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Investigation of optic disc oedema cases: A clinical profile and visual field pattern analysis

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Abstract

Introduction and Background: Clinical manifestations of optic disc oedema (ODE) include papilledema, optic neuritis, and ischaemic optic neuropathy, among other ocular and systemic diseases. The purpose of this perimetry-based study is to examine the clinical characteristics of ODE patients and to describe their visual field patterns.

Materials and Methods: In a hospital-based observational research, 120 individuals who had been diagnosed with optic disc oedema were examined. This study was conducted at the department of Ophthalmology, SS Institute of Medical Sciences, Davangere, Karnataka, India from the February 2014 to January 2015. Extensive eye and systemic exams were performed on patients. We analysed the visual field defect patterns of patients and classified them according to the aetiology of ODE, such as papilledema, optic neuritis, or anterior ischaemic optic neuropathy (AION). To determine if there was an association between clinical characteristics and visual field abnormalities, statistical comparisons were carried out.

Results: Papilledema accounted for 40% of the 120 cases with optic disc oedema, followed by optic neuritis at 30% and AION at 20%. Hypertensive optic neuropathy (10%), toxic optic neuropathy (5%), and diabetic papillopathy were among the less common causes. Enlarged blind spots were seen in 85% of papilledema cases and inferior nasal abnormalities in 52% of cases. In 78% of patients, scotomas were located centrally or centrocecally in optic neuritis. Altitudinal deficiencies were observed in 64% of AION cases, with inferior altitudinal loss accounting for 42% of those cases. Diffuse field depression was one of the patterns seen in the remaining cases (10%).

Conclusion: Different causes of optic disc oedema cause different visual field patterns. The majority of cases are caused by papilledema, which manifests as larger blind patches. Optic neuritis, on the other hand, causes central scotomas, while AION shows altitudinal abnormalities. To help diagnose and treat optic disc oedema quickly, telltale signs of the condition in the visual field should be looked for as soon as possible.

Keywords: Optic disc edema, visual field defects, papilledema, optic neuritis, ischemic optic neuropathy

Introduction

Inadequate diagnosis and treatment of optic disc oedema (ODE), a common clinical finding in a variety of ocular and systemic diseases, can result in permanent vision loss. This condition develops when the optic nerve fibres enlarge at the axonal level, which is mostly due to a blockage of the axoplasmic flow [1-3]. Many different ailments and injuries can lead to optic nerve inflammation or elevated intracranial pressure (papilledema), as well as vascular traumas, chemical exposures, and systemic diseases like diabetes and hypertension. Correct diagnosis and treatment depend on a thorough understanding of the patient's clinical presentation and any related visual field abnormalities [3-5].

Assessing the functional impact of ODE relies heavily on visual field evaluation. Disc oedema can have many causes, each of which manifests differently in the visual field; hence, perimetry is a crucial diagnostic technique. Optic neuritis, caused by inflammatory demyelination of the optic nerve, usually manifests as central or centrocecal scotomas [6-8], whereas papilledema, which is often linked to elevated intracranial pressure, is marked by enlarged blind patches and inferior nasal field abnormalities. In contrast, altitudinal visual field abnormalities are a common result of AION, which is a reflection of the segmental nature of the optic nerve head's ischaemic injury.

The diagnostic approach can be made more complicated when other disorders, such toxic or metabolic optic neuropathies, cause arcuate abnormalities or broad field depression [7-9].

The early identification of functional and structural alterations in ODE has grown more accurate with the use of standard automated perimetry (SAP) and optical coherence tomography (OCT). Optical coherence tomography (OCT) measures optic nerve injury objectively by measuring the thickness of the retinal nerve fibre layer (RNFL), and simultaneous angiography (SAP) measures visual field loss quantitatively. Nevertheless, additional research is required to develop more accurate diagnostic and prognosis indicators due to the fact that the clinical manifestation and visual field abnormalities caused by ODE might vary widely [8-10].

In order to improve diagnosis accuracy, this study intends to assess the visual field patterns and clinical profile of individuals with optic disc oedema, comparing perimetric data with RNFL thickness. In order to better recognise optic disc oedema and its causes early on, monitor the condition, and manage it, this research aims to uncover distinct patterns of visual field abnormalities and how they correlate with structural alterations [9-11].

Material and Methods

An observational research conducted in a hospital involved 120 individuals diagnosed with optic disc oedema. This study was conducted at the department of Ophthalmology, SS Institute of Medical Sciences, Davangere, Karnataka, India from the February 2014 to January 2015. Patients received comprehensive ocular and systemic assessments. Patients were classified according to the aetiology of ODE, and their visual field defect patterns were examined. Statistical analyses were used to evaluate relationships between clinical characteristics and visual field impairments.

Inclusion Criteria

- Adults with clinically confirmed optic disc edema.
- Patients presenting with visual symptoms or incidentally detected ODE.
- Documented optic disc swelling on fundus examination or OCT.
- Willingness to undergo perimetry, OCT, and relevant diagnostic tests.
- Stable systemic condition allowing proper ophthalmic assessment.

Exclusion Criteria

- Media opacities interfering with visual field/OCT.
- History of glaucoma, prior optic neuropathy, or retinal disease.
- Uncontrolled systemic illnesses affecting visual function.
- Neurological conditions impairing perimetry reliability.
- Recent ocular trauma or intraocular surgery.

Results

The study found that optic disc oedema (ODE) could have many causes among the 120 individuals who participated. First, papilledema (40%), then optic neuritis (30%), and finally anterior ischaemic optic neuropathy (AION) (20%) were the most common causes. Diabetic papillopathy, hypertensive optic neuropathy, and toxic optic neuropathy were among the other disorders that caused ODE in the 10% of patients. The underlying cause of ODE was compatible with the visual field abnormalities found by standard automated perimetry (SAP).

Table 1: Distribution of Causes of Optic Disc Edema

Cause of Optic Disc Edema	Number of Patients (n=120)	Percentage (%)
Papilledema	48	40
Optic Neuritis	36	30
AION	24	20
Other Causes	12	10

The study group (n=120) had optic disc oedema (ODE) for a variety of reasons, as shown in the table. Optic neuritis (30%), anterior ischaemic optic neuropathy (AION) (20%), and papilledema (40%) were the most common causes of optic nerve epilepsy (ODE). Other causes, such as hypertensive optic neuropathy and diabetic papillopathy, accounted for a smaller percentage of cases (around 10%). This distribution illustrates the variety of causes that can lead to optic disc oedema.

Table 2: Visual Field Defect Patterns by Etiology

Etiology	Enlarged Blind Spot (%)	Inferior Nasal Defects (%)	Central/Centrocecal Scotoma (%)	Altitudinal Defects (%)	Diffuse/Arcuate Defects (%)
Papilledema	85	52	0	0	0
Optic Neuritis	0	0	78	0	22
AION	0	0	0	64	36
Other Causes	0	0	33	0	67

The visual field defect patterns that are related with each aetiology of optic disc oedema are presented in this table. The most common type of papilledema defect is an enlarged blind spot, which accounts for 85% of cases. Second most common are inferior nasal defects, which account for 52% of cases and are often associated with disorders involving increased intracranial pressure. Arcuate deficits (found in 22%) and central or centrocecal scotomas (78%) were the most common visual symptoms of optic neuritis, suggesting demyelination of the optic nerve.

Table 3: Retinal nerve fiber layer thickness in different etiologies

Etiology	RNFL Thickness (µm)±SD	Statistical Significance (p-value)
Papilledema	176.4±24.8	$p<0.001$
Optic Neuritis (acute)	159.2±18.3	$p = 0.045$
Optic Neuritis (chronic)	141.5±16.1	$p<0.05$
AION	132.8±20.5	$p = 0.003$
Other Causes	145.2±19.7	$p>0.05$

Patients with optic disc oedema caused by various sources have their retinal nerve fibre layer (RNFL) thickness compared in the table. The increased RNFL thickness in papilledema patients, with a mean of 176.4±24.8 µm (p<0.001), suggests that the optic disc is swelled as a result of the elevated intracranial pressure. The RNFL thickened moderately in acute ocular neuritis (159.2±18.3 µm, p = 0.045), but it was significantly thinner in chronic optic neuritis, indicating injury to the optic nerve over a longer period of time.

Discussions

One of the most notable clinical signs of several ocular and systemic diseases is optic disc oedema (ODE). Clinical examination, visual field analysis, and imaging techniques like Optical Coherence Tomography (OCT) are essential for accurate diagnosis and management. The purpose of this study was to compare the visual field defect patterns and clinical profile of patients with optic disc oedema to the thickness of the retinal nerve fibre layer (RNFL) in order to increase the accuracy of diagnostics [12-14].

Papilledema accounted for 40% of the instances of ODE in our cohort, making it the most prevalent cause. Consistent with previous research, this suggests that increased intracranial pressure (ICP) is the primary factor responsible for optic disc oedema. Impairment of axoplasmic flow resulting from elevated intraocular pressure (ICP) is associated with the distinctive inferior nasal visual field abnormalities and larger blind patches seen in papilledema patients. On the other hand, 30% of cases were caused by optic neuritis [15-17].

This finding is consistent with previous research showing that this condition, especially in younger people, can lead to inflammation and demyelination of the optic nerve, which in turn can produce optic disc oedema. Our study's patients with optic neuritis had central and centrocecal scotomas, which are common field abnormalities caused by inflammation of the optic nerve [16-18]. Twenty percent of patients had anterior ischaemic optic neuropathy (AION), a leading cause of optic disc oedema, particularly in elderly people with vascular risk factors. We observed altitudinal visual field abnormalities in 64% of our AION patients, which is a frequent complication of the condition. The discovery highlights how the disorder is ischaemic, meaning that blood supply to the optic nerve head is impeded, leading to visual loss in the affected field [17-19].

This study's visual field patterns match the pathophysiology of all the underlying conditions. Enlarged blind spots and inferior nasal abnormalities, indicative of elevated intracranial pressure, are the most common symptoms of papilledema. On the other hand, central or centrocecal scotomas were linked to optic neuritis, which is characterised by demyelinating of the optic nerve and consequently disrupts central vision. Ischaemia damages specific segments of the optic nerve head, which is the established vascular aetiology of AION [18-20].

Patients with this syndrome also have altitudinal vision field abnormalities.

Patients with other ODE causes, including hypertensive optic neuropathy or diabetic papillopathy, had more widespread or curved abnormalities, which could be a reflection of the optic nerve's larger influence from these systemic diseases. Even though these patterns aren't as exact as others, they help narrow down the possible causes of optic disc oedema [19-21].

The substantial association between RNFL thickness and visual field abnormalities was one of the most important findings of this investigation. The increased RNFL thickness (176.4±24.8 µm) observed in papilledema is a sign of optic nerve swelling caused by elevated intracranial pressure. This agrees with prior research showing that an enlarged optic disc is associated with a thickened RNFL [20-22].

During the acute phase, patients with optic neuritis showed RNFL thickness of 159.2±18.3 µm, which is probably a result of the inflammatory reaction and swelling surrounding the optic nerve. Nevertheless, after the acute inflammatory process fades away, the RNFL thickness in chronic instances decreases to 141.5±16.1 µm, indicating axon loss and nerve atrophy [21-23]. The optic nerve fibres were found to have ischaemic damage since the RNFL thickness was the lowest in AION patients, measuring 132.8±20.5 µm. Patients with AION have thinner RNFLs because inadequate blood supply to the optic nerve head causes persistent axonal injury. Contrary to the more damaging processes observed in papilledema, optic neuritis, and AION, other sources of ODE had rather steady RNFL thickness, which could indicate less severe or reversible damage [22-24].

In order to get a better picture of optic disc oedema, this study shows that it's worth it to combine visual field testing with structural OCT measures. Measuring the RNFL thickness provides objective proof of the optic nerve's structural integrity, whereas visual field abnormalities give functional insights. Together, they can help narrow down the possible causes of optic disc oedema and direct therapy choices [23-25]. In addition, the study's trends highlight the importance of a multidisciplinary strategy for ODE diagnosis. In addition to aiding in the management of the illness, determining the aetiology of ODE can help avoid possible visual loss. In the case of elevated intracranial pressure (ICP), early detection of papilledema allows for faster intervention, and in the case of optic neuritis, correct therapy can avert irreversible vision loss [24-26].

The limitations of this study should not be overlooked, since they offer important insights into the visual field and clinical features of optic disc oedema. Additional research with bigger cohorts is required because the sample size of 120 patients could not be representative of the overall population [27-29]. Furthermore, while SAP was used to evaluate visual field defects, more recent methods like frequency-doubling technology and short-wavelength automated perimetry may be more sensitive to early changes in the visual field. To determine how visual field abnormalities and RNFL thinning develop over time and how they relate to clinical outcomes, longitudinal investigations are necessary [28-30].

Conclusion

Finally, this study highlights how crucial it is to diagnose optic disc oedema with a combination of clinical evaluation, visual field analysis, and OCT imaging. Clinicians can use the diagnostic signals provided by the characteristic visual field abnormalities and RNFL thickness patterns seen in ODEs of different sources to assist them decide how to treat their patients. Improved early detection and prognosis for individuals with optic disc oedema, as well as the refinement of diagnostic criteria, call for additional research.

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None

Conflict of Interest
None

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