ECRONICON OPEN ACCESS

EC OPHTHALMOLOGY
Research Article

Evaluation of Underlying Etiological Factors in Patients with Papilledema in Rural Karnataka

Sri Archana[1], Chaitra MC[2]* and Rashmi G[3]

[1]*Senior Resident, Department of Ophthalmology, Yenepoya Medical College, Mangalore, India*

[2]*Associate Professor, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamka, Kolar, India*

[3]*Associate Professor, Department of Ophthalmology, SDUMC, Kolar, India*

***Corresponding Author:** Chaitra MC, Associate Professor, Department of Ophthalmology, Sri Devaraj Urs Medical College, Tamka, Kolar, India.

Received: January 13, 2025; **Published:** February 13, 2025

Abstract

Aims: Papilledema diagnosis is found to be an important fundus finding which may indicate a systemic pathology. Various studies have revealed that papilledema if left untreated can lead to progressive irreversible loss of vision and in upto 31% of the patients secondary optic atrophy. In literature few unusual causes of papilledema case series are present [1], regarding the importance of neuroimaging in papilledema [2] are given, but there is only one other study which was conducted to look for the etiological factors and visual field defects in papilledema [3]. Hence this study aims at determining the etiological factors in patients with papilledema in a rural population.

Material and Methods: Hospital based prospective, observational case series, study was conducted for a duration of two years.

Results: Women were found to be more affected than males (39 males and 54 women). Of the 93 patients cerebral infection (40%) was found to be the most common cause, other causes included seizure disorder 17%, intracranial space occupying lesion 10%, cerebral venous thrombosis in six percent, nine percent were suspected to have idiopathic intracranial hypertension (IIH). Early papilledema (84%) was seen as the most common type followed by established and chronic papilledema.

Keywords: *Papilledema; Space Occupying Lesions; IIH; Cerebral Infection*

Introduction

Papilledema (optic disc swelling) is the most common manifestation of increased intracranial pressure (ICP) which usually develops within hours to several days from the onset of the disease [4,5]. But certain times if this finding is not present it does not rule out an intracranial lesion [2]. Patient diagnosed with papilledema it has been found that there are various causes and some the causes are intracranial space occupying lesions (ICSOL), Idiopathic intracranial hypertension (IIH), various drugs such as tetracycline, nalidixic acid, vitamin a, steroids and contraceptive pills may produce IIH [6], reduced cerebrospinal fluid drainage (e.g. venous sinus thrombosis, inflammatory processes, meningitis, subarachnoid hemorrhage) and increased production of CSF due to tumors. Headache which is worse on waking up and is aggravated by coughing, nausea and vomiting, poor colour perception, transient obscurations of vision, flickering sensation and diplopia are the presenting symptoms of papilloedema [6]. Fundus examination signs are blurring of the margins of the

optic disc, filling of the optic cup, folds in retina and choroid, venous congestion, splinter hemorrhages, cotton-wool spots, absence of venous pulsations and hyperemia of the optic nerve head [6]. The most common visual field defect seen is enlargement of the blind spot [7]. By definition, papilledema cannot be present in the absence of raised ICP, but raised ICP can occur in the absence of papilledema. It is commonly bilateral and symmetric, but may be asymmetric or unilateral. Management is directed at correcting the underlying cause. The available options include both medical and surgical modalities. The mainstays for treatment of IIH is weight loss and diuretics and surgery is reserved for those who fail, are intolerant to, or non-compliant with maximum medical therapy [1].

Pathogenesis

Monro-Kellie doctrine is the pressure-volume relationship between ICP, volume of cerebrospinal fluid (CSF), blood, brain tissue, and cerebral perfusion pressure. Since the total volume inside the cranium is fixed, any rise in volume of one of the cranial constituents must be compensated for by a reduction in volume of the others or it will lead to a high ICP. ICSOL often causes intracranial hypertension, which leads to papilledema [8].

Therefore, raised ICP may occur by any one or combination of the following mechanisms: an increase in the total amount of intracranial tissue by a space-occupying lesion, an increase in intracranial tissue volume by focal or diffuse cerebral edema; an increase in production of CSF; a decrease in total available volume within the cranial vault by thickening of the skull; a decrease in the outflow of CSF within the ventricular system (e.g. obstructive or non-communicating hydrocephalus) or within the arachnoid granulations (e.g. meningitis, subarachnoid hemorrhage [SAH]); and a decrease in the absorption of CSF from intracranial or extracranial obstruction or compromise of venous outflow (e.g. venous sinus thrombosis). Another probable mechanism of high ICP in IIH is high intra-abdominal pressure, which may increase pleural pressure and cardiac filling pressure, leading to increased intracranial venous pressure and high ICP.

Causes of increased intracranial pressure [9]

Space occupying lesions	Reduction in size of cranial vault
Abscess	Cranio-synostosis
Hemorrhage	Thickening of skull
Arteriovenous malformation	Blockage of CSF flow
Focal or diffuse cerebral edema	Non communicating hydrocephalus
Trauma	Reduction in CSF reabsorption
Toxic	Communicating hydrocephalus
Anoxia	Meningitis
Elevated cerebral venous sinus pressure	Elevated CSF protein
Increased CSF production	Idiopathic intracranial hypertension

Table

Aim of the Study

This study aims at determining the etiological factors in patients with papilledema in a rural population.

Methodology

A 2-year prospective observational study between the period of June 2019 to June 2021 was carried out in a tertiary care hospital in the department of ophthalmology. All patients coming to the hospital and being diagnosed with papilledema either in out patient or in patient

department were included in the study. After institutional ethical clearance and with written informed consent of patient demographic details, duration, mode of onset, associated features, and the pattern of presentation was documented. History of patients was carefully taken to determine the presence or absence of risk factors. All subjects underwent a comprehensive ophthalmologic examination including best-corrected visual acuity, colour vision, Visual field test by Humphrey Field analyser, intraocular pressure measurement Goldmann's applanation tonometry. Fundus examination with 90D and indirect ophthalmoscope. Computed tomography brain imaging and magnetic resonance imaging will be done in selected cases suspected of neurologic dysfunction.

Inclusion criteria

- All patients presenting with disc edema due to raised intracranial pressure.

Exclusion criteria

- All patients presenting with disc edema due to other causes (i.e. non-cerebral causes).
- All patients having blurring of disc margins but due to pseudopapilloedema.

Results

Data was collected among 93 patients attending the hospital and diagnosed with papilledema based on fundus examination.

Among the total patients 54 were females and 39 were males (Figure 1).

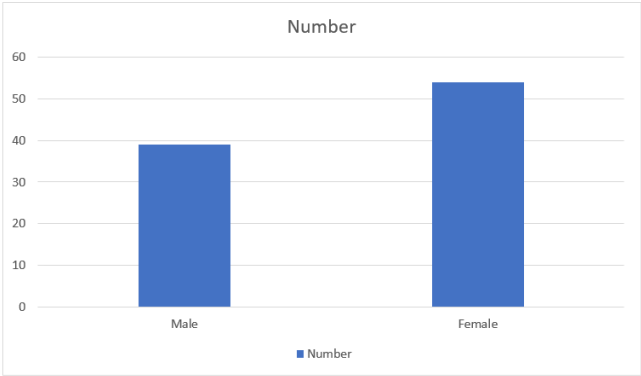

Figure 1: *Sex distribution.*

The most common cause (Table 1) for papilledema was found to be cerebral infection (40%) which included meningitis, encephalitis, either due to bacterial, viral or parasitic etiology, other causes included seizure disorder 17%, intracranial space occupying lesion 10%, cerebral venous thrombosis in six percent, nine percent of the patients who were diagnosed papilledema had complaints of associated headache and were suspected to have idiopathic intracranial hypertension(IIH) but did not review back with the required investigations to confirm the diagnosis (Figure 2).

Causes for papilledema	No. of patients	Percentage
Intracranial bleed	17	18%
Intracranial space occupying lesion	9	10%
Cerebral Venous thrombosis	5	6%
Suspected IIH	9	9%
Infection	37	40%
Seizure disorder	16	17%

Table 1: *Causes for papilledema with percentage.*

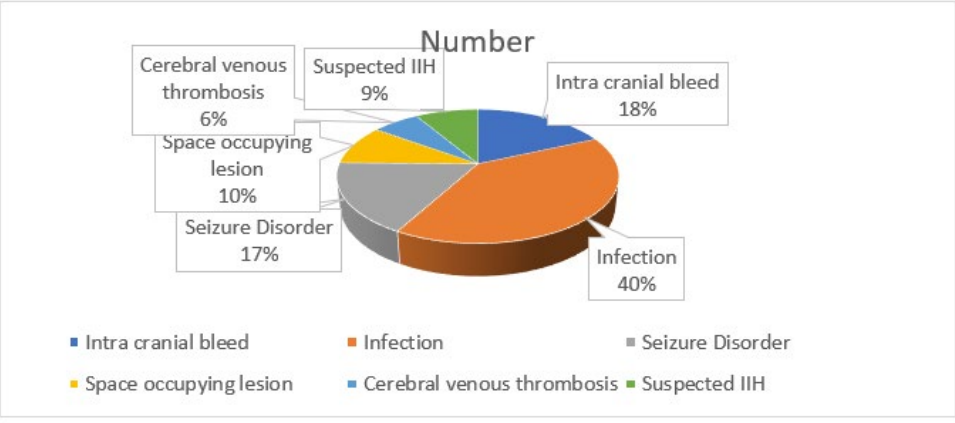

Figure 2: *Causes for papilledema.*

When classifying the papilledema the maximum cases were detected as Early papilledema (4%) followed by established (13%) and chronic (4%) papilledema (Figure 3).

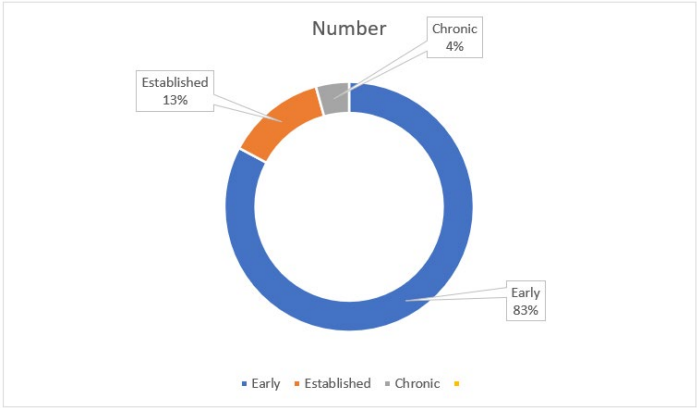

Figure 3: *Classification of papilledema.*

Discussion

Slowing of axoplasmic transport will lead to disc oedema, which might be resultant of ischemia, inflammation, certain toxins as well as compression. Papilledema is a condition which can be observed at optic nerve head due to raised intracranial pressure [10].

This study attempts to find out the most common cause and presentation of papilledema in rural Karnataka. Literature review showed very few similar studies and none in our geographical area. The study included cases of bilateral papilledema assessed based on history and examination and compared with published literature. In our study it was seen that females (57%) were affected greater than males, which maybe due to the geographic distribution and the incidence of disease at the time of data collection. This is similar to the finding by Vaidya., *et al.* [12] in which 64% were females. With Solanki., *et al*. [11] 42% of patients were male and 58% of were female and the studies by Ijeri and Jyoti [13] and Shah [14] who reported a slight male preponderance in 53.5% and 55.3%, respectively, Distelmaier F., *et al*. [15] and Gandhi U., *et al.* [16] also observed higher male preponderance which may be due to different sex ratio in their respective geographical areas, and also may be due to increased male population seeking better tertiary care for their ailments. In symptomology the most common symptom is headache which is the similar to a study by Julayanont., *et al*. [17] and Meena and Sharma [18]. Visual impairment is a serious complication that may not be recognized by the patients [18].

In our study cerebral infections (40%) due to various etiology was found to be the most common precursor of papilledema followed by intracranial bleed (18%), seizure disorders (17%) due to various factors and intracranial space occupying lesions (10%). In a study by Meena and Sharma [18] found that intracranial space-occupying lesions (24%) formed the most common aetiology followed by meningitis (12%). In a study conducted by Rani., *et al*. [19] main cause was idiopathic intracranial hypertension. Agrawal., *et al.* [19] found systemic causes are more prominent than local causes, in which ICSOL are most common among all causes.

Treatment at the earliest leads to resolution of papilloedema and complete visual recovery. Severe and long standing papilloedema may result in bilateral optic nerve dysfunction and secondary optic atrophy [19]. This leads to irreversible visual loss, constriction of visual fields and poor colour vision.

The prognosis for papilloedema is largely dependent on the cause [7].

Conclusion

Our study brings out a major concern in our geographical area, mandating early and urgent neurological workup of papilloedema to save life and vision. It is a need of hour to accommodate infectious disease specialist in a multidisciplinary approach along with ophthalmologist, neurophysician and neurosurgeons for carefully monitoring of visual acuity, visual fields and color vision.

Bibliography

1. Nguyen HS., *et al*. "Unusual causes of papilledema: Two illustrative cases". *Surgical Neurology International* 4 (2013): 60.
2. Keerthivarman R., *et al*. "Clinical profile and yield of neuroimaging in papilledema". *International Journal of Medical and Health Research* 3.10 (2017): 66-71.
3. Rani MRH., *et al*. "Multidisciplinary approach to papilledema: A prospective case series". *International Journal of Scientific Study* 4.4 (2016): 68-70.
4. Beri S., *et al*. "Idiopathic intracranial hypertension without papilledema". *Pediatric Neurology* 42.1 (2010): 56-58.

5. Nazir S., *et al*. "Sensitivity of papilledema as a sign of shunt failure in children". *Journal of the American Association for Pediatric Ophthalmology and Strabismus* 13.1 (2009): 63-66.

6. Sadun AA. "Papilloedema and raised intra cranial pressure". Chapter 189. In: Ophthalmology, Yanoff M, Duker JS, 3rd edition. Mosby Elsevier (2009): 960-963.

7. Gantela Sirisha., *et al*. "Evaluation of etiological factors and visual field defects in patients with papilloedema". *International Journal of Contemporary Medical Research* 3.9 (2016): 2729-2731.

8. Robertson DN and Dinsdale HB. "The Nervous system". Baltimore, MD, USA: Williams & Wilkins (1972).

9. Rigi M., *et al*. "Papilledema: epidemiology, etiology, and clinical management". *Eye and Brain* 7 (2015): 47-57.

10. Hayreh SS. "Pathogenesis of optic disc edema in raised intracranial pressure". *Progress in Retinal and Eye Research* 50 (2016): 108-144.

11. Solanki D., *et al*. "Optic disc edema/papilledema: a clinical profile". *Journal of Evolution of Medical and Dental Sciences* 5.16 (2016): 795-800.

12. Vaidya K., *et al*. "Etiologies of optic disc edema in tertiary eye care centre in Nepal". *Nepalese Journal of Ophthalmology* 10.20 (2018): 139-142.

13. Ijeri R and Jyoti RC. "Optic disc oedema: Presentation and causes at a tertiary centre in North Karnataka". *Delhi Journal of Ophthalmology* 29.1 (2018): 31-34.

14. Shah RK. "Clinical and etiological profile of patients with optic disc edema in tertiary care centre of Nepal". *Journal of Institute of Medicine Nepal* 41 (2019): 24-30.

15. Distelmaier F., *et al*. "Pseudotumor cerebri as an important differential diagnosis of papilledema in children". *Brain and Development* 28.3 (2006): 190-195.

16. Gandhi U., *et al*. "Optical coherence tomography angiography in acute unilateral nonarteritic anterior ischemic optic neuropathy: A comparison with the fellow eye and with eyes with papilledema". *Indian Journal of Ophthalmology* 66.8 (2018): 1144-1148.

17. Julayanont P., *et al*. "Idiopathic intracranial hypertension: ongoing clinical challenges and future prospects". *Journal of Pain Research* 9 (2016): 87-99.

18. Meena V and Sharma U. "To evaluate the profile of patients with disc edema/ papilledema and their presenting pattern". *International Journal of Medical Research and Review* 4.3 (2016): 301-308.

19. Agarwal A and Yadav P. "Papilledema (choked disc)". *Journal, Indian Academy of Clinical Medicine* 1.3 (2000).

20. Rani MRH., *et al*. "Multidisciplinary approach to papilledema: A prospective case series". *International Journal of Scientific Study* 4.4 (2016): 68-70.

21. Ehlers JP and Shah CP. "Papilloedema. The Wills Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease". 5th edition. Baltimore, Md: Lippincott Williams & Wilkins (2008): 252-254.

Volume 16 Issue 2 February 2025