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Study Of Assessment and Comparison of Patterns of Ganglion Cell Complex in Non-Glaucomatous Optic Neuropathies

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Abstract

AIMS: To assess the thickness of Ganglion Cell Complex (GCC) in Non-Glaucomatous optic neuropathies. To find out if there is reduction in GCC thickness in such patients as compared to that of normal population and to look for the type of pattern of GCC loss.

SETTINGS AND DESIGN: Hospital based cross sectional study

METHODS AND MATERIAL: 19 eyes of 13 patients of non-glaucomatous optic neuropathy and 19 eyes of 15 controls were included. The means of average, superior and inferior hemisphere thickness values of GCC were assessed and compared. Also, the pattern in which the GCC loss occurred was assessed.

STATISTICAL ANALYSIS USED: SPSS 20th version and Microsoft excel 2013

RESULTS: There was statistically significant ($p<0.05$) decrease in GCC thickness among patients for average, superior and inferior hemispheres as compared to controls. The different patterns of loss of GCC among patients was diffuse loss in 11 eyes (57.89%), perifoveal loss in 5 eyes (26.31%), predominantly nasal loss in 2 eyes (10.53%) and crescentic loss in 1 eye (5.26%).

CONCLUSION: GCC scan can be used in Non-Glaucomatous Optic Neuropathies as an additional tool for better diagnosis and effective management. Majority of patients had diffuse type of loss; indicating that GCC does not respect any vertical or horizontal meridian.

Keywords: Ganglion Cell Complex, Non-Glaucomatous, Optic Neuropathy, OCT

INTRODUCTION

Optic neuropathies are characterised by decrease of optic nerve function which can be decrease in visual acuity and/or alteration of the colour vision [1]. They can be broadly classified as Glaucomatous and Non-Glaucomatous Optic Neuropathies. Glaucomatous Optic Neuropathy occurs secondary to Glaucoma leading to typical fundus signs of high cup to disc ratio, nasal shifting of blood vessels, neuroretinal rim thinning, bayonetting sign and typical arcuate scotoma or its classical field defects [2].

Non-Glaucomatous Optic Neuropathies include: - Toxic Optic neuropathy and Nutritional optic neuropathy both being very similar clinically are characterised by painless gradual bilateral visual loss due to damage to papillomacular bundle and loss of colour vision occurring commonly in developing

countries like India [3]. Ischemic Optic neuropathy includes Anterior (Arteritic and Non arteritic) and Posterior Ischemic Optic Neuropathy. It mainly causes apoptosis of retinal ganglion cells and optic nerve atrophy due to ischemia caused at optic nerve head in lamina cribrosa region [4]. In Optic neuritis, axonal degeneration occurs very slowly leading to decrease in retinal nerve fibre layer thickness [5]. Compressive Optic Neuropathy occurs due to mechanical compression of optic nerve at any location from orbit to brain more commonly at optic chiasma due to pituitary tumour [6]. Traumatic Optic Neuropathy occurs due to any direct or indirect trauma to optic nerve which can vary from minimal diminution of vision to complete loss of vision by avulsion [7]. The course of these diseases affects the ganglion cells leading to cell death and eventual thinning of Ganglion Cell Complex. The Ganglion Cell Complex is a collective term used for the layers Internal Plexiform Layer, Ganglion Cell Layer & Retinal Nerve Fibre Layer as measured together by Optovue Optical Coherence Tomography [8,9]. It correlates with visual field defects more strongly than retinal nerve fibre layer measurements. Retinal nerve fibre layer measurements underestimate the true amount of axonal damage due to optic disc edema and retinal nerve fibre layer edema, especially in the early stages [10,11]. Hence ganglion cell complex thinning can be detected earlier than retinal nerve fibre layer thinning [12]. So the measurement of ganglion cell complex can be useful in detecting early axonal damage making it a better predictor of visual outcome [1]. Optical coherence tomography provides two- dimensional cross-sections of the retina with very good spatial resolution rendering qualitative (localization, shape, structure) and quantitative analysis including whole retinal thickness, ganglion cell complex thickness and individual retinal layer thickness. This way, an in vivo "biopsy" of the retina provides information of high quality and resolution about all its layers [1].

Lacunae in current literature - Very few studies have been conducted in patients of non-glaucomatous optic neuropathies. Also, very few studies have evaluated ganglion cell complex in non-glaucomatous optic neuropathies. Most of the previous studies have evaluated ganglion cell complex in individual non glaucomatous optic neuropathy. Studies with all types of non-glaucomatous optic neuropathies taken together are lacking. Even fewer have evaluated the pattern of ganglion cell complex loss in such patients. Also, as per our knowledge, there is hardly any study from Indian population done in such patients.

This study was done in patients of all types of non-glaucomatous optic neuropathies taken together. We have measured the ganglion cell complex thickness for superior half, inferior half and average thickness and compared with that of age matched healthy subjects by spectral domain optical coherence tomography. Also, the pattern of ganglion cell complex loss in eyes of different types of non-glaucomatous optic neuropathies was assessed.

MATERIALS AND METHODS

The study was a Comparative Cross-Sectional Hospital based study in eyes diagnosed with Non-Glaucomatous Optic Neuropathy and Healthy Subjects coming to Mahatme Eye Bank & Eye Hospital Somalwada, Wardha Road, Nagpur, Maharashtra for Ophthalmic Examination. The duration of study was from Ethical Committee clearance (August 2019) to July 2020. The total sample size was 38 eyes (19 each in group of patients and controls). The eyes that were included were of patients diagnosed with Optic Neuropathy and willing to participate for patient group and that of healthy subjects with no ocular pathology for control group.

The eyes that were excluded were of patients with Glaucomatous Optic Neuropathy; patients with hazy media (e.g Corneal Opacity, Cataract, Vitreous Haemorrhage) or in which Optical Coherence Tomography cannot be done with adequate signal strength of more than 30 ;Patients with high refractive error (more than ± 4 D) ;patients with coexisting ocular pathology that affects retinal layer thickness (eg. Diabetes Mellitus, Macular Dystrophy, etc) and patients with age less than 12 years.

The Study Protocol has been approved by local Ethics Committee at Mahatme Eye Bank Eye Hospital, Nagpur in August 2019.

OBSERVATIONS AND RESULTS

Patients and controls were distributed over age ranging from 36 years to 67 years with majority of eyes of patients between age group of 41-50 years. There were 7 eyes in this group which was 36.84% of the total number of eyes (19) of patients.

Volume 4, Issue 5; September-October 2021; Page No 576-582

From control group, majority of eyes were from 51-60 years age group. There were 11 eyes (57.89%) in this age group.

Mean Age of Patients was 50.89 ± 9.049 years while that of Normal Subjects was 51.05 ± 6.485 years.

19 eyes of 13 patients were included in the study, out of which 8 were males and 5 were females and 19 eyes of 15 controls were included, out of which 7 were males and 8 were females. Both eyes of 6 patients were eligible for the study, out of which 3 were males and 3 were females. Both eyes of 4 controls were included in the study, out of which 2 were males and 2 were females.

Statistically significant ($p<0.05$) decrease in Ganglion Cell Complex thickness (mean of Average thickness, Superior half thickness and Inferior half thickness) is seen in patients of Non-Glaucomatous Optic Neuropathy.

Eyes of different types of Non-Glaucomatous Optic Neuropathy that were included in the study were 4 (21.1%) each of Nutritional and Toxic Optic Neuropathy, Post Neuritic Optic Neuropathy, Non Arteritic Anterior Ischemic Optic Neuropathy and Compressive Optic Neuropathy. 2 eyes (10.5%) of Primary Optic Neuropathy and 1 eye (5.3%) of Traumatic Optic Neuropathy were included in the study making it to the total of 19 eyes.This table shows the mean thickness of superior, inferior and average of both hemispheres in eyes of different types of individual non glaucomatous optic neuropathies.

Four different types of patterns of loss of Ganglion Cell Complex were found in the study. Majority of eyes 11 (57.89%) showed Diffuse GCC loss. Other patterns found were Perifoveal loss in 5 eyes (26.31%), Predominantly nasal loss in 2 eyes (10.53%) and Crescentic loss in 1 eye (5.26%).

DISCUSSION

This study was done in 19 eyes of 13 patients of Non-Glaucomatous Optic Neuropathy and 19 eyes of 15 normal subjects (controls). The patients of non-glaucomatous optic neuropathy were diagnosed based on clinical findings and relevant investigations. Optical Coherence Tomography for Ganglion Cell Complex scan was done in patients and controls and the mean of average, superior and inferior thicknesses of Ganglion Cell Complex was compared. Also, the pattern in which Ganglion Cell Complex was lost in different non glaucomatous optic neuropathies was assessed.

Patients and Controls were distributed in the age range from 36 to 67 years with mean age of patients 50.89 ± 9.049 years and that of controls was 51.05± 6.485 years.

In our study, 8 patients were males and 5 were females from patients' group while 7 were males and 8 were females from control group.

Atsuya Miki et al. [13] conducted a study in patients of Non glaucomatous optic neuropathy with mean age of 56 ± 16.6 years. Out of total 14 patients, 9 were females and 5 were males.

Vieira et al. [14] conducted a study in 16 eyes of 8 patients of Toxic and Nutritional Optic neuropathy and 16 eyes of 12 controls. The mean age of patients was 66.0 ± SD of 21.1 years while mean age of controls was 58.2 ±9.3 years. 4 were males while 4 were females in patients' group while 7 were males and 5 were females in control group.

Aggarwal et al. [4] conducted a study in 20 NAAION patients and 25 controls. Mean age of patients' group was 61 ± 14 years while that of controls was 61 ± 6 years. Among the patients' group, 75% were males while 25% were females while among the control group 32% were males and 68% were females.

Marisa G Tieger et al. [15] conducted a study in 23 patients of Compressive Optic Neuropathy and 23 controls. Mean age of patients' group was 52 ± 16 years and that of controls was 51.9 ± 15.8 years. Among the patients' group, 15 were males and 8 were females while among the control group 8 were males and 15 were females.

Iorga et al. [1] conducted a study in patients of non-glaucomatous optic neuropathy patients which included Nutritional and Toxic optic neuropathy, NAAION, Optic Neuritis and Compressive optic neuropathy and found significant thinning ($p<0.05$) in ganglion cell complex in patients.

In our study, mean GCC thickness of average of both hemispheres of patients was 72.40 ± 11.19 microns and that of controls was 98.99 ± 1.99 microns. There was statistically significant ($p<0.05$) decrease in mean of average thickness in patients.

Volume 4, Issue 5; September-October 2021; Page No 576-582

Mean GCC thickness of superior hemisphere of patients was 72.00 ± 12.15 microns and that of controls was 98.60 ± 2.11microns. There was statistically significant ($p<0.05$) decrease in mean of average thickness in patients.

Mean GCC thickness of inferior hemisphere of patients was 72.77 ± 10.67 microns and that of controls was 99.47 ± 2.33 microns. There was statistically significant ($p<0.05$) decrease in mean of average thickness in patients.

Atsuya Miki et al. [13] measured GCC thickness in 11 eyes of non-glaucomatous patients with vision NO Perception of Light. They got mean of average GCC thickness 65.7 micron; superior 67.9 micron and inferior 67.5 micron. All of the above thickness values were lesser than those obtained by us, which were average 72.40 micron; superior 72.00 micron and inferior 72.77 micron. None of the patients in our study had absent perception of light. Thus it can be speculated that GCC thickness and visual acuity are interrelated. However, there exists a significant variability in GCC thickness in normal eyes [16]. Also, floor effect exists for GCC. The floor effect is the minimum thickness of GCC beyond which there is no reduction inspite of perfect loss of retinal ganglion cells.

Vieira et al [14] conducted a prospective observational cross-sectional study in 16 eyes of 8 patients of toxic and nutritional optic neuropathy and 16 eyes of 12 controls. They found decreased retinal ganglion cell layer thickness and volume which supports the proposed pathophysiologic mechanism of toxic and nutritional optic neuropathies that there is toxicity of ganglion cells leading to their apoptosis. This loss is reflected in the values of GCC thickness leading to statistically significant thinning. They found greatest decrease in retinal ganglion cell layer thickness and volume in inferonasal quadrant.

Our study had 4 eyes of toxic and nutritional optic neuropathy which also showed decrease in mean of average, superior and inferior thicknesses of GCC. This is in accordance with the above study. We found 1 eye out of the 4 showing diffuse GCC loss while the other three showed perifoveal GCC loss.

Sahin Muhammed et al. [17] conducted a study in patients of NAAION who were chronically affected with the disease and healthy controls. There were 25 patients and 50 healthy subjects. They found out that there was significant decrease in GCC thickness in patients' group than that of control group and also in unaffected other eyes of patients. The thinning was seen in all quadrants making it a diffuse pattern of loss of Ganglion Cell Complex.

Our study had 4 eyes of NAAION in which we found decrease in mean of Average, Superior and Inferior GCC thickness. Also we found all 4 eyes showed diffuse pattern of GCC loss. This is in accordance with above study.

Aggarwal et al.[4] conducted a study in 25 eyes of 20 patients of NAAION and 25 eyes of Normal age matched subjects. They divided patients in 3 groups based on Visual Field loss patterns into Superior field loss, Inferior field loss and bi-hemisphere field loss. Majority of the patients with superior field loss group had inferior loss of GCC and vice versa. They found a high degree of correlation between visual field loss and GCC loss.

Marisa G Tieger et al. [15] conducted a study on 23 patients with brain tumors with optic chiasmal compression and 23 age matched subjects. In patient group, average GCC thickness showed statistically significant reduction than that of controls. Also, patients with bitemporal field loss showed GCC thinning in all sectors except inferotemporal, attributing to preserved nasal field. They did not find any significant difference in GCC thinning in patients before and after decompression surgeries.

G Cennamo et al [6] conducted a study in patients of pituitary macroadenoma with chiasmal compression (as detected on MRI) and healthy controls. 43 eyes of 22 patients and 36 eyes of 18 controls were included in the study. GCC thickness was measured in all eyes. There was statistically significant reduction in mean of average, superior and inferior thickness of patients than that of controls.

Both above studies suggested a role of GCC in early diagnosis of compressive optic neuropathy so that effective management can be done.

In our study we included 4 eyes of 2 patients of compressive optic neuropathy. There was statistically significant ($p<0.05$) thinning in mean of average, superior and inferior hemispheres of GCC in patients. This is in accordance with above study. However, we

Volume 4, Issue 5; September-October 2021; Page No 576-582

found that 2 eyes showed diffuse pattern GCC loss and 2 eyes showed perifoveal GCC loss.

Akiyasu Kanamori et al. [7] conducted a prospective study in 4 patients of Traumatic Optic Neuropathy where they measured average GCC thickness in affected eyes. They found that GCC thickness was stable at 1 week after injury and started to decrease from 2nd week and plateaued at 20 weeks.

In our study we had 1 patient of traumatic optic neuropathy which came after 10 days (2nd week) of injury. We found decrease in mean of average, superior and inferior GCC thickness. We found crescentic loss of pattern of GCC in the patient.

Our study also had 2 eyes with no secondary cause of optic neuropathy and was diagnosed as primary optic neuropathy. GCC thickness was decreased for mean of average, superior and inferior hemispheres of retina in both the eyes.

The strengths of the study are that this study probed the less explored area of ganglion cell complex in non-glaucomatous optic neuropathies.

This study demonstrated the use of Optical Coherence Tomography for assessing ganglion cells in Non-Glaucomatous Optic Neuropathy, which is a non-invasive objective test demonstrating significant thinning.

Majority of the Non-Glaucomatous Optic Neuropathy patients had diffuse pattern of GCC loss, indicating accelerated ganglion cell loss in them. The loss did not respect any meridian (horizontal or vertical).

The limitations of the study were that this was a single centre study.

It was a cross-sectional study, in which natural course of any disease cannot be extrapolated.

Sample size was small.

People of Indian ethnicity from central India only were included, hence it cannot represent wider population.

Peripapillary Retinal Nerve Fibre Layer thickness was not correlated with Ganglion Cell Complex thickness.

Visual Fields and Ganglion Cell Complex were also not correlated in this study.

Electrodiagnostic tests like Visual Evoked Potential and Electroretinogram were not documented.

CONCLUSION

Ganglion Cell Complex scan of Optical Coherence tomography can be used in Non-Glaucomatous Optic Neuropathies as an additional noninvasive objective investigatory modality for better diagnosis, effective management and explaining the cause of loss of vision in normal looking fundus. Despite varied etiologies of different types of non-glaucomatous optic neuropathies, the common effect is on the ganglion cells leading to accelerated loss and thinning can be seen. The pattern of loss in different types of non-glaucomatous optic neuropathies is diffuse loss in majority of patients indicating that ganglion cell loss does not respect any vertical or horizontal meridian. The loss is not related with etiology of non-glaucomatous optic neuropathy.

RECOMMENDATION

Optical Coherence Tomography is a noninvasive objective investigatory modality which can be used in diagnosis of various diseases. Ganglion Cell Complex thickness scan of Optical Coherence Tomography can be used as an additional diagnostic tool in patients of Non-Glaucomatous Optic Neuropathy. In such patients GCC thinning can be seen which can be useful in effective diagnosis and management.

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Volume 4, Issue 5; September-October 2021; Page No 576-582

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Volume 4, Issue 5; September-October 2021; Page No 576-582

Table 1 : Age distribution of patients and controls

Age range	Patients		Controls	
	No. of Eyes	Percentage	No. of Eyes	Percentage
31-40	3	15.78	2	10.52
41-50	7	36.84	5	26.31
51-60	6	31.57	11	57.89
61-70	3	15.78	1	5.26
Total	**19**	**100**	**19**	**100**

Table 2 : Gender distribution of patients and controls

Gender	Patients	Controls
Male	8	7
Female	5	8
Total	13	15

Table 3 : Mean GCC thickness of patients and controls

Ganglion Cell Complex thickness	Patients		Controls		
	Mean (micron)	SD (micron)	Mean (micron)	SD (micron)	P value
Average	72.40	11.19	98.99	1.99	P=0.001
Superior	72.00	12.15	98.60	2.11	P=0.001
Inferior	72.77	10.67	99.47	2.33	P=0.001

Table 4: Percentage of eyes of different types of Non-Glaucomatous Optic Neuropathy

Type of Optic Neuropathy	Number of Eyes	Percentage (%)
Nutritional and Toxic Optic Neuropathy	4	21.1
Post Neuritic Optic Neuropathy	4	21.1
NAAION	4	21.1
Compressive Optic Neuropathy	4	21.1
Primary Optic Neuropathy	2	10.5
Traumatic Optic Neuropathy	1	5.3
Total	19	100

Table 5: Mean GCC thickness of different types of Non glaucomatous optic neuropathies

Type Of Optic Neuropathy	Ganglion Cell Complex thickness (micron)					
	Average		Superior		Inferior	
	Mean	SD	Mean	SD	Mean	SD
Toxic and Nutritional	75.70	2.57	75.83	2.49	75.62	2.72
Post Neuritic	71.52	2.38	71.24	2.41	71.81	2.44
NAAION	71.88	2.85	71.27	2.89	72.51	2.89
Compressive	72.04	2.68	71.64	2.92	72.42	2.56
Primary	80.14	0.27	78.50	0.16	81.79	0.72
Traumatic	86.04	_	90.36	_	81.71	_

Table 6: Different patterns of GCC loss in patients

Sr. No.	Pattern loss	Eyes	Percentage
1	Diffuse	11	57.89
2	Perifoveal	5	26.31
3	Predominantly Nasal	2	10.53
4	Crescentic	1	5.26
	Total	19	100

Volume 4, Issue 5; September-October 2021; Page No 576-582