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STUDY OF RETINAL NERVE FIBRE LAYER THICKNESS IN CASES OF PRIMARY OPEN ANGLE GLAUCOMA IN CORRELATION WITH PERIMETRIC CHANGES AND CENTRAL CORNEAL THICKNESS

ABSTRACT

Glaucoma has is one of the leading causes of blindness in the developed countries. In developing countries like India, glaucoma is the second leading cause of blindness after cataract. Primary open angle glaucoma is a multifactorial disorder characterised by death of retinal ganglion cells which results in a characteristic optic neuropathy and concomitant visual field reduction.The central corneal thickness (CCT) as well as the intraocular pressure (IOP) are important for assessing glaucoma since a low CCT will underestimate IOP which will affect the prognosis of glaucoma. In this study, we sought to assess the correlation between central corneal thickness (CCT) and quantitative measurements of retinal nerve fiber layer (RNFL). Our findings indicated that glaucomatous eyes with thinner corneas were associated with larger and deeper optic disc cups, suggesting that these eyes may be at an increased risk for RNFL loss.

KEYWORDS

Primary Open angle glaucoma (POAG), Central Corneal thickness (CCT), Intraocular pressure (IOP), Spectral Domain Optical coherence Tomography (SD-OCT), Retinal nerve fibre layer (RNFL), Ocular hypertension (OHT)

INTRODUCTION

Primary open-angle glaucoma (POAG) is a chronic progressive optic neuropathy with characteristic morphological changes at the optic nerve head and retinal nerve fibre layer in the absence of other ocular disease or congenital anomalies. Progressive retinal ganglion cell death and visual field loss are associated with these changes.[1]IOP is considered the most important risk factor for the development of POAG, and remains the only known modifiable risk factor. Population studies have shown increased prevalence of glaucoma with increasing IOP. In patients with OHT (raised IOP but no signs of glaucomatous optic disc or visual field changes), higher IOP is associated with a higher risk of developing POAG.[1]The prevalence of POAG increases with age.Myopia has been shown to be a risk factor for POAG in several

31 studies.A thinner cornea has been shown to be a risk factor for OHT patients developing

32 POAG. This may be in part due to IOP measurement error (IOP tends to be read lower in

33 patients with thinner corneas), but there are also theories that a thinner cornea may

34 indicate less rigid support structures around the optic nerve head, and a resultant increased

35 propensity to damage.A first-degree relative with POAG is a risk factor for the development

36 of POAG.Several genes associated with POAG have been identified.A high prevalence of

37 POAG has been found in diabetic patients, and a high prevalence of diabetes has been found

38 in POAG patients. Hypertension may predispose to glaucomatous damage via increased

39 peripheral vascular resistance in small vessels, while a low blood pressure may reduce the

40 perfusion pressure of the optic disc.[1]

41

42 **PATHOPHYSIOLOGY**

43 **MECHANISM OF AQUEOUS HUMOR FORMATION**

44 Three mechanisms are involved in aqueous humor formation: diffusion, ultrafiltration and

45 active secretion.Diffusion occurs when solutes, especially lipid soluble substances, are

46 transported through the lipid portions of the membrane of the tissues between the

47 capillaries and the posterior chamber, proportional to a concentration gradient across the

48 membrane.Ultrafiltration is the flow of water and water-soluble substances, limited by size

49 and charge, across fenestrated ciliary capillary endothelia into the ciliary stroma, in response

50 to an osmotic gradient or hydrostatic pressure.Active secretion is thought to be the major

51 contributor to aqueous formation, responsible for approximately 80% to 90% of the total

52 aqueous humorformation . The main site for active transport is believed to be the

53 nonpigmented epithelial cells. Active transport takes place through selective trans-cellular

54 movement of anions, cations, and other molecules across a concentration gradient in blood-

55 aqueous barrier. This is mediated by protein transporters distributed in the cellular

56 membrane. Aquaporins (AQPs) are molecular water channels which aid with rapid bulk

57 transport of fluid or transport of fluids against an insufficient osmotic pressure gap. The

58 energy required for the transport is generated by hydrolysis of adenosine triphosphate (ATP)

59 to adenosine diphosphate (ADP), which is activated by Na+ and K+ (66) mediated by Na+-K+-

60 ATPase, an enzyme located in both the non-pigmented and pigmented ciliary

61 epithelia.Another enzyme, carbonic anhydrase, found in the nonpigmented and pigmented

62 ciliary epithelia , mediates the transport of bicarbonate across the ciliary epithelium by the

63 reversible hydration of CO2 to form HCO3-and protons.[2]

PHYSIOLOGY OF AQUEOUS OUTFLOW

65 The trabecular meshwork (trabeculum) is a sieve-like structure at the angle of the anterior

66 chamber (AC) through which 90% of aqueous humour leaves the eye. It has three

67 components

68 The uveal meshwork is the innermost portion and offers little resistance to the passage of

69 aqueous.

70 The corneoscleral meshwork lies external to the uveal meshwork to form the thickest

71 portion of the trabeculum conferring greater resistance to flow.

72 The juxtacanalicular (cribriform) meshwork is the outer part of the trabeculum, and links

73 the corneoscleral meshwork with the endothelium of the inner wall of the canal of

74 Schlemm.The outer wall is lined by smooth flat cells and contains the openings of collector

75 channels, which leave the canal at oblique angles and connect directly or indirectly with

76 episcleral veins. Septa commonly divide the lumen into 2–4 channels.[3,4]

77 There are multiple proposed mechanisms of damage, some of which are IOP- dependent,

78 and others are IOP- independent.

79 Raised IOP is thought to damage the optic nerve head via induced mechanical

80 changes at the lamina cribrosa, or via vascular dysfunction and resultant ischemia.

81 IOP independent mechanisms of damage

82 • Reduced ocular perfusion pressure (and hence the association with vascular

83 diseases such as diabetes, hypertension and migraine)

84

85

86 Diagnosis of POAG requires assessment of:

87 • Intraocular pressure

88 • Open- normal appearing anterior chamber angle

89 • Characteristics signs of optic disc damage

90 • Visual function loss on perimetry [1]

MATERIALS AND METHODS

STUDY DESIGN

This was a cross sectional observational study which was conducted over a period of two years (August 2022-2024).

INCLUSION CRITERIA

1.   Patients of age above 18 years.

2.   Patients of both gender.

3.   Patients presenting with intraocular pressure greater than 21 mm Hg

4.   Characteristic VF changes.

5.   Patients with co morbidities like diabetes and hypertension.

EXCLUSION CRITERIA

1.   Pregnant and lactating women.

2.   Patients with history of cornea opacity.

3.   Patients with congenital /pathological myopia

4.   Disc pathologies like papiloedema

5.   Patients with signs of secondary glaucoma

6.   Non glaucomatous optic neuropathy.

7.   Angle closure suspects

8.   Patients not willing to give consent

SAMPLE SIZE

Patients visiting Ophthalmology outpatient department of a tertiary healthcare centre were enrolled in the study.

Sample Size Calculation: By using 'Cup:disc area ratio 0.71±0.14 0.63±0.13

<0.005 a sample size of Total 72 (36 cases in each arm) completed cases needed to assess the study objective at 80% power and 5% level of significance with 1:1 allocation.

N = 80 (40 in each arm) Considering 10% drop out to be enrolled in this study.


STUDY METHOD

123   Complete medical history including previous medications, ocular trauma or ocular

124   surgeries were recorded in the case record form. Subjects underwent complete

125   ophthalmological examination including uncorrected and best corrected visual acuity

126   (BCVA) measurement, slit lamp examination, Goldmann Applanation tonometry for

127   IOP measurement, gonioscopic evaluation and fundus examination. Visual field test

128   was carried out using Humphrey octopus perimeter with SITA without pupil

129   dilatation. A Glaucomatous VF was defined as a glaucoma hemifield test outside of

130   normal limits on atleast 2 consecutive baseline tests and the presence of atleast

131   three contiguous test points within the same hemifield on pattern deviation plot at

132   P<1%, with atleast one at P<0.5%.CCT was measured using ultrasound pachymeter.

133   RNFL and ONH parameters were recorded through SD-OCT.All eyes had open-angle

134   observed by gonioscopy, and the patients fulfilled at least two of the following

135   criteria: glaucomatous optic neuropathy, IOP >21 mmHg on at least three occasions,

136   and glaucomatous visual field defects. Eyes were divided into two groups on the

137   basis of their median central corneal thickness (CCT) and classified into thick cornea

138   group CCT ≥540 µm (Group 1) and thin cornea group (Group 2) CCT < 540 µm.

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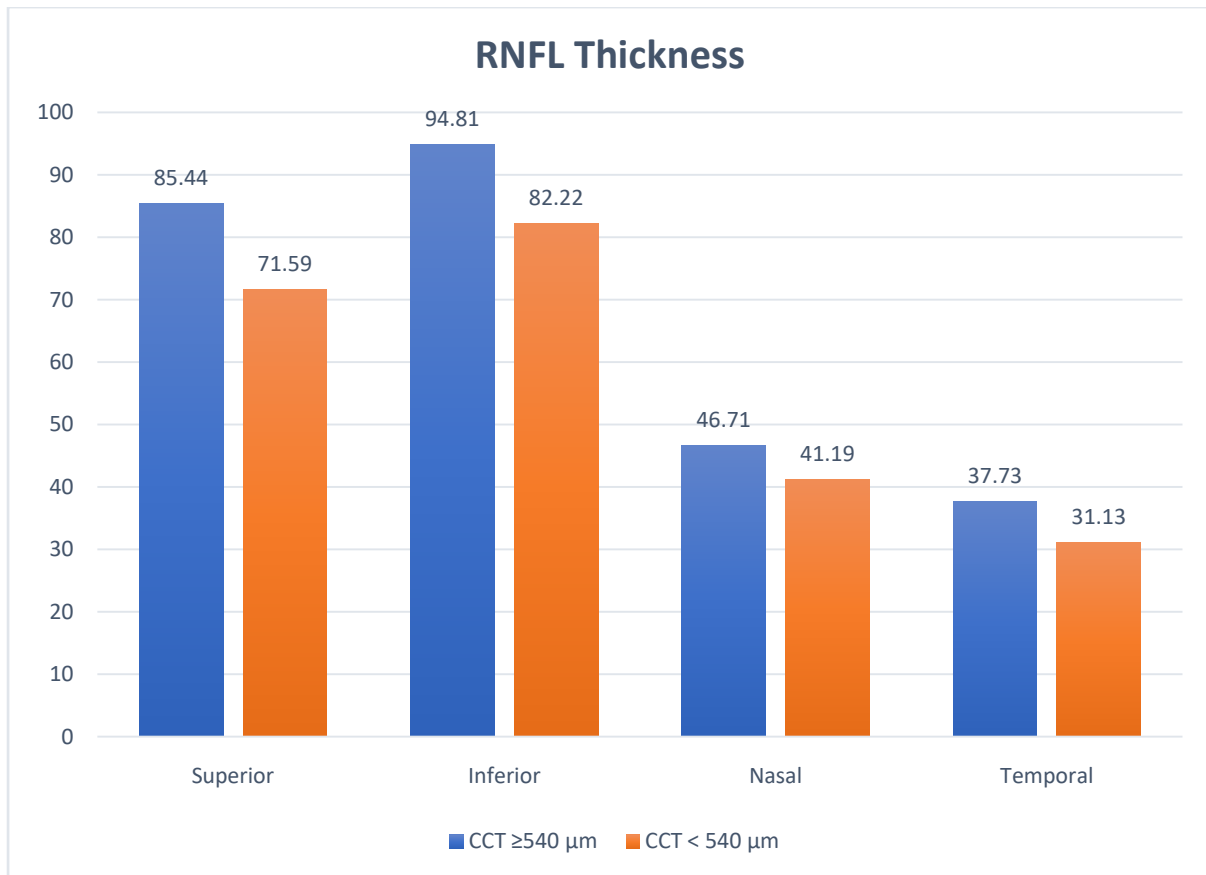
140   **RESULTS AND DISCUSSION**

141 In this study, the age of cases was ranging from **34.00 – 87.00 years** with average age being

142 **61.71** years among CCT ≥540 µm which was comparable to **61.38 years** in CCT < 540 µm and

143 the difference was statistically not significant.In study conducted by Wangsupadilok et al the

144 mean age was 60.96 +/- 7.44 years which was comparable to this study.[5]

145 **62.5%** of total cases were males in CCT ≥540 µm which was more as compared to **41.7%**

146 cases in CCT < 540 µm but the difference was not statistically significant. **37.5%** of total cases

147 were females in CCT> 540 µm which was more as compared to **25%** cases in CCT <540 µm

148 but the difference was not statistically significant.

149 Mean IOP was **23.71 mmHg** among CCT ≥540 µm which was significantly more as compared

150 to **21.81 mmHg** in CCT < 540 µm. A study conducted by Dhiman et al in a cross sectional

151 study on 103 eyes of 58 patients of POAG and divided patients into two groups- thick and

152 thin CCT taking a reference range of 529µm and came to a conclusion that as the CCT

153 increases there is falsely elevated IOP.[6]

Mean Intra ocular pressure in both groups

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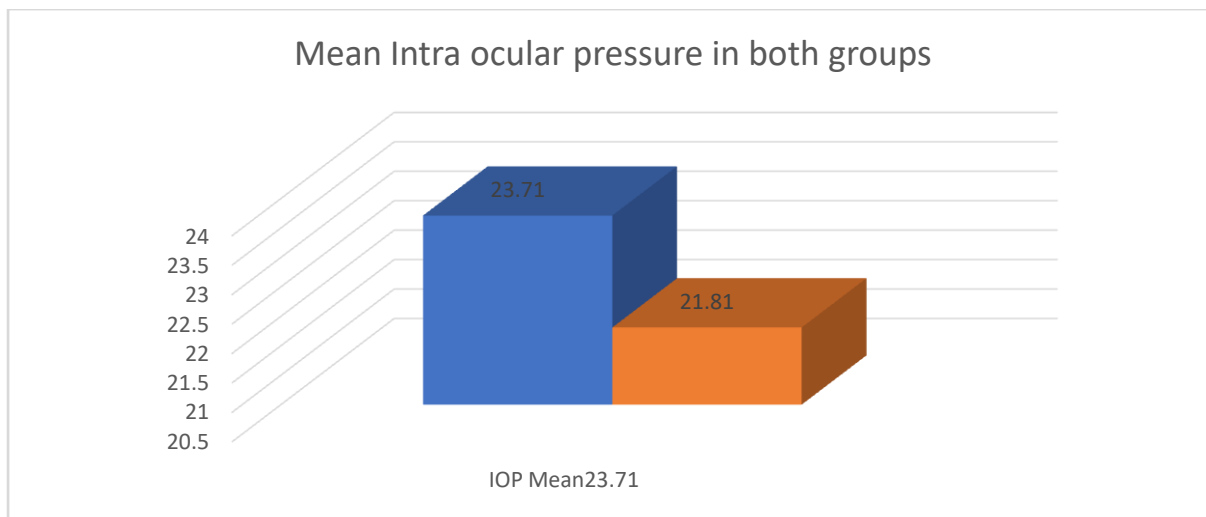
157   The mean average RNFL was **85.85µm** among CCT ≥540 µm which was significantly more as

158   compared to **67.19µm** in CCT <540 µm. Mean Superior RNFL was **85.44 µm** among CCT ≥540

159   µm which was significantly more as compared to **71.59 µm** among CCT < 540 µm. Mean

160   Inferior RNFL was **94.81 µm** among CCT ≥540 µm which was significantly more as compared

161   to **82.22 µm** in CCT < 540 µm. Mean Nasal RNFL was **46.71 µm** among CCT ≥540 µm which

162   was significantly more as compared to **41.19 µm** in CCT < 540 µm. Mean Temporal RNFL was

163   **37.73 µm** among CCT ≥540 µm which was significantly more as compared to **31.13 µm** in

164   CCT < 540. In a cross sectional study of 234 eyes conducted by Barua et al patients were

165   categorized into three groups POAG, ocular hypertensive and normal and it showed GCC and

166   RNFL parameters had equal predictive capability in perimetric versus normal group.[7]Bhat et

167   al conducted a study on 49 patients of POAG and inferred the average RNFL loss in mild,

168   moderate, and severe POAG was 25.44%, 29.67%, and 44.15%, respectively.[8]A statistically

169   significant correlation (P < 0.05) between RNFL loss and severity of glaucoma was found in

170   all except the superior and temporal sectors. A study conducted by Wangsupadilok and

171   associates found a significant correlation was found between CCT and RNFL thickness (r =

172   0.487, p = 0.001).[5]Kaushik et al. discovered that the retinal nerve fiber layer (RNFL) in

173   individuals with ocular hypertension and a central corneal thickness (CCT) of 555 µm or less

174   was significantly thinner compared to those with thicker corneas, as measured by optical

175   coherence tomography (OCT).[9]
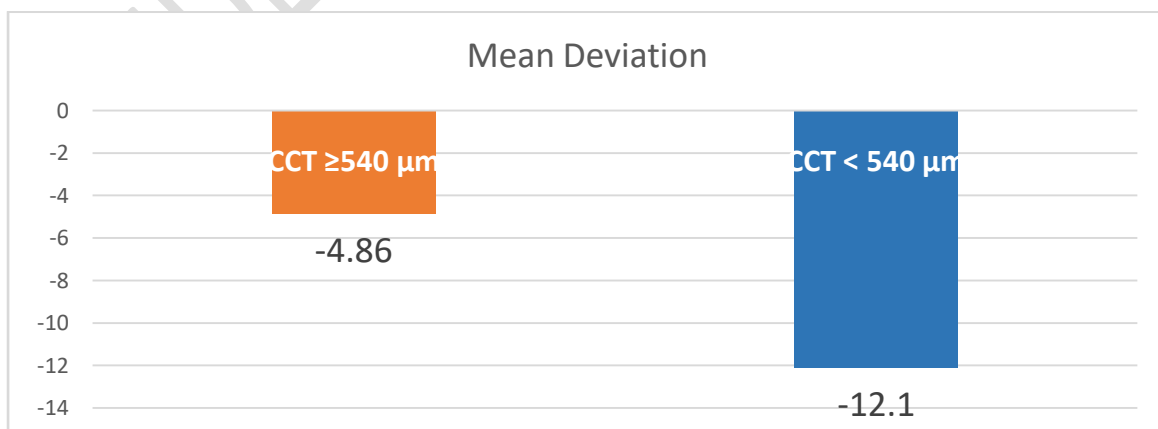
RNFL Thickness

Legend: CCT ≥540 μm, CCT < 540 μm

176

Mean MD was **-4.86dB** among CCT ≥540 μm which was significantly more as compared to -

12.10dB in CCT < 540 μm. Mean PSD was **2.46dB** among CCT ≥540 μm which was

significantly less as compared to **6.92dB** in CCT < 540 μm. Research has shown that eyes

with thinner central corneal thickness (CCT) exhibit more severe visual field (VF) defects

compared to those with thicker CCT. [10]

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Mean Deviation

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184

185 **CONCLUSION**

186 **In conclusion, SD OCT is an effective tool in evaluating the ONH and RNFL thickness to**

187 **detect early to moderate glaucomatous changes. The average RNFL thickness and the**

188 **Superior and inferior RNFL quadrant thickness are the most sensitive parameters to detect**

189 **glaucomatous changes. Both the ONH and RNFL parameters are equally reliable as a**

190 **diagnostic tool but their role in detecting the progression needs to be studied further with**

191 **the long term study. Overall, the study underscores the importance of considering CCT in**

192 **glaucoma assessment and highlights the role of OCT in providing valuable insights into**

193 **RNFL and ONH changes associated with glaucoma. This early detection is crucial for timely**

194 **intervention, helping to monitor glaucomatous damage and potentially reduce the risk of**

195 **blindness associated with the disease.**

196 **LIMITATIONS**

197 The study was undertaken only on known cases of primary open angle glaucoma to

198 evaluate the glaucomatous changes but the efficacy of these parameters in detecting the

199 progression of these changes were not studied. The glaucoma suspects were not included in

200 the study, hence the efficacy to predict future glaucomatous changes with these parameters

201 is not known.The RNFL parameters were not studied for the clock hourwise distribution,

202 hence focal glaucomatous changes could have been.

203

204

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