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Severity and spatial distribution of visual field defects in primary glaucomas

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Abstract

Purpose: To find out the spatial distribution and to assess the severity of visual field defects in Primary Open Angle Glaucoma (POAG) and Primary Angle Closure Glaucoma (PACG) patients and also to compare among the two groups.

Materials and Methods: 45 eyes of 28 patients with Primary Open angle Glaucoma and 39 eyes of 28 patients with Primary Angle Closure Glaucoma who were clinically diagnosed and monitored in the Glaucoma Service of our hospital for a period of 16 months were recruited into the study. Main outcome measures were 1) distribution of visual field defects, 2) severity of visual field defects and 3) comparison among the two groups.

Results: There was no significant statistical difference in Mean Deviation and Pattern Standard Deviation between the two groups. Superior Field was more depressed in both groups. Glaucoma Hemifield Test also shows that all the zones of superior field were depressed in both the groups but the severity is more in open angle group.

Conclusion: My study shows that superior field is more severely affected than the inferior in both groups. However, trans meridional variation in field loss is less pronounced in subjects with angle closure glaucoma.

Keywords: Visual Field Defects, Humphrey Field Analysis, POAG, PACG.

Introduction

Glaucomatous field damage results from damage to the intra ocular portion of the optic nerve extending from the retinal ganglion cells to the portion just posterior to the lamina cribrosa. Axonal damage is the cause of defects in Glaucoma and the pattern of these defects corresponds to the pattern of distribution of intra ocular axons. The nasal portion of the visual field is often affected in early Glaucoma. These defects may be isolated or associated with Bjerrum's area defects.

In chronic POAG, in the early stages, there may be a generalised depression that progresses gradually or sometimes in steps through paracentral scotoma to arcuate and finally to end stage defects. Defects become denser and then increase in area in one hemifield before progression to other hemifield.

In angle closure Glaucoma, the acute phase with high intra ocular pressure, corneal edema and retinal ischemia produce bizarre field defects. After the pressure has been normalised, if ischemic atrophy of the nerve has occurred, visual field defects may be extensive and may not correspond well to the amount of cupping of the optic nerve head.

Materials and Methods

45 eyes of 28 patients with POAG and 39 eyes of 28 patients with PACG who were clinically diagnosed and monitored in Glaucoma Service of our hospital were recruited into the study for a period of 16 months.

Subjects aged 30 years and older, Patients with Triad of open angle in Gonioscopy, evidence of Glaucomatous Optic Nerve Head changes in Slit Lamp Biomicroscopy and intra ocular pressure >21 mm of Hg. with Applanation Tonometry in POAG group, Patients with occludable angle (if pigmented Trabecular Meshwork was not seen over 270 or more of the angle without indentation) and with intra ocular pressure >21 mm of Hg. in PACG group were included in the study.

Subjects with Normo Tension Glaucoma, Subjects with history of uveitis, any neovascularisation, trauma, epithelial in growth, previous intra ocular or conjunctival surgery, longterm use of topical or systemic steroids, Subjects with secondary Glaucoma including pseudo exfoliation, pigment dispersion were excluded from the study.

All patients underwent preliminary examination in the Glaucoma Department which included visual acuity testing by Snellen chart, Slit lamp Biomicroscopy of anterior segment, Gonioscopy with Goldman single mirror contact lens, Intra ocular pressure (basal and at the time of enrolment) measured with Goldman Applanation Tonometer, optic nerve head changes seen stereoscopically at the slit lamp with 90D volk lens and visual field examination with Humphrey Field Analyser using SITA standard 24-2 strategy.

Results

Of 84 subjects assessed, 45 had POAG and 39 had PACG. There were no significant differences between the groups in age (p=0.173). There were more men in POAG group and more women in PACG group [Table 1].

Table 1: Subject Characteristics

S. No.	Characteristics	Subjects All(n=84)	POAG (n=45)	PACG (n=39)	POAG vs PACG (p Value)
1.	Age	48.2	47.27	49.1	.173
2.	Sex				
	Men	43	31	12	
	Women	41	14	27	.000
3.	Initial IOP(mm		23.4	22.3	

	Hg)				
4.	IOP at enrolment(mm Hg)		18.9	17.6	

Mean baseline recording of IOP in PACG group is 22.3mm of Hg. and that of POAG group is 23.4 mm of Hg. [Table 1; Fig. 1]

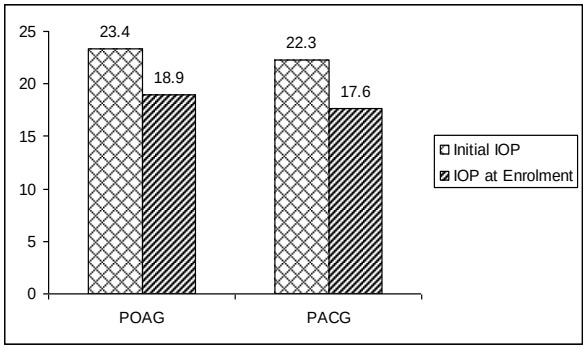

Fig. 1: IOP levels in POAG & PACG

Visual Field Analysis using central 24-2 SITA Standard Programme on Humphrey II was done and the parameters MD, PSD and GHT values were compared among the two groups.

There was no significant difference in Mean Deviation (p=0.615) and Pattern Standard Deviation values (p=0.107) between POAG and PACG[Table 2; Fig. 2 & 3].

Table 2: Global indices for subjects with POAG and PACG

Index	Subjects, Mean (SD) POAG (n=45)	PACG (n=39)	POAG vs PACG p value
Mean Deviation, dB	-13.3(8.2)	-14.79(9.6)	.615
Pattern Standard Deviation	8.5(3.2)	7.2(3.1)	.107

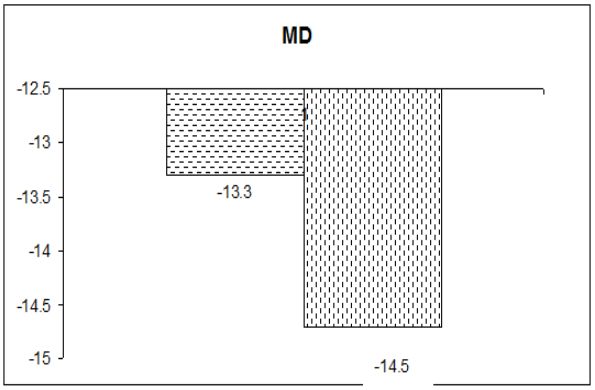

Fig. 2: MD Comparison between POAG & PACG

Comparing the variation in sensitivity between superior and inferior hemifield among POAG and PACG groups, sensitivity was significantly less in the superior hemifield in POAG group; A similar but smaller difference was detected in PACG group [Table 3; Fig. 4 & 5].

Table 3: Superior-inferior hemifield comparison between POAG and PACG

Subjects	Hemifield Pattern Deviation Mean (SD) Superior	Inferior	p value
POAG(n=45)	-10.1(6.4)	-5.3(3.5)	.000
PACG(n=39)	- 7.9(5.2)	-5.6(3.9)	.038

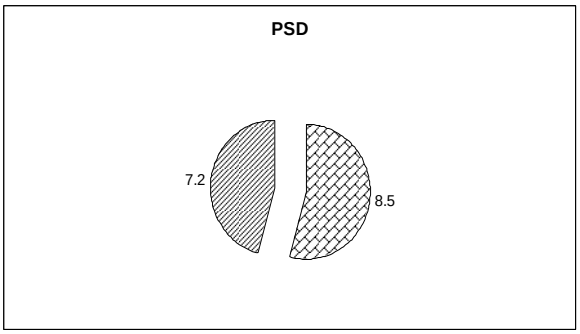

Fig. 3: PSD Comparison between POAG & PACG

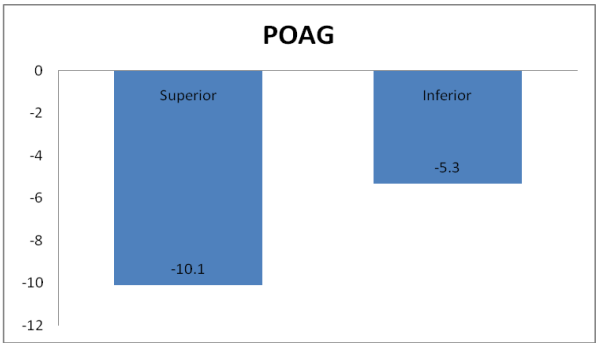

Fig. 4: Superior Vs Inferior Field Comparison

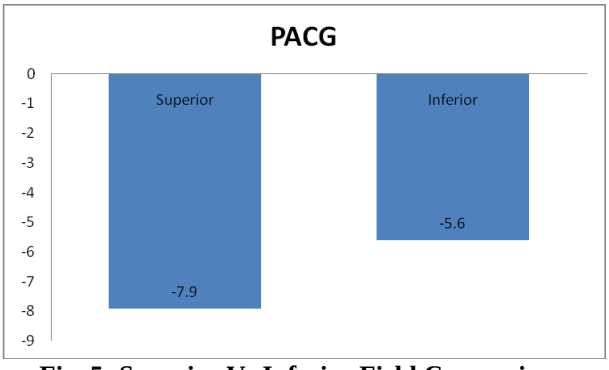

Fig. 5: Superior Vs Inferior Field Comparison

Glaucoma Hemifield Test showed that all the zones of the superior field were significantly depressed than the inferior zones in both groups [Fig. 6].

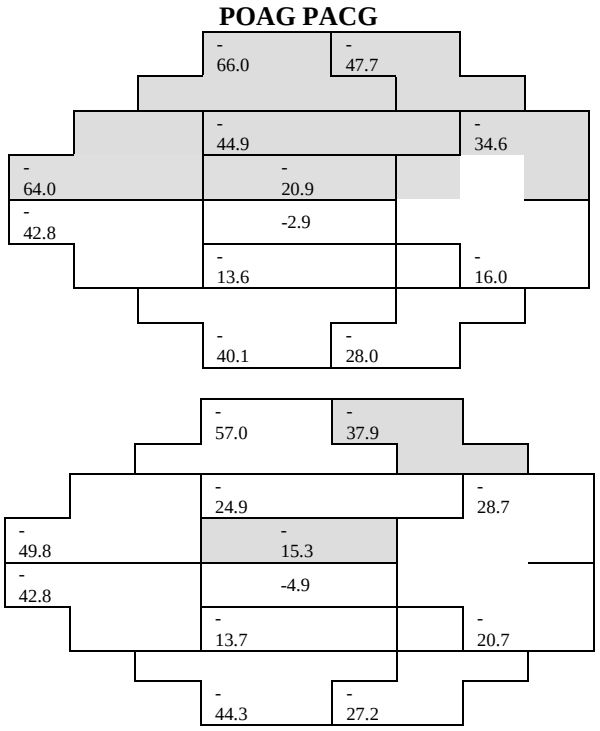

Fig. 6: Glaucoma Hemifield Test Mean Sensitivity in Superior Vs Inferior GHT Regions

Discussion

Our data shows that the superior field is severely affected than the inferior in both POAG and PACG groups. In superior to inferior field comparison, mean of SF in PACG group is -7.9 and Mean of IF in PACG is -5.6. Mean of SF in POAG is -10.1 and of IF in POAG is -5.3[Table 3]. The variation is comparatively more in POAG group.

Comparison of the Visual Field defects between POAG and PACG patients by Gus Gazzard also found that superior hemifield was more severely affected than the inferior in both POAG and PACG groups.(1)

Boomi L and his team studied the effect of acute attack of PACG on the visual field and found that upper nasal quadrant was frequently affected with common involvement of area within 9-20 degree.(2)

Lau Ll and his co-workers studied the field defects in CACG patients and concluded that nasal field was commonly affected in early stage of CACG and the MD of the nasal area was worser than those of arcuate and paracentral area.(3)

Joseph Caprioli MD and his co-workers hypothesize that diffuse loss of visual field sensitivity from glaucoma is largely pressure dependent and the field loss in localised loss group is less pressure dependent.(4)

Greve and Geijssen detected difference in the distribution of visual field defects between high tension and low tension groups- in low tension group, large defects were frequently in the upper half the visual field but the field loss was closer to fixation in high tension group.(5)

Aung's team analysed the visual field following acute PACG and majority of them had hemifield defects consistent with NFB pattern loss.(6)

To conclude, my study shows that the superior field is severely affected than the inferior in both POAG and PACG groups. However, trans meridional variation in field loss is less pronounced in subjects with PACG compared to the other group.

References

1. Gazzard G, Foster PJ, Viswanathan AC, et al. The Severity and Spatial Distribution of visual field defects in Primary Glaucoma. Arch Ophthalmol 2002;120:1636-43.
2. Boomi L, Marrafa M, Marchini G, Canali M, et al. Perimetric defects after a single acute angle closure glaucoma attack. Graefes Arch Cliss Exp Ophthalmol. 1999;237;908-14.
3. Lau LI. The pattern of visual field defects in CACG with different disease severity. Ophthalmology. 2003;110(10):1890-4.
4. Caprioli J, Sears M. Miller JM. Patterns, et al of early visual field loss in open angle glaucoma. Am J Ophthalmol. 1987;103:512-17.
5. Greve, E.L., and Geijssen, C.: Comparison of glaucomatous visual field defects in patients with high and low intra ocular pressures. Fifth International Visual field Symposium. The Hague, W. Junk Publishers, 1983, pp. 101-105.
6. Aung T. The Visual Field following acute Primary Angle Closure Glaucoma. Acta Ophthalmol, Scand. 2001;79:298-300.
7. Douglas GR. Drance SM, Schulzer M, et al. The visual field and nerve head in angle closure glaucoma; a comparison of the effects of acute and chronic angle closure. Arch Ophthalmol. 1975;93:409-11.
8. Dhillon B, chew PT, Lim ASM, et al. Field loss in primary angle closure glaucoma. Asia Pac J. Ophthalmol. 1990;2:85-87.
9. Drance SM. The early field defects in glaucoma. Invest Ophthalmol 1969;8:84-1.
10. Drance SM. The glaucomatous visual field. Invest Ophthalmol. 1972;11:85-6.
11. Coughlan M. Friedmann A1. The frequency distribution of early visual field defects in Glaucoma. Doc Ophthalmol Proc Ser. 1981;26:345-9.
12. Heijl A, Drance SM, Douglas GR. Automatic Perimetry: ability to detect early glaucomatous field defects. Arch Ophthalmol. 1980;98:1560-3.
13. Asman P, Heijj A. Evaluation of methods for automated hemifield analysis in perimetry. Arch Ophthalmol. 1992;110:820-26.
14. Wild JM, Pacey IE, O'Neill EC, Cunliffe IA. The SITA perimetric threshold algorithms in glaucoma. Invest Ophthalmol Vis Sci. 1999;40:1998-2009.
15. Airaksinen, PJ., Drance, S.M., Douglas, G.R., Mawson, D.K., and Nieminen, H, et al. Diffuse and localized nerve fibre loss on glaucoma. Am. J. Ophthalmol. 1984;98:566.
16. Chauhan BC, Drance SM. The relationship between intraocular pressure and visual field progression in

glaucoma. Graefes Arch Clin Exp Ophthalmol, 1992,2301:521-6.
17. Asman P, Heijl A. Glaucoma Hemifield Test: Automated visual field evaluation. Arch Ophthalmol. 1992;110:812-9.
18. Zeyen TG, Zulauf M, Caprioli J. Priority of test locations for automated perimetry in glaucoma. Ophthalmology. 1993;100:518-22.
19. Sample PA, Weinreb RN. Color perimety for assessment of Primary Open Angle Glaucoma. Invest Ophthalmol Vis Sci. 1990;31:1869-75.
20. Beble H, computer assisted evaluation of visual fields. Graefes Arch Clin Exp. Ophthalmol. 1990;228:242-45.
21. Bengtsson B, Heijl A, Evaluation of a new perimetric threshold strategy, SITA in patients with manifest and suspect glaucoma. Acta Ophthalmol Scand. 1998;268-72.
22. Mc Naught E1, Rennie A, McClure E, Chisholm IA, et al. Pattern of visual damage after acute angle closure glaucoma. Trans Ophthalmol Soc U.K. 1974;94:406-15.
23. Shapiro A, Zauberman H. Diurnal changes of the intra ocular pressure of patients with angle closure glaucoma. Br J Ophthalmol. 1979;63:225-27.
24. Konstas AG, Mantziris DA, Stewart WC. Diurnal intra ocular pressure in untreated exfoliation and primary open angle glaucoma Arch Ophthalmol. 1997;115:182-85.
25. King, D., Drance, S.M., D ouglas, G., Schulzer, M., and Wijsman, K., et al. Comparison of visual field defects in normal tension glaucoma and high tension glaucoma. Am. J. Ophthalmol. 1986;101:204.
26. Lewis, R.A., Haureh, S.S., and Phelps, C.D.: Optic disc and visual field correlations in primary open angle and low tension glaucoma. Am. J. Ophthalmol. 1983;96:148.
27. Henson DB, Chauhan BC, Hobley A, Screening for glaucomatous visual field defects: The relationship between sensitivity, specificity and the number of test locations. Ophthalmic Physiol Opt 1988;8:123-27.
28. Anterson, S., and Hitchings, RA: A comparative study of visual fields of patients with low tension glaucoma and those with chronic simple glaucoma. In Greve, E.L., and Heijl, A. editors, Fifth International Visual Field Symposium. The Hague, W. Junk Publishers, 1983. P. 97-99.
29. Adams AJ. Johnson CA, Lewis RA. S cone pathway sensitivity loss in ocular hypertension and early Glaucoma has nerve fibre bundle pattern. In: Drum, B, Moreland J, Serra A, eds. Proceedings of the 10th. Symposium of the International Research group on colour vision Deficiencies. The Hague, the Netherlands: Kluwer Academic Publishers. 1991; P.534-42.
30. Armaly MF. Visual Field defects and clinical perimetry in open angle glaucoma. In: Bellows JG, ed. Glaucoma: Contemporary International Concepts. New York: Masson, 1978;123-38.
31. Ravi Thomas, Ronnie George. Interpreting Automated Perimetry. IJO 2001;49:125-40.