International Journal of Medical Ophthalmology

E-ISSN: 2663-8274
P-ISSN: 2663-8266
www.ophthalmoljournal.com
IJMO 2021; 3(2): 103-106
Received: 11-05-2021
Accepted: 16-06-2021

Dr. Apurva H Suthar
Associate Professor, Department of Ophthalmology, GMERS Medical College, Himmatnagar, Gujarat, India

Corresponding Author:
Dr. Apurva H Suthar
Associate Professor, Department of Ophthalmology, GMERS Medical College, Himmatnagar, Gujarat, India

Association between central corneal thickness, intro ocular pressure and visual field changes in primary open angle glaucoma

Dr. Apurva H Suthar

DOI: https://doi.org/10.33545/26638266.2021.v3.i2b.100

Abstract
Background and Aim: Primary open angle glaucoma (POAG) suspects are individuals with at least one of the following features in one or both eyes like suspicious cupping of optic disc/ visual field defect suspicious for glaucomatous damage/elevated intraocular pressure in the presence of normal optic disc, visual fields. Our objective is to correlate Central corneal thickness, intraocular pressure & Visual field changes in patients diagnosed as the POAG suspects. In order to accurately identify patients at risk of developing glaucoma so that treatment of high-risk individuals can be considered to prevent/delay the development of POAG.
Material and Methods: It was a prospective comparative study conducted at tertiary care institute of Gujarat after taking ethical approval of the institutional ethical committee. The study included 300 eyes of 150 patients who attended the outpatient department over a period of 1 year. Slit lamp bio microscopy, ultrasound pachymetry was done to measure CCT and IOP was measured with Goldman applanation tonometer the visual field assessment was done with the octopus field analyzer.
Results: The mean CCT was 521.12± 23.10 µm, 524.67±22.42 µm among females, males respectively. The mean uncorrected IOP (GAT) was 19.34 mm Hg and 20.01 mmHg on right, left sides respectively statistically significant (P≤0.05). Highest percentage of abnormal visual field changes was seen in eyes with low CCT (<510µm) (P≤0.05). Mean corrected IOP reading was 21.78 mmHg for the 23 patients with abnormal visual fields on right side and this was statistically significant at P≤0.05.
Conclusion: Thinner corneal readings in population especially when presenting at younger age should alarm the ophthalmologist to evaluate the patient thoroughly for glaucoma and should be followed up for the progression of glaucoma. CCT is one factor that is necessary to adjust IOP to achieve a more accurate IOP and it allows monitoring for the risk of progression to be more precise.

Keywords: Central corneal thickness, intraocular pressure, primary open angle glaucoma, ultrasound pachymetry

Introduction
Primary open angle glaucoma (POAG) is a type of glaucoma defined as open, normal appearing anterior chamber angle and raised intraocular pressure (IOP), with no other underlying disease. POAG is typically characterized by: An open, normal-appearing anterior chamber angle and increased intraocular pressure (IOP) without any apparent ocular or systemic abnormality that might account for the elevated IOP, Typical optic nerve head damage and Glaucomatous visual field damage. Glaucoma is the third leading cause of blindness in India and accounts for 5.80% of total blindness in India [1]. It is the leading cause of irreversible blindness in general population worldwide [2, 3]. The estimated prevalence of glaucoma in the world was 60.5 million in 2010 and is expected to rise to 79.6 million by 2020 [3].
Variability in central corneal thickness (CCT) is a confounder with respect to accuracy of tonometry. It has been estimated that tonometric inaccuracy may be significant [4], although the degree of inaccuracy has been questioned [5]. Perhaps of more importance is the apparent predictive power that CCT values may have with respect to the risk of developing glaucoma. The Ocular Hypertension Treatment Study [6] demonstrated that the presence of a thin cornea increased the chance of developing glaucoma, the risk of conversion in those with CCT 555 µm being over three times higher than in those with corneas 588 µm thick. However, it has to be remembered that the error in intraocular pressure (IOP) measurement due to variability in CCT could explain the association between CCT and the development of glaucoma [7]. Considerable evidence suggests that abnormal optic nerve blood flow has a role in the development of glaucomatous optic neuropathy [8].

Recent data indicate that optic nerve head neuroretinal rim blood flow improves significantly in patients with OAG after sustained therapeutic IOP reduction [9]. Among patients with ocular hypertension (OHT), such improvements were limited to vasospastic subjects [10]. The prognostic significance of these blood flow changes remains to be determined. Findings suggest that the presence of a thin cornea is linked to the development of glaucoma among patients with OHT [11] as well as to the severity of OHT [12, 13] and OAG [14, 15]. In OHT and OAG, a thin cornea is more strongly associated with disease severity than IOP.[15] Underestimated Goldmann tonometric pressures seem to only partly explain the relationship between thin corneas and increased glaucoma risk. The other mechanisms underlying this relationship are unknown. Corneal thickness has been linked to scleral thickness [16-18].

A glaucoma suspect is an individual with clinical findings and/or a constellation of risk factors that indicate an increased likelihood of developing primary open-angle glaucoma (POAG). POAG suspects are individuals with at least one of the following features in one or both eyes like suspicious cupping of optic disc/ visual field defect suspicious for glaucomatous damage/elevated intraocular pressure in the presence of normal optic disc, visual fields. POAG is a multi-factorial disease with a devastating impact on the quality of life of the patient in the moderate and severe stages of the disease. Recognition of all risk factors for POAG is important for early diagnosis and intervention [19].

In this study, our objective is to correlate Central corneal thickness, intraocular pressure & Visual field changes in patients diagnosed as the POAG suspects. In order to accurately identify patients at risk of developing glaucoma so that treatment of high-risk individuals can be considered to prevent/delay the development of POAG.

Material and Methods

It was a prospective comparative study conducted at tertiary care institute of Gujarat after taking ethical approval of the institutional ethical committee. The study included 300 eyes of 150 patients who attended the outpatient department over a period of 1 year. All patients were between age group of 35 & 75 years & clinically diagnosed as primary open angle glaucoma suspects.

Patients with open anterior chamber angles on gonioscopy and Patients with Consistently elevated IOP (>21mmHg) associated with normal appearance of the optic disc and retinal nerve fiber layer and with normal visual field test results were included in the study.

Exclusion criteria included any cases below the age of 18 years, any subjects with ocular comorbidity such as previous corneal surgery or disease, any subjects with a diagnosis of secondary glaucoma, or where the diagnosis was unknown.

After taking consent of the patients, detailed ocular, systemic & family history was taken. Any history of refractive errors, use of corrective glasses or contact lenses, glaucoma, use of topical steroids & any previous records with respect to IOP, optic nerve head status or visual field was taken. Any history of ocular surgery like cataract/ LASIK/ photorefractive keratectomy was noted. This is important as refractive error correction procedures are known to be associated with thinning of cornea & hence falsely low IOP and cataract surgery has been associated with lower IOP as compared to pre-surgery baseline. The BCVA was assessed using an illuminated Snellen's chart, with the patient seated at 6 meters distance. Near vision was assessed using Jaeger's near vision chart. Colour vision was checked using Ishihara's pseudoisochromatic charts. Slit lamp examination was performed to rule out any corneal/anterior segment pathology or infections. Gonioscopy was performed with Goldmann three-mirror lens and the anterior chamber angle was graded according to Modified Shaffer's grading. Only patients with open anterior chamber angles were included in this study. Detailed fundus examination by indirect ophthalmoscopy, followed by Slit-lamp biomicroscopic evaluation with 90 D lens. Posterior pole & Optic Nerve Head findings were noted down.

Results

The patient population consisted of 63 females and 87 males with average age of 52.4 years. The mean CCT was 521.12± 23.10 µm, 524.67±22.42 µm among females, males respectively. The mean uncorrected IOP (GAT) was 19.34 mm Hg and 20.01 mmHg on right, left sides respectively statistically significant (P≤0.05). The corrected was 19.12 mm Hg and 19.94 mmHg right and left side respectively the difference being statistically significant (P≤0.05). Significant and moderate positive correlation between the CCT and IOP was observed for the right side (r) = 0.45 and left side(r) =0.31 statistically significant (P≤0.05).

Highest percentage of abnormal visual field changes was seen in eyes with low CCT (<510µm) (P≤0.05). Mean corrected IOP reading was 21.78 mmHg for the 23 patients with abnormal visual fields on right side and this was statistically significant at P≤0.05. Higher sensitivity of Corrected IOP (35.2% to 64.6%) as compared to uncorrected IOP (21.6% to 35.1%) when diagnosing POAG suspects with visual field changes suspicion for glaucoma.

Table 1: Age & gender distribution among study subjects

Variables	Mean	Standard Deviation
Age	52.4	6.9
Gender	Number	Percentage
Male	87	58
Female	63	42

Table 2: Comparison of mean CCT between genders

Gender	N	Mean	SD	P value
Male	87	524.67	22.42	
Female	63	521.12	23.10	0.98

Statistically significance at p≤0.05
Test applied independent student t test

Table 3: Distribution of Central Corneal Thickness [CCT] as normal, low, high CCT

Sides	CCT	N	Percentage%
Right side	Normal	64	43
	Low	45	30
	High	41	27
Left side	Normal	64	43
	Low	39	26
	High	47	31

Table 4: Comparison of CCT with visual field changes on right & left side using chi-square test

Sides	CCT	Normal	Abnormal	P value
Right side	Normal	60	4	0.05*
	Low	30	12	
	High	34	7	
Left Side	Normal	56	8	0.88
	Low	30	9	
	High	41	6	

*indicates statistically significance at p≤0.05
Test applied chi-square test

Discussion

Clinical decision-making relies on established and quantifiable parameters; these must be used rather than a reliance on medical impression and intuition. The importance of CCT is now well known and a proven parameter [20, 21]. Shih *et al.* [22] had a similar objective: to ascertain whether CCT affected patient management. Their study, although set within a specialist glaucoma service, had similar results which showed that half their study population required an adjustment of IOP ± 1.5 mmHg. What is interesting is that 8–10% of their cohort had a change in their medication. A review of the literature suggests that, after an initial hypercompliant phase, the lamina cribrosa becomes more rigidin glaucoma [23-26].

A thin central cornea is emerging as a major risk factor for severity of OHT and OAG [27-29]. Diurnal and longterm IOP fluctuations are also a major risk factor for progression in OAG [30-31]. These results suggest that a thin central cornea may be a marker for physiological differences in the biomechanical properties of the lamina cribrosa. In other words, it may be that a thin central cornea is connected to a thin sclera, which, in turn, is connected to a thin lamina.

In our study population, the mean age was 52.4 ± 6.9 years. The mean CCT among females was 521.12± 23.10 µm & among males was 524.67± 22.42 µm. CCT was divided into 3 categories i.e Low (530 µm). The mean uncorrected IOP (GAT) was 19.34 mm Hg and 20.01 mmHg on right, left sides respectively statistically significant (P≤0.05). In our study, out of 3 categories highest % of abnormal visual field changes was seen in eyes with low CCT that is thin cornea. OHTS results showed high IOP correlated with visual field defects. In our study we got a Mean corrected IOP reading was 21.78 mmHg for the 23 patients with abnormal visual fields on right side and this was statistically significant at P≤0.05. In our study highest percentage of abnormal visual field changes was seen in eyes with low CCT. Therefore knowledge of CCT can help to attribute the likelihood of disease progression and assigning the risk can change clinical management decisions to reach a personalized target pressure. A prior study examined the accuracy of the presently available tonometric correction factors and reported that they are potentially inaccurate with a tendency to overestimate the effect of CCT on IOP.[5] There is no universally acceptable and correct algorithm that is available as a CCT-tonometric correction factor. Thus it is difficult to obtain a true estimate of the effect of CCT on IOP and hence difficult to substantiate or negate any bias issue due to improper measurement of IOP.

Although the result of the present study provides information on associations in a selected group of patients, a possible selection bias may account for these associations and a larger population-based study is required to confirm the findings.

Conclusion

Thinner corneal readings in population especially when presenting at younger age should alarm the ophthalmologist to evaluate the patient thoroughly for glaucoma and should be followed up for the progression of glaucoma. CCT is one factor that is necessary to adjust IOP to achieve a more accurate IOP and it allows monitoring for the risk of progression to be more precise. Any decision in glaucoma, in the absence of CCT is an uninformed one.

References

1. Ritch R, Shields MB, Krupin T. The Glaucomas. 2nd ed. C.V. Mosby: St. Louis. 1996, 1, 2.
2. Friedman W, Wolfs RCW, O'Colmain BJ, Klein BE, Taylor HR, West S. Prevalence of open-angle glaucoma among adults in the United States. Arch Ophthalmol. 2004;122:532-8.
3. Recent advances in glaucoma management. Indian J Clin Exp Ophthalmol. 2019;5(2):140-1. doi:10.18231/j.ijceo.2019.034.
4. Ehlers N, Bramsen T, Sperling S. Applanation tonometry and central corneal thickness. Acta Ophthalmol (Copenh). 1975;53:34-43.
5. Gunvant P, O'Leary DJ, Baskaran M, *et al*. Evaluation of tonometric correction factors. J Glaucoma. 2005;14:337-43.
6. Gordon MO, Beiser JA, Brandt JD *et al*. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary openangle glaucoma. Arch Ophthalmol. 2002;120:714-20. Discussion 829–30.
7. Herndon LW, Weizer JS, Stinnett SS. Central corneal thickness as a risk factor for advanced glaucoma damage. Arch Ophthalmol. 2004;122:17-21.
8. Flammer J, Orgul S, Costa VP, *et al*. The impact of ocular blood flow in glaucoma. Prog Retin Eye Res. 2002;21:359-393.
9. Hafez AS, Bizzaro RLG, Rivard M, Lesk MR. Changes in optic nerve head blood flow after therapeutic intraocular pressure reduction in glaucoma patients and ocular hypertensives. Ophthalmology. 2003;110:201-210.
10. Hafez AS, Bizzaro RLG, Descovich D, Lesk MR. Correlation between finger blood flow and changes in optic nerve blood flow following therapeutic intraocular pressure reduction. J Glaucoma. 2005;14:448-454.
11. Gordon MO, Beiser JA, Brandt JD, *et al*. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. Arch Ophthalmol. 2002;120:714-720.
12. Medeiros FA, Sample PA, Weinreb RN. Corneal thickness measurements and frequency doubling technology perimetry abnormalities in ocular hypertensive eyes. Ophthalmology. 2003;110:1903-1908.
13. Medeiros FA, Sample PA, Weinreb RN. Corneal thickness measurements and visual function abnormalities in ocular hypertensive patients. Am J Ophthalmol. 2003;135:131-137.
14. Medeiros FA, Sample PA, Zangwill LM, *et al*. Corneal thickness as a risk factor for visual field loss in patients with preperimetric glaucomatous optic neuropathy. Am J Ophthalmol. 2003;136:805-813.
15. Herndon LW, Weizer JS, Stinnett SS. Central corneal thickness as a risk factor for advanced glaucoma damage. Arch Ophthalmol. 2004;122:17-21.

16. Albekioni Z, Joson P, Tello C, Liebmann JM, Ritch R. Correlation between central corneal thickness and scleral thickness [ARVO abstract]. Invest Ophthalmol Vis Sci. 2003;44:103.
17. Oliviera C, Tello C, Ritch R, Liebmann JM. Correlation between central corneal thickness, scleral thickness, and refractive error. [ARVO abstract]. Invest Ophthalmol Vis Sci. 2004;44 e-abstract, 963.
18. Rolle T, Lo Presti Constantino L, Morgese A, *et al*. Structural glaucomatous damage and ocular biometric parameters [ARVO abstract]. Invest Ophthalmol Vis Sci. 2004;45 e-abstract, 3335.
19. Maurya RP. Glaucoma Burden: Indian Scenario. Indian J Clin Exp Ophthmol. 2017;3(4):387-8.
20. Kass MA, Heuer DK, Higginbotham EJ, Johnson CA, Keltner JL, Miller JP *et al*. for the Ocular Hypertension Treatment Study Group: The Ocular Hypertension Treatment Study: a randomized trial determines that topical ocular hypotensive medication delays or prevents the onset of primary open-angle glaucoma. Arch Ophthalmol. 2002;120:701-713.
21. Gordon MO, Beiser JA, Brandt JD, Heuer DK, Higginbotham EJ, Johnson CA *et al*. for the Ocular Hypertension Treatment Study Group: The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. Arch Ophthalmology. 2002;120:714-720.
22. Shih CY, Graff-Zivin JS, Trokel SL, Tsai JC. Clinical significance of central corneal thickness in the management of glaucoma. Arch Ophthalmol. 2004;122:1270-1275.
23. Burgoyne CF, Quigley HA, Thompson HW, *et al*. Early changes in optic disc compliance and surface position in experimental glaucoma. Ophthalmology. 1995; 102:1800-1809.
24. Heickell AG, Bellezza AJ, Thompson HW, Burgoyne CF. Optic disc surface compliance testing using confocal scanning laser tomography in the normal monkey eye. J Glaucoma. 2001;10:369-382.
25. Burgoyne CF, Morrison JC. The anatomy and pathophysiology of the optic nerve head in glaucoma. J Glaucoma. 2001;10(suppl 1):S16-S18.
26. Quigley HA. Overview and introduction to session on connective tissue of the optic nerve in glaucoma. In: Drance SM, ed. Optic Nerve in Glaucoma. Amsterdam, the Netherlands: Kugler Publications. 1995, 15-36.
27. Gordon MO, Beiser JA, Brandt JD, *et al*. The Ocular Hypertension Treatment Study: baseline factors that predict the onset of primary open-angle glaucoma. Arch Ophthalmol. 2002;120:714-720.
28. Medeiros FA, Sample PA, Weinreb RN. Corneal thickness measurements and frequency doubling technology perimetry abnormalities in ocular hypertensive eyes. Ophthalmology. 2003;110:1903-1908.
29. Medeiros FA, Sample PA, Weinreb RN. Corneal thickness measurements and visual function abnormalities in ocular hypertensive patients. Am J Ophthalmol. 2003;135:131-137.
30. Asrani S, Zeimer R, Wilensky J, Gieser D, Vitale S, Lindenmuth K. Large diurnal fluctuations in intraocular pressure are an independent risk factor in patients with glaucoma. J Glaucoma. 2000;9:134-142.
31. Werner EB, Drance SM, Schulzer M. Trabeculectomy and the progression of glaucomatous visual field loss. Arch Ophthalmol. 1977;95:1374-137.