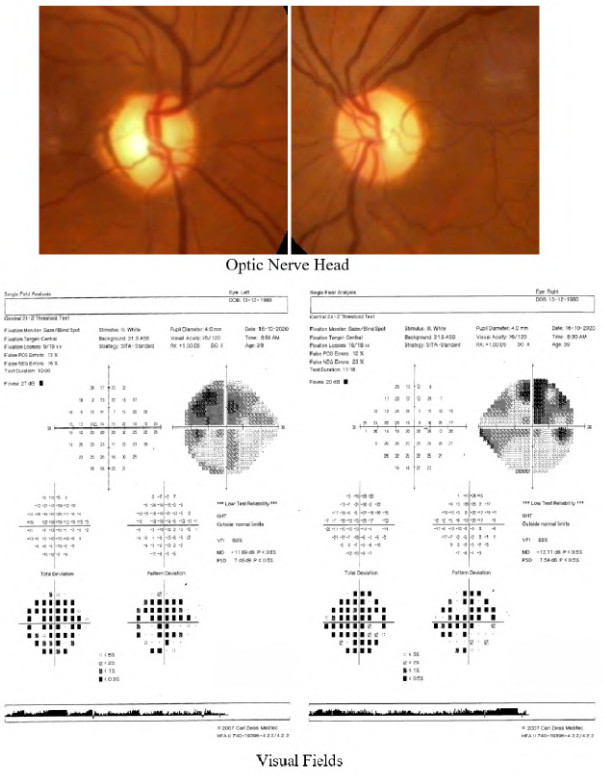

Figure 10: Case 10: Leber's hereditary optic neuropathy

12. Discussion

In case 1 a timely diagnosis with an optimum surgical intervention saved the patient from systemic & ocular morbidity, with an excellent visual recovery in LE from 6/60 pre-op to 6/5 post –op. Case 2 was being treated for glaucoma elsewhere. Resection of tumor unfortunately lead to collateral damage, with loss of vision in the better seeing eye, which could have been avoided / mitigated with a timely diagnosis & intervention. In Case 3 neurological diagnosis was clear but in view of the occludable angles, thin CCT, antiglaucoma medication was also started, as disc & field picture was inconclusive in making a confirmed diagnosis of a concurrent glaucoma. In cases 1-3 Visual field analysis confirmed a neurological pathology.[1] Case 4 – Its important to thoroughly investigate a patient before labeling them as malingerer to avoid missing a potentially vision/ life threatening pathology.[2] Case 5 Toxic optic neuropathy is often under /late diagnosed, with patients presenting with painless, progressive, bilateral symmetrical visual loss, papillomacular bundle damage, central or cecocentral scotoma, and reduced color vision, where both toxic and nutritional factors play a synergistic role.[3] Case 6 & 7 – In NA- AION there is transient non-perfusion / hypoperfusion of ONH circulation in posterior ciliary arteries [PCAs], seen more often in microangiopathy associated with hypertension & Diabetes mellitus, hence usually less severe & extensive ONH damage than A-AION, in which there is thrombotic occlusion of PCAs. Almost half of the N-AION patients can have almost normal Vision & about 40% show spontaneous visual improvement,[4] in sharp contrast to A-AION, where no such recovery is seen.[5] A relative altitudinal defect with RAPD, unilateral disease, good vision & a small cup in other eye were helpful diagnostic criteria for N-AION in case 6. A massive visual loss with no recovery, bilateral involvement, extensive field defect in RE with an absolute inferior altitudinal field defect in LE helped clinch the diagnosis of A-AION in case 7. Absence of systemic sypmtoms like jaw claudication does not rule out the disease. Temporal artery biopsy should be done for confirming the diagnosis.[4–9] History taking & ONH examination, with relevant investigations are crucial, as infrequent or a single episode of transient obscuration of vision, a feature of papilledema, may not be registered by the patients.[10] Correlating complaints with clinical picture seen on ONH can confirm a diagnosis of anterior typical optic neuritis. The optic neuritis treatment trial (ONTT) showed that IV methylprednisolone followed by a tapering course of oral prednisone accelerated visual recovery by a few weeks, & , that the presence of asymptomatic white matter lesions on the MRI scan is the strongest predictor for MS.[11,12] LHON presents with simultaneous or sequential painless loss of vision with either an acute or sub-acute onset & defects in color vision.[12]

13. Conclusion

A thorough detailed examination of Optic Nerve Head can give insight into a plethora of ocular & systemic diseases. A timely diagnosis, followed by appropriate treatment can thus avoid major ocular & systemic morbidities.

14. Source of Funding

None.

15. Conflict of Interest

None.

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Cite this article: Bhatt J. Optic nerve head: A diagnostic Saga!. *Indian J Clin Exp Ophthalmol* 2024;10(3):603-608.