

# Compressive and Glaucomatous Optic Neuropathy: A Comparative Study

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## ABSTRACT

This research was aimed to study the etiology of optic atrophy and comparison of optic disc morphology between compressive and glaucomatous optic atrophy. It included 72 cases of optic atrophy admitted in Maharani Laxmi Bai Medical College, Jhansi between January 2016 to May 2017. Assessment of present complaints, examination of the eyes, visual acuity, refraction, perimetry, Optical Coherence Tomography(OCT) and slit lamp examination was done. The quantitative parameters of Optic Nerve Head (ONH) structure were compared using the Spectralis OCT with an enhanced depth imaging method. Out of 72 cases, 42 were males(58.33%) and 30 were females(41.67%). The disease was bilateral in 55 patients(76.39%). The disease manifested as primary optic atrophy in 36 patients(50%), secondary optic atrophy due to papilloedema in 9 cases(12.5%) and due to papillitis in 9 cases(12.5%). Six cases(8.33%) had consecutive optic atrophy and 12 cases(16.67%) had glaucomatous optic atrophy. The main causes were meningitis in 12 cases(16.67%), syphilis in 8 cases(11.1%) and intra-cranial space occupying lesions in 6 cases(8.33%). The mean and maximum cup depths of Compressive optic neuropathy(CON) were significantly smaller than those with Glaucomatous optic neuropathy(GON). The distance between Bruch's membrane opening and anterior surface of the lamina cribrosa (BMO-anterior LC) of CON was also significantly smaller than that of glaucoma. 72 cases of optic atrophy involving 127 eyes have been studied. Measurements of the cup depths and the LC depth showed ability to differentiate between CON with a glaucoma-like disc and glaucoma.

**KEY WORDS:** compressive optic neuropathy, glaucomatous optic neuropathy, optic atrophy, optical coherence tomography

## INTRODUCTION:

Optic Atrophy is the end result of lesions of the visual pathway from ganglion cell layer to the lateral geniculate body. Clinically, optic atrophy is diagnosed from the well known triad of pallor of the optic disc, diminution in the visual acuity and visual field defects<sup>[1]</sup>. Depending upon the histology, etiology and ophthalmoscopic picture, optic atrophy has been classified. The atrophy was classified according to the ophthalmoscopic picture as under<sup>[2]</sup>: [1] Primary Optic Atrophy: pallor of the disc involving the entire disc or temporal pallor extending upto the disc margin, with well defined borders of the papilla; normal

calibre or slight constriction of the bigger vessels and disappearance of the vessels of small calibre. Physiological cup is slightly deeper than normal and lamina cribrosa is seen more clearly. [2] Secondary Optic Atrophy: pallor of the disc with evidence of present or preceding exudation, including obstruction of the physiological cup, irregularity and distortion of the neuro-retinal outlines, veiling of the lamina cribrosa with fibrous or glial tissues which may extend along the retinal vessels. Such a picture may be due to papilloedema or papillitis. Secondary Optic Atrophy is attributed to papilloedema when there are other evidences of raised intra-cranial tension, history of slow gradual progressive loss of vision, and to papillitis when history of sudden loss of vision and no evidence of raised intracranial tension are evident. [3] Consecutive Optic Atrophy: Waxy looking disc with evidence of inflammatory and degenerative changes in the chorio-retinal tissues. [4] Glaucomatous Atrophy Besides the atrophy, the main features are

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deep cupping and displacement of the vessels to the nasal side of the disc. Optic Atrophy may follow injury or inflammation of the optic nerve or raised intracranial tension as in cases of intracranial tumours or raised intraocular pressure as in glaucoma or from local pathological lesions of the retina such as chorioretinitis and primary pigmentary degenerations of the retina. Optic atrophy can be ascending or descending, depending upon the site of the primary lesion which caused the atrophy: e.g. a local lesion such as chorioretinitis or retinal degeneration like primary pigmentary degeneration of the retina or glaucoma would give rise to an ascending optic atrophy; whereas intraorbital or intracranial lesions would give rise to descending optic atrophy. Enlargement of optic disc cupping is a classical sign of glaucoma, but it also can result from non-glaucomatous neurological lesions, such as ischemic optic neuropathy, hereditary optic neuropathy, traumatic optic neuropathy, and compressive optic neuropathy (CON)<sup>[3-5]</sup>. Many reports indicate that intracranial lesions often mimic the clinical presentation of glaucoma and result in misdiagnosis<sup>[5,6]</sup>. Detecting CON among eyes with glaucoma and a glaucoma-like disc is critically important because intracranial lesions, including a brain tumor and intracranial aneurysm, are life-threatening and require treatments that are entirely different from that of glaucoma, and the delay in diagnosis or misdiagnosis can be fatal.

Differentiating glaucomatous from non-glaucomatous disc cupping is often difficult. Trobe and associates showed that pallor of the neuroretinal rim is useful in predicting nonglaucomatous cupping in a review of optic nerve head (ONH) photographs<sup>[4]</sup>. Other clinical findings such as dyschromatopsia or certain visual field changes can help differentiate between the two diseases. The depth of optic disc cupping is considered one of the most important objective findings of the ONH, which helps to differentiate non-glaucomatous optic neuropathy from glaucoma. Mashima and associates compared the ONH between eyes with glaucoma and those with hereditary optic neuropathy using the Heidelberg Retina Tomograph (HRT) parameters<sup>[7]</sup>. They reported that 73% of eyes with hereditary optic neuropathy were misdiagnosed with glaucoma, but the mean cup depth and maximum cup depth of eyes with hereditary optic neuropathy were significantly smaller than those of eyes with glaucoma. However, characteristics of cupping depth for eyes with CON remained unknown. The quantitative analysis of optic disc cupping may provide useful data to distinguish

CON from glaucoma. Additionally, other ONH structures characteristic of glaucoma, such as lamina cribrosa (LC) degeneration<sup>[8]</sup>, are candidates for objective parameters for differentiating sight-threatening and life-threatening optic atrophy from glaucoma. A new approach to optical coherence tomography (OCT), known as enhanced depth imaging (EDI) OCT, allows visualization of deeper layers of the ONH, including the LC<sup>[9,10]</sup>. These studies showed that the LC was located posteriorly and significantly thinner in patients with glaucoma than in healthy controls.

## MATERIALS AND METHODS:

This prospective study included 72 cases of optic atrophy admitted in Maharani Laxmi Bai Medical college, Jhansi between January 2016 to May 2017 and the study was conducted as per the Declaration of Helsinki. Written informed consent was obtained from each participant. **The Objectives of the study included:** [a] to study the types and etiology of Optic Atrophy and [b] comparison of optic disc morphology between Compressive(CON) and Glaucomatous Optic Neuropathy(GON).

**The inclusion criteria:** for CON were: (1) optic atrophy caused by compression of the anterior visual pathway by a brain tumor or aneurysm confirmed by cranial neuroimaging; (2) history of surgical treatment for the causative disease >1 year before this study; (3) mean deviation (MD) measured with the Humphrey Perimetry  $\leq -6$  dB; and (4) IOP  $\leq 20$  mm Hg. The inclusion criteria for open-angle glaucoma subjects were: (1) clinical diagnosis of open-angle glaucoma with, such as a vertical cup-to-disc(C/D) ratio of 0.7 or greater, asymmetry of  $\geq 0.2$ , or the presence of focal thinning, notching, and disc hemorrhage, and associated glaucomatous loss of visual field; (2) glaucomatous visual field loss in Perimetry with 24-2 SITA program on at least 2 subsequent tests; (3) treated IOP  $\leq 20$  mm Hg; and (4) MD in Perimetry  $\leq -6$  dB.

**The Exclusion criteria were:** (1) high myopia ; (2) astigmatism  $>3$  D; (3) any disease, including media opacity, diabetic retinopathy, ischemic optic neuropathy, optic neuritis, uveitis and trauma; (4) tilted optic disc, which was defined as an index of tilt (ratio of minimum to maximum optic disc diameter)  $< 0.75$  on stereodisc photography and (5) a history of intraocular surgery or laser treatment except for uncomplicated cataract surgery.

An assessment of present complaints, ophthalmological check up as external examination of the eyes, visual acuity, refraction, ophthalmoscopy, perimetry, Optical Coherence Tomography(OCT) and slit lamp examination was done. The quantitative parameters of Optic Nerve Head(ONH) structure were compared using the Spectralis OCT with an enhanced depth imaging(EDI) method. The EDI technique was also used for measuring the prelaminar tissue thickness(PLT), diameter of Bruch's membrane opening (BMO), and distance between BMO and the anterior surface of the LC (BMO-anterior LC). The details and advantages of this technology for evaluating the LC have been described previously<sup>[11]</sup>. The PLT was defined as the distance between the optic cup surface and the anterior border of the highly reflective region that corresponded to the LC. The BMO was defined as the termination of the Bruch's membrane, and we measured the diameter of BMO<sup>[12]</sup>. The BMO-anterior LC was defined as the vertical distance between the reference line connecting BMO and the anterior lamellar surface. In principle, we measured the PLT, diameter of BMO, and BMO-anterior LC at the midpoints between the BMOs on both vertical and horizontal images. An average value was obtained from two images. Medical check up

including general physical, neurological, respiratory and cardio-vascular examination were carried out in each case. Clinical investigations with complete blood count(CBC), Mantoux test, serological test for syphilis and complete urine and stools examination were done. In addition complete cerebrospinal fluid examination, skiagrams of skull and nasal sinuses and chest screening were done, when indicated.

**Statistical Analysis:** The patient's protocols were recorded in data collection form. Quantitative data were expressed as mean±SD(standard deviation) and qualitative variables were expressed using percentages. We applied Student's unpaired 't' test for equal or unequal variances, after calculating the variance of each data groups respectively. The p-value of < 0.05 for one - tailed hypothesis was considered statistically significant to reject the 'null hypothesis'. All statistical analyses were made with the help of data analyses tool of Microsoft Excel 2007. The data shown in the tables herein are based on extensive data set used in our detailed study.

## RESULTS:

Out of 72 cases, 42 were males(58.33%) and 30 were females(41.67%) (Table I). The incidence of the disease was more in first four decades of life. The

**Table 1:** Demography and etiology of optic atrophy in patients (n=72).

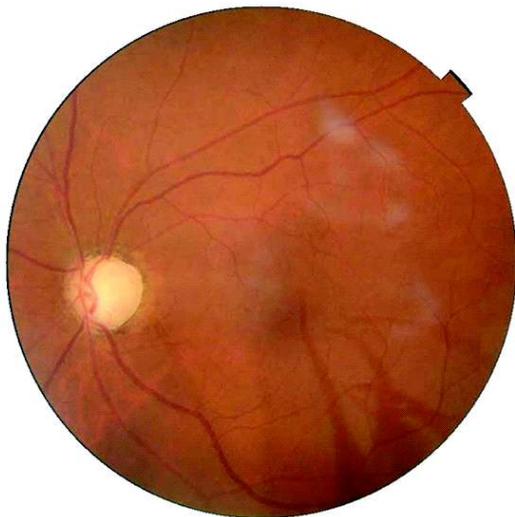
Age in year (Mean±SD)		52.6±16.3			
(Range 5 -75 years )					
Gender (Male/Female)		42 (58.33%)/ 30(41.67%)			
Optic atrophy	Unilateral	17(23.61%)			
	Bilateral	55(76.39%)			
		Ophthalmoscopic classification of optic atrophy			
Etiology of optic atrophy (no. of cases)	Primary	Secondary		Consecutive	Glaucomatous
		Papilloedema	Papillitis		
Unknown (12)	10	-	2	-	-
Syphillis (8)	7	-	1	-	-
Trauma (5)	5	-	-	-	-
Demyelinating diseases (5)	3	-	2	-	-
Space occupying intracranial lesions (6)	2	4	-	-	-
Tubercular meningitis (8)	3	2	3	-	-
Patchy meningitis (4)	1	2	1	-	-
Cranio stenosis (2)	1	1	-	-	-
Anaemia (2)	2	-	-	-	-
Hydrocephalus (2)	2	-	-	-	-
Choroidal sclerosis (3)	-	-	-	3	-
Retinitis pigmentosa (3)	-	-	-	3	-
Total (72)	36	9	9	6	12

**Table 2:** Comparison of OCT parameters among compressive and glaucomatous optic neuropathy.

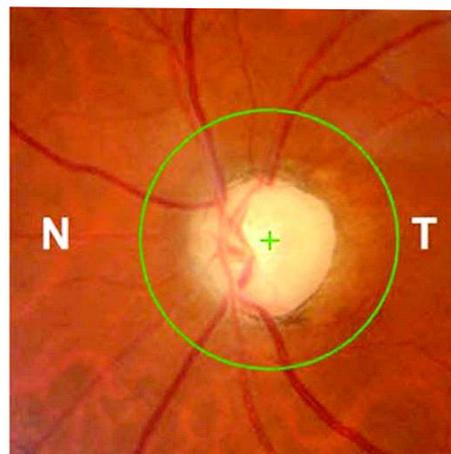
	Group 1 (CON)	Group 2 (GON)	p-value
RNFL ( $\mu\text{m}$ )	51.8 $\pm$ 14.6	57.2 $\pm$ 12.1	0.04
Mean PLT ( $\mu\text{m}$ )	111.3 $\pm$ 54.2	108.1 $\pm$ 56.0	0.75
BMO width ( $\mu\text{m}$ )	1602 $\pm$ 162.1	1611 $\pm$ 159.3	0.90
Disk size (mm)	2.24 $\pm$ 0.56	2.36 $\pm$ 0.59	0.42
C/D area ratio	0.32 $\pm$ 0.12	0.36 $\pm$ 0.12	0.01
BMO-anterior LC ( $\mu\text{m}$ )	364 $\pm$ 120.1	498 $\pm$ 156.2	0.004
Mean cupping depth (mm)	0.18 $\pm$ 0.08	0.36 $\pm$ 0.12	0.004
Maximum cupping depth (mm)	0.48 $\pm$ 0.15	0.73 $\pm$ 0.19	0.003

**Table 3:** Baseline characteristic of patients.

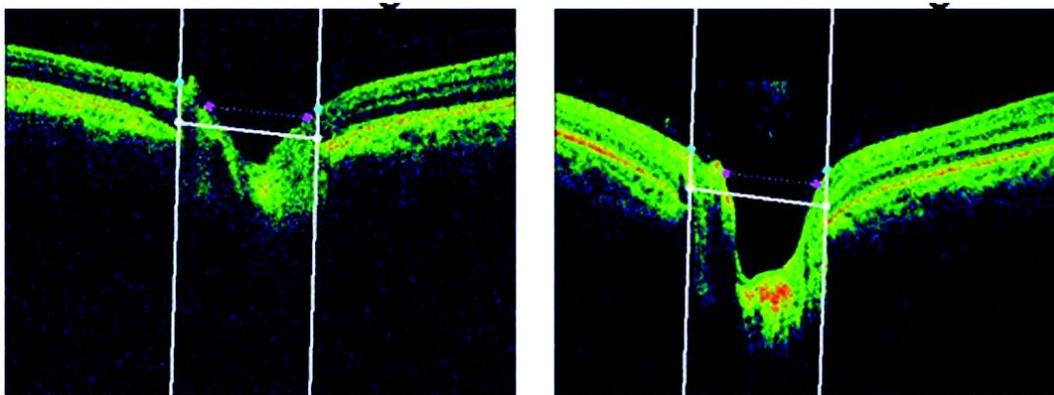
	Group 1 (CON)	Group 2 (GON)	p-value
Number of eyes	24	24	-
Age in years (Mean $\pm$ SD)	56.2 $\pm$ 14.1	57.3 $\pm$ 4.6	0.92
Gender (M/F)	8/4	7/5	-
Spherical equivalent (DIOPTRE)	-2.1 $\pm$ 2.2	-2.0 $\pm$ 2.1	0.32
Axial length (mm)	24.2 $\pm$ 1.0	24.1 $\pm$ 1.3	0.53
IOP (mmHg)	15.8 $\pm$ 3.2	16.3 $\pm$ 3.8	0.50
Mean deviation (dB ) (Perimetry)	-16.8 $\pm$ 7.3	-17.5 $\pm$ 6.5	0.41

**Figure 1:** Fundus photograph showing secondary optic atrophy (due to Compressive Optic Neuropathy) in left eye of a male patient aged 52 years diagnosed with meningioma.

disease was bilateral in 55 patients (76.39%). In unilateral cases (17 cases, 23.61%), right and left eyes were involved equally. The disease manifested as primary optic atrophy in 36 patients (50%), secondary optic atrophy due to papilloedema in 9 cases (12.5%) and due to papillitis in 9 cases (12.5%). 6 cases (8.33%) had consecutive optic atrophy while 12 cases (16.67%) had glaucomatous optic atrophy. In 12 cases (16.67%)

**Figure 2:** Fundus photograph showing glaucomatous optic atrophy (GON) in left eye of a female patient aged 57 years.

no cause could be detected. Main diagnosis were meningitis 12 cases (16.67%), syphilis 8 cases (11.1%), intracranial space occupying lesions 6 cases (8.33%), demyelinating process 5 cases (6.9%), trauma 5 cases (6.9%), choroidal sclerosis 3 cases (4.1%) and pigmentary degeneration of retina 3 cases (4.1%). The mean and maximum cup depths of Compressive optic neuropathy (CON) were significantly smaller than those with Glaucomatous optic neuropathy (GON) (P=0.004 and 0.003 respectively) (Table 2). The distance between Bruch's



**Figure 3:** Optic nerve head parameters measured with Optical Coherence Tomography (3a. Compressive Optic Neuropathy, 3b. Glaucomatous Optic Neuropathy).

membrane opening and anterior surface of the lamina cribrosa (BMO-anterior LC) of CON was also significantly smaller than that of glaucoma ( $p=0.004$ ). The cup/disc area ratio also showed significance between cases of CON with a glaucoma-like disc and cases of GON ( $p=0.01$ ).

Moreover, 12 subjects with CON and 12 cases of GON subjects were initially included (24 eyes of 12 subjects in each group A and B). Of 12 subjects with CON, 3 had pituitary adenoma, 2 had intracranial meningioma and 1 had craniopharyngioma. The MD of the visual field was  $-16.8 \pm 7.3$ ,  $-17.5 \pm 6.5$  in group A and B respectively (Table 3). There was no significant difference in the MD between the CON and glaucoma groups ( $p=0.41$ ). There were no significant difference in the diameter of the BMO and disc size in OCT measurements among both groups. The mean RNFL (retinal nerve fibre layer) of the group A was significantly thinner than that of the glaucoma group ( $p=0.04$ ) (Table 2). The C/D area ratio of group B was significantly greater than that of group A ( $p=0.01$ ). The mean PLT of group A was almost the same as that of group B ( $p=0.75$ ). Of 24 eyes with CON, 10 eyes (41.7%) presented with a C/D ratio  $\geq 0.8$ . The BMO-anterior LC and the mean and maximum cup depths of CON subjects with a glaucoma-like disc were smaller than those of the glaucoma group ( $p=0.004$ ,  $p=0.004$ , and  $p=0.003$ , respectively).

## DISCUSSION:

There exists variation in the incidence of various etiological factors in optic atrophy depending on the prevalence of a particular disease in a particular era and area. Some of the causes like intra-cranial space occupying lesions, craniostenosis, retinal degeneration and tumours of the optic nerve are self

explanatory whereas in others, more common causes like syphilis, tuberculosis and demyelinating diseases, atrophy of the optic nerve ensues differently. In children, the most important cause of bilateral optic atrophy is tubercular meningitis, and meningoencephalitis whereas unilateral atrophy is mostly of traumatic nature.

In his study of ocular aspect of tuberculosis, Mooney<sup>[13]</sup> found three types of lesions giving rise to optic atrophy.

(a) Edema of brain and meninges leading to obstruction of the ventricular foramina in the posterior cranial fossa producing internal hydrocephalus and manifesting as papilloedema; (b) Granulation tissue or fibrous bands around the chiasma and optic nerve causing strangulation of nerve fibres manifesting as primary optic atrophy; (c) Interstitial perineuritis extending upto the disc manifesting as papillitis and neuro-retinitis.

In our study 8 cases (11.1%) of tubercular meningitis (6 children below the age of 10 years) presented with papilloedema, papillitis and primary optic atrophy in equal distribution, none of them developed choroidal tubercles during any stage of the disease. In 8 cases syphilis was responsible for the optic atrophy which was primary in 7 cases and post neuritic following papillitis in 1 case. Out of these 5 cases (62.5%,  $n=8$ ) were above 50 years of age. In 5 cases, atrophy was due to trauma to the temporal region. In none of these cases any fracture could be detected on the base of skull and the optic canal. Trauma was generally of minor to moderate intensity producing sudden loss of vision followed by primary type of atrophic changes within a period of 4 weeks. This is believed to be due to hemorrhage in the sheath of the optic nerve or haematoma pressing the nerve fibres producing pressure atrophy. The incidence of

demyelinating disease is believed to be low in our country. In 6.9% of cases, of this study the optic atrophy was thought to be due to demyelinating diseases on clinical grounds. Recently, more stress has been laid on the demyelinating diseases as the cause of unilateral or bilateral optic atrophy. Hierons<sup>[14]</sup> suggested a localized form of encephalomyelitis responsible for optic atrophy whereas Scott<sup>[15]</sup> and Stansbury<sup>[16]</sup> reported neuromyelitis optica responsible for bilateral retrobulbar neuritis and papillitis with or without transverse myelitis. Thus isolated primary or secondary optic atrophy can occur due to demyelinating pathology.

Compared to eyes with glaucoma, eyes with CON had a shallower cup depth. Additionally, 50% of eyes with CON presented with enlargement of the optic cup on fundus photographs, and 42% were judged to have a glaucoma-like disc. For distinguishing CON with a glaucoma-like disc from glaucoma, ONH parameters specifically a shallow maximum cup depth, increase the likelihood of identifying an intracranial mass lesion. This study elucidated the difference in ONH deeper structures between glaucoma and CON. Glaucomatous cupping has been histologically shown to result from the loss of both axons and astroglia in the optic disc<sup>[17]</sup> and posterior LC displacement and thinning of the LC<sup>[18]</sup>. Portney and associates conducted a pathological study in a case of disc cupping associated with compression of the optic nerve and showed loss of axons and reported that glial tissue within the optic nerve head would account for the cupping<sup>[19]</sup>. Our study showed that eyes with CON had a smaller LC depth than eyes with glaucoma. Previous studies have reported that eyes with glaucoma presented with posterior LC displacement and a thinner LC than normal eyes<sup>[20]</sup>. Our results indicated that retrograde axonal degeneration caused by optic nerve compression does not accompany laminar remodeling, which is unlike that observed in glaucomatous optic neuropathy. This result was consistent with the hypothesis that the pathogenesis of optic nerve degeneration in glaucomatous optic neuropathy is located at the LC, while the pathogenesis of other optic neuropathies is not<sup>[21]</sup>.

## CONCLUSION:

Measurements of the cup depths and the LC depth in OCT showed ability to differentiate between CON with a glaucoma-like disc and glaucoma (GON). A further prospective study should be performed for a larger population. However, quantitative analysis of

ONH morphology by OCT provides the necessary evidence to distinguish cupping caused by intracranial lesions from that caused by glaucoma.

## LIMITATIONS OF THE STUDY:

This study has the limitation of small sample size and as diagnostic tests are indicated for use in patients with suspected disease and not in patients with confirmed CON or glaucoma. However, we compared only patients who had already been diagnosed with CON or glaucoma.

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