Case Report

www.ijrhs.com
ISSN (o):2321–7251

Homonymous hemianopia and glaucomatous optic disc changes: Rare co-existence

Sharma V[1], Sharma K[2], Parmar D[3], Pandey ML[4]

Dept of Ophthalmology Indira Gandhi Medical College Shimla. 1- Associate Professor, 2- Senior Resident, 3- Junior Resident, 4-Professor.

Corresponding Address: Dr Kalpana Sharma , Dept of Ophthalmology Indira Gandhi Medical College Shimla ,171001.
Email: doctorkalpana.84@gmail.com

Abstract:

Homonymous hemianopia is a visual field defect involving either two right or the two left halves of the visual field of both eye. It results from the damage of the visual pathway in its suprachiasmatic part. The causes of homonymous heminopia include stroke, brain tumors, head injuries, neurosurgical procedures, multiple sclerosis and miscellaneous conditions. It is challenging task to differentiate whether optic disc changes or visual field defects are secondary to glaucoma or neurological causes. We report a case of 42 yrs old male with chief complaint diminution of vision in left eye for 5 months with optic cup disc ratio of right eye 0.6 and left eye 0.8 with inferior focal notching in left eye, temporal pallor both eyes on fundus examination and left homonymous hemianopia on perimetry. Thus this case presents a rare coexistence of glaucomatous optic disc changes associated with left homonymous hemianopia.

Key words: homonymous hemianopia, focal notching, glaucoma

Introduction:

A common challenge in ophthalmology and neuro-ophthalmology is whether to consider an alternative cause for visual loss in a patient with glaucoma. Glaucomatous optic neuropathy is the most commonly acquired optic neuropathy encountered in clinical practice. While it has clinical features that overlap with non-glaucomatous optic neuropathies—including the presence of vision loss, visual field (VF) loss and optic disc cupping glaucoma usually presents with characteristic visual field defects. However, neuro-ophthalmologic disease may produce similar field defects. We report a case in which there is rare co-existence between neurologic disease and glaucoma which was challenging and most confusing issue. In a series of patients with non-glaucomatous optic atrophy 20% had cupping and in 6% this was typical for glaucoma [1]. When looked at more carefully, it turns out that in addition to cupping, the rim of the optic disk is most often pale in neurologic disease. Rarely does the cupping extend to completely obliterate the rim in neurologic disease [2].

We report a case of 42 yrs old male with chief complaint diminution of vision left eyes for 5 months. In past history patient had head injury when he was 15 years old and recovered subsequently without any morbidity. On general physical examination patient was conscious, cooperative. His central nervous system including sensory ,motor system, cranial nerves and autonomous system was normal. On vision assessment patient had 6/6 vision in right eye (OD) and 6/12 vision in left eye. The patient had normal direct and consensual pupillary reflexes in bilateral eyes. Anterior segment was normal in both eyes. Fundus examination showed optic cup disc ratio of right eye 0.6 and left eye 0.8 with inferior focal notching in left eye and temporal pallor bilateral eyes(figure 1,2). The intraocular pressure was 20 in right eye and 24 in left eye on applanation tonometer. On Perimetry examination patient had left homonymous hemianopia along with supero-temporal and infero-temporal paracentral scotomas in left eye correlating with optic disc changes in left eyes (figure 3) . On OCT(figure 4) of optic nerve head and RNFL thickness, (superior and inferior Avg of 84.99 and 68.69 respectively in right eye and superior and inferior Avg of 74.53 and 69.81 respectively in left eye) patient had thinning of NRRIM and generalised loss of ganglion cell layer clinically correlating with optic disc changes in left eyes. On MRI patient had encephalomalacia with surrounding gliosis in right occipital lobe communicating with occipital horn of

right lateral ventricle with chronic residual hemorrhage in right occipital lobe.

Discussion

A normal optic disc usually forms a vertical oval shape with a vertical diameter that is 7–10% greater than the horizontal diameter. The optic cup is horizontally oval, thus, the horizontal cup/disc (C/D) ratio is larger than the vertical C/D ratio. In healthy individuals, the median C/D ratio value is <0.3 and the difference in the C/D ratios between the fellow eye is <0.2. Therefore, a ratio greater than 0.6 is indicative of optic disc cupping. The color of a normal rim is orange due to the presence of capillaries. In the majority of cases, a normal width follows the inferior, superior, nasal, temporal (ISNT) rule, thus, the neuroretinal rim is broadest in the inferior disc region, followed by the superior disc region, the nasal disc area and finally the temporal disc portion. However, violation of the ISNT rule also occurs in large optic disc cups of nonglaucomatous origin, with a greater frequency in the pediatric population [3].

A loss in the retinal nerve fibre layer (RNFL) layer has been divided into a diffuse loss and localized defects . Since a sharply delineated localized RNFL defect (RNFLD) does not occur in normal eyes, it has a high predictive value for abnormality. The reasons for localized RNFLDs are manifold and include, besides glaucomatous optic neuropathy, diabetic retinopathy and other disease leading to retinal cotton-wool spots, optic disc drusen, nonarteritic anterior ischemic optic neuropathy, pituitary gland tumors, and other causes[4]. The authors have documented that, although the loss of nerve fibres in eyes with band atrophy occurs predominantly in the nasal and temporal segments, the superior and inferior areas of the optic disc lost approximately 50% of their fibres, as ganglion cell axons originating from the nasal retina also penetrate in the superior and inferior portions of the disc. This study, documenting the ability of OCT to measure RNFL in eyes with band atrophy, is extremely important when studying neuro-ophthalmic conditions showing a predilection for RNFL loss in the nasal and temporal portions of the optic disc, such as chiasmal and optic tract compression, heredodegenerative diseases, and toxic, nutritional, compressive, and even inflammatory optic neuropathies [5].

Changes in the optic disc as a result of glaucoma include focal or concentric enlargement, where the vertical diameter change is disproportionate to the horizontal change. Additional traits include deepened excavation, increased exposure of the lamina cribrosa, diffuse rim loss, wedge-shaped nerve fiber layer defects, flame-shaped disc hemorrhages and beta zones of parapapillary atrophy in accordance with nerve fiber layer defects [6].

Several key differences were observed between non glaucomatous optic disc cupping and glaucomatous optic disc cupping. Firstly, the color of the rim is the most important. The rim of non glaucomatous cupping of optic disc often exhibits pallor, while the rim in glaucomatous optic disc is pink. However, differentiating between these two according to rim color is very difficult in end-stage glaucoma when the C/D ratio is ~1.0. Secondly, the presence of focal or diffuse rim loss is important. Focal rim loss is predominantly associated with glaucoma, while eyes with non glaucomatous diseases are often characterized by diffuse rim loss. Although focal rim loss may occasionally be present in non glaucomatous optic disc cupping, total loss of the disc rim is never observed [7-9].

Optic disc cupping is apparent prior to visual field defects in glaucoma . Visual acuity decreases markedly in non glaucoma optic disc changes with apparent visual field losses, but with marginal associated changes in the optic cup. Finally, peripapillary atrophy is an increasingly common observation in glaucoma as compared with non glaucoma optic disc changes On the visual examination, one expects a relatively good visual acuity compared to the degree of cupping in glaucoma patients. If the visual acuity, color vision or visual field defect seems out of proportion to the cupping, then neurologic disease must be considered. When examining the visual field, arcuate defects may occur with glaucoma and other optic neuropathies. However, certain field defects are distinctly less common in glaucoma and more common in neurologic disease. These include central or cecocentral visual loss, defects that respect vertical meridian particularly temporal hemianopic defects with chiasmal lesions [10].

Homonymous hemianopia is a visual field defect involving either two right or the two left halves of the visual field of both eye. It results from the damage of the visual pathway in its suprachiasmatic part. The causes of homonymous heminopia include stroke, brain tumors, head injuries, neurosurgical procedures, multiple sclerosis and miscellaneous conditions [11,12]. In a study retinal nerve fiber layer thickness was reduced in patients with cerebral infarction, providing evidence for the transneuronal retrograde degeneration of Retinal ganglion cells. Transneuronal retrograde degeneration was more pronounced in the nasal nerve fiber layer of the contralateral side and in the temporal nerve fiber layer

of the ipsilateral side of cerebral damage. Pathogenesis of homonymous hemianopia includes mainly ischemic strokes in 70% of cases. The rest is caused by tumors, haemorrhages and other lesions .Approximately 20 to 30% of all patients in neurological rehabilitation centers are reported to have a homonymous visual field disorder. Hemianopia is connected with lesion in: (1) the occipital lobe in 40%, (2) the parietal lobe in 30%, (3) the temporal lobe in 25% and (4) the remaining 5% in the optic tract and the lateral geniculate body. In 7% of all cerebral infarctions both hemi-fields are affected, which results in cerebral blindness [13].

In this case patient had old history of head injury corresponding to which he developed homonymous hemianopia. Fundus examination showed optic cup disc ratio of right eye 0.6 and left eye 0.8 with inferior focal notching in left eye and temporal pallor bilateral eyes and OCT findings suggestive of glaucoma. Thus

- loss of visual acuity - In glaucoma, visual acuity is not affected except in the advanced stages
- Cup to field mismatch - Field changes should correlate with the disc appearance, otherwise the diagnosis of glaucoma should be revised.
- Hemianopic visual field loss - Indicates a neurological disease.
- Pallor is greater than cupping and band atrophy- Glaucomatous optic cup shows polar notching in the upper and lower pole, but nasal and temporal atrophy indicates neurological disease and not glaucoma [14].
- **Glaucomatous Optic Neuropathy**

Characteristics often noted when observing the glaucomatous optic disc include-

- Neuroretinal rim tissue that does not respect the "ISNT" rule.
- Notching of the rim.
- Verticalization of the optic cup.
- Baring of a circumlinear vessel.
- Vessel bayoneting at the optic rim (indicating bean-pot cupping).
- Nasalization of vessels.
- Disc hemorrhage (Drance hemorrhage).
- Abnormally large or atypical pattern of peripapillary atrophy (beta zone atrophy).
- Nerve not exhibiting rim pallor.

Compressive Optic Neuropathy

Compressive optic neuropathy can be distinguished from glaucomatous optic neuropathy by :

- Acute vision loss secondary compressive optic neuropathy has the potential to be marked (20/100 or worse).
- IOP typically is within normal range unless altered by an intraorbital mass (mass effect).
- In cases where the mass effect evolves inside the orbit, there may be proptosis with poor retropulsion, lid retraction, extraocular muscle restriction or choroidal folds.
- Visual field defects occur in the central or cecocentral portion of the visual field.
- The visual fields often demonstrate a steep depth with respect to virtual vertical hemianopic line.
- Compressive lesions often induce disc pallor.
- Dyschromatopsia, red color desaturation defects, brightness desaturation defects and an afferent pupillary defect that is inconsistent with the appearance of the disc's cupping or visual field severity.
- Unilateral compressive optic neuropathy is asymmetric compared to open-angle glaucoma and lesions evolving from the chiasm or behind induce congruous or incongruous homonymous VF defects that respect the neuroanatomical architecture (quadrant or hemianopic defects).
- The age and demographics of compressive optic neuropathy often do not match the demographics of the typical glaucoma patient.

Criterias	Glaucomatous Optic Neuropathy	Neurological optic atrophy
Vision	Gradual, painless, progressive	Acute and marked
IOP	Increased or Normal	Normal
Visual Field Defects	Respect horizontal meridian	Respect vertical hemianopic line.
Optic Disc Pallor	Nerve not exhibiting rim pallor	Disc Pallor
Age Related	Age related	Not related to age

Conclusion

Therefore we must consider glaucoma in patients with neuro-ophthalmic disease. So the four clinical features to identify glaucoma in these patients, namely visual field defects, cupping of the optic disks , elevation of intraocular pressure and RNFL thickness may be considered in patients with neuro-ophthalmologic disorders.

RNFL Thickness measurements by stratus OCT in Asian Indian Eyes

	Asian Indian Eyes		
	Mean ± SD (microns)		
	Kaushik et al	SONY et al	Ramkrishanan et al
Average	101.52±10.1	104.2±8.5	105±39
Superior	123.2± 15.5	131.1±14.1	138±22
Inferior	128.7±13.5	132.3±14.7	129±26
Nasal	87.8±17.4	85.9±17.8	86±21
Temporal	65.9±14.6	67.1±12.8	66±17

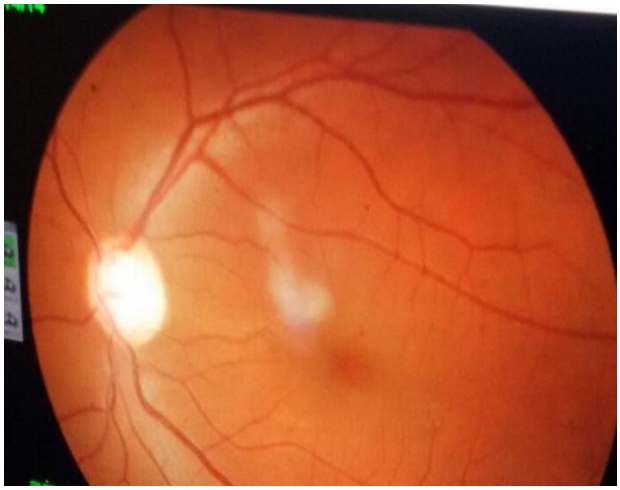


Figure 1: Fundus photograph of left eye showing inferior notching and temporal pallor

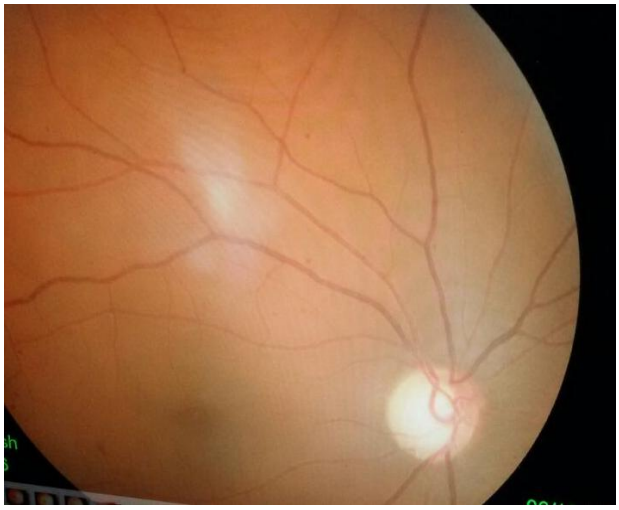
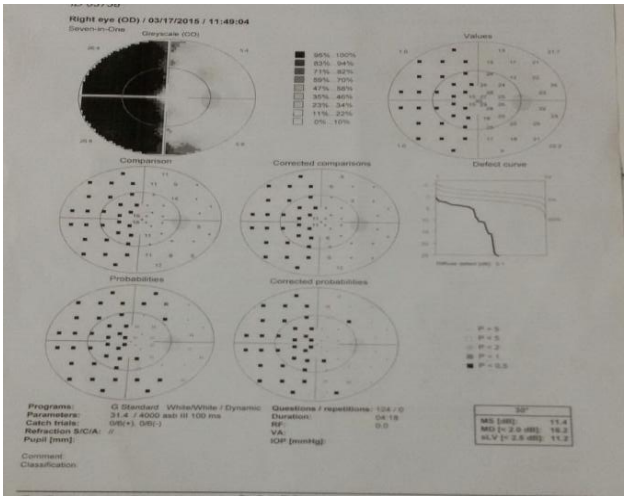


Figure 2: Fundus photograph of right eye showing temporal pallor



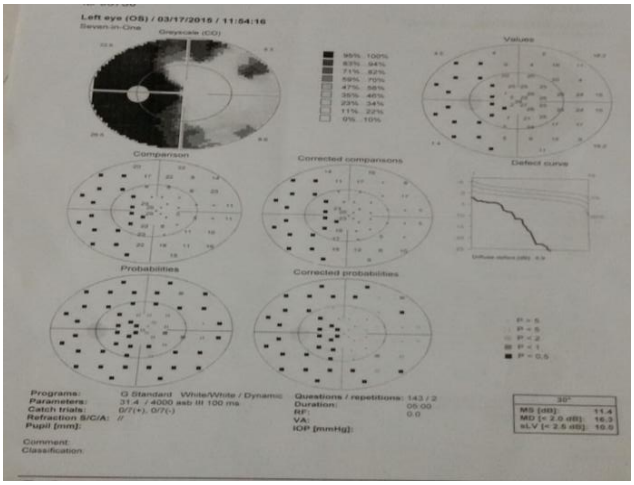


Figure 3: Perimetry of right eye and left eye showing left homonymous heminopia

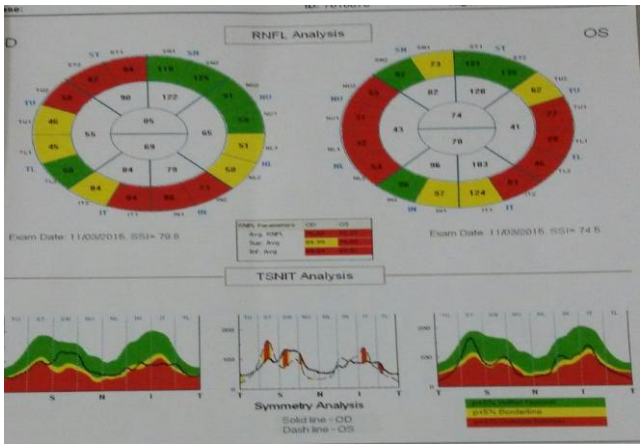


Figure 4: OCT of right eye showing thinning in inferonasal and superotemporal quadrant OCT of left eye showing thinning in nasal and temporal quadrant

Source of Funding: Nil
Source of Conflict: Nil

Acknowledgement

The authors are grateful to authors/editors/publishers of all those articles, journals and books from where the literature for this article has been reviewed and discussed.

References

1. Trobe JD, et al. Nonglaucomatous excavation of the optic disk. Arch Ophthalmol 1980;98(6):1046-50.
2. Trobe JD, Glaser JS, Cassady JC. Optic atrophy. Differential diagnosis by fundus observation alone. Arch Ophthalmol 1980;98(6):1040-5.
3. . Pogrebniak AE, Wehrung B, Pogrebniak KL, Shetty RK, Crawford P. Violation of the ISNT rule in nonglaucomatous pediatric optic disc cupping. Invest Ophthalmol Vis Sci. 2010;51:890–895. [PubMed]
4. Zhao L, Wang YX, Zhang W, Zhang JS, Chen CX, Xu L, et al. (2013) Localized Retinal Nerve Fiber Layer Defects Detected by Optical Coherence Tomography: The Beijing Eye Study. PLoS ONE 8(7): e68998.
5. Roh S, Noecker RJ, Schuman JS, et al. Effect of optic nerve head drusen on nerve fiber layer thickness. Ophthalmology 1998;105:878–85.
6. Jonas JB, Budde WM. Diagnosis and pathogenesis of glaucomatous optic neuropathy: morphological aspects. Prog Retin Eye Res. 2000;19:1–40. [PubMed]
7. Lee AG. Differentiating glaucomatous from nonglaucomatous optic atrophy. Ophthalmology. 1999;106:855. [PubMed]
8. Greenfield DS. Glaucomatous versus nonglaucomatous optic disc cupping: clinical differentiation. Semin Ophthalmol. 1999;14:95–108. [PubMed]
9. Greenfield DS, Siatkowski RM, Glaser JS, Schatz NJ, Parrish RK., II The cupped disc. Who needs neuroimaging? Ophthalmology. 1998;105:1866–1874. [PubMed]
10. Zhang X, Kedar S, Lynn MJ, Newman NJ, Biousse V. Homonymous hemianopias: clinical-anatomic correlations in 904 cases. Neurology. 2006 Mar 28;66(6):906-10.
11. Hae-Young Lopilly Park, Young Gun Park, A-Hyun Cho, Chan Kee Park, Transneuronal Retrograde Degeneration of the Retinal Ganglion Cells in Patients with Cerebral Infarction Ophthalmology June 2013 ;120,(6):1292–1299
12. Obuchowska I, Mariak Z. Homonymous hemianopsia. Klin Oczna. 2012;114(3):226-9
13. Fujino T, Kigazawa K, Yamada R .Homonymous hemianopia: A retrospective study of 140 cases. Neuroophthalmology 1986; 6: 17–21.
14. Nayak BK, Shahanand D. Common avoidable mistakes in the management of glaucoma. J Clin Ophthalmol Res 2013;1:163-8.