

## Analysis of Papilloedema in ICU Patients

Sujata Priyambada<sup>1</sup>, Divya Mohindru<sup>2</sup>, Pragnya Paramita Mishra<sup>3</sup>

<sup>1</sup>Associate Professor, Department of Ophthalmology, Hi-Tech Medical College and Hospital, Rourkela, Odisha, India

<sup>2</sup>Assistant Professor, Department of Medicine, Hi-Tech Medical College and Hospital, Rourkela, Odisha, India

<sup>3</sup>Assistant Professor, Department of Pathology, Hi-Teach Medical College and Hospital, Rourkela, Odisha, India

---

Received: 09-01-2023 / Revised: 05-02-2023 / Accepted: 23-03-2023

Corresponding author: Dr. Sujata Priyambada

Conflict of interest: Nil

---

### Abstract

**Objective:** The term "papilloedema" refers to an almost usually bilateral, non-inflammatory edema of the optic nerve head caused by increased intracranial pressure. One of the true neuro-ophthalmic emergencies is papilloedema. Papilledema, or swelling of the optic nerve head, is one of the few physical examination-accessible objective symptoms that might validate a suspicion of elevated ICP.

**Method:** From January 2021 to December 2022, 100 patients with disc edema/papilledema who were sent from different departments to the Department of Ophthalmology, Hi-Tech Medical College and Hospital, Rourkela are the subjects of the current study, which is a non-randomized prospective case series. All patients had thorough history-taking, ocular examination, complete blood count, blood sugar, urea, creatinine, serum lipid profile, thyroid, and, in certain cases, a chest x-ray. A comparison was made between the follow-up examination and CSF analysis (including opening pressure).

**Results:** Of 100 patients in this study, the age group with the highest incidence of cases—31%—was between 20 and 30 years; 41% of those affected were men, and 59% were women. 31% of patients with papilledema had a local aetiology, including 21% cases of optic neuropathy, 5% cases of AION in the 50–60 year age range, and 3% cases of BRAO in the 40–50 year range. Among systemic causes, ICSOL accounted for 23% of cases, followed by meningitis (11%), malignant hypertension (9%), medication usage (7%), malaria (5%) and each of diabetes, pseudotumor cerebri, anaemia, encephalopathy, and head injury (1%).

**Conclusion:** In order to improve clinical decision-making regarding the diagnosis and treatment of papilledema, ophthalmic evaluation and subjective grading of papilledema by FUNDAL examination have the potential to be low cost objectively and quantitatively, and to assess progression and efficacy of treatment directed towards lowering ICP.

**Keywords:** Increased Intracranial Pressure (ICP), Intracranial Hypertension, and Papilledema.

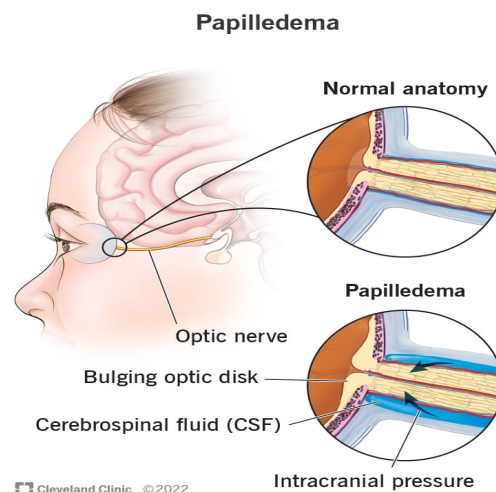
This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

---

### Introduction

According to definitions, "papilloedema" is a passive, non-inflammatory oedema of the optic nerve head brought on by increased intracranial pressure. It almost invariably

affects both sides of the brain and causes no visual impairment. [Fig. 1; 1]. Papilloedema is one of the genuine neuro-ophthalmic crises [2].



**Figure 1: Occurrence of Papilledema**

The following are papilloedema aetiologies: 1- Space-occupying lesions (neoplasm, abscess, inflammatory mass, haemorrhage, infarction, arteriovenous malformation) and focal or diffuse cerebral edoema are causes of increased intracranial pressure [3]. (Trauma, Toxic, Anoxia), reduction in CSF resorption, communicating hydrocephalus, meningeal processes (infectious meningitis, inflammatory (aseptic) meningitis), carcinomatous meningitis, elevated CSF protein, elevated venous pressure, craniosynostosis, thickening of the skull, blockage of cerebro spinal flow, increased CSF production, and idiopathic intracranial hypertension. The following criteria are used to diagnose pseudotumor cerebri: normal head imaging scan, increased intracranial tension as determined by lumbar puncture, [4] normal composition of cerebrospinal fluid, and absence of an intracranial mass lesion or cerebral venous thrombosis.

According to the length of the condition, there are five stages of papilloedema: early, established, chronic, and atrophic stages (Secondary Optic Atrophy). Differential diagnoses of papilloedema are; Pseudo papilloedema(Optic disc drusen, [6] Congenitally anomalous disc), Hypermetropic eyes, Papillitis, [7]

Hypertensive optic neuropathy, Central retinal vein occlusion, Ischemic optic neuropathy, Leber optic neuropathy, Diabetic alopathy (edema of optic nerve sheath in the absence of significant visual dysfunction), Thyroid related optic neuropathy, and Amiodarone toxicity.

Papilloedema is a crucial marker and alert indication of intracranial disease. Also, it can assist in determining the severity of systemic disorders like hypertension and preeclampsia, managing them, and averting subsequent vascular crises in other organs.

### Methods

**Study Design:** The current study is a non-randomized prospective case series being undertaken from January 2021 to December 2022 in patients with disc edema/papilledema who are referred from other departments to the Department of Ophthalmology, Hi-Tech Medical College and Hospital, Rourkela.

**Methodology:** All patients had thorough history-taking, ocular examination, complete blood count, blood sugar, urea, creatinine, serum lipid profile, thyroid, and, in some circumstances, a chest x-ray. and CSF evaluation (including opening pressure). The ocular examination includes measuring visual acuity using a Snellen chart, examining the anterior segment using

slit lamp biomicroscopy, applanation tonometry, taking stereoscopic fundus photos, and assessing the visual fields using automated perimetry with the Humphrey 30-2 programme. Frisen's system was used to grade the severity of the papilloedema. Follow-up was done after 1 year.

**Sample Size:** 100 patients that met the inclusion criteria were recruited in this study.

**Inclusion criteria:** Cases of disc edema and papilloedema

**Exclusion criteria:** All cases of grade IV hypertensive retinopathy, grade IV papilloedema, and papilloedema attributable to grade IV hypertensive retinopathy in pregnant women.

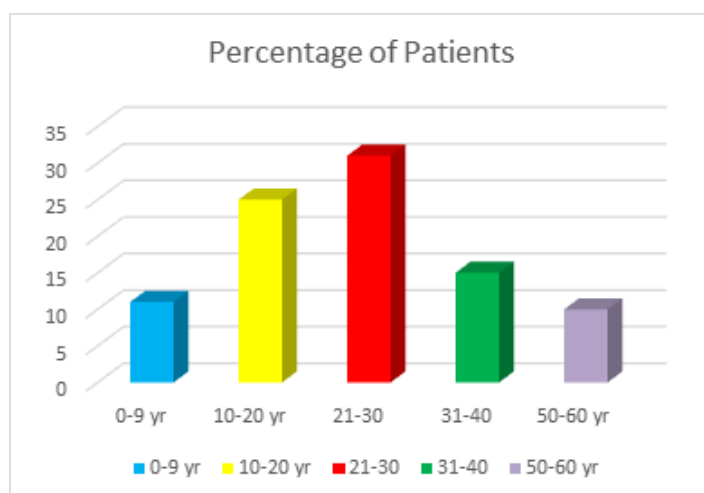
**Statistical analysis:** The analyses were mostly descriptive; for continuous

variables, averages, standard deviations, and ranges were provided; for categorical variables, counts and percentages were reported. Depending on the situation, either Pearson correlation coefficients or Spearman rank correlation coefficients are used to explain associations between continuous variables.

**Ethical Consideration:** The study was approved by the ethical committee of Hi-Tech Medical College and Hospital, Rourkela after written consent was obtained from the subjects.

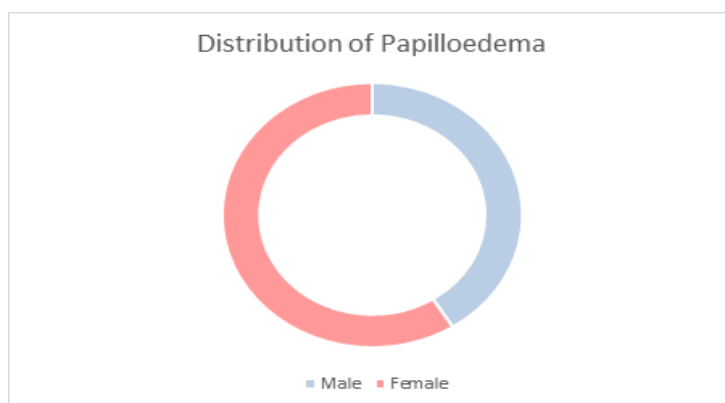
### Results

The age group that was afflicted the most frequently (31% of cases) was between 20 and 30 years, and the least frequently (8% of cases) was between 50 and 60 years [Figure 2].



**Figure 2: Distribution of papilloedema in different age groups**

In this study, 41% of men and 59% of women were affected [Figure 3].



**Figure 3: Distribution of Papilloedema on the basis of genders**

In this investigation, 29% of cases of unilateral disc edema and 71% of bilateral cases of papilloedema were noted.

Out of 100 patients in this study, 31% had local causes, of which 23% had optic

neuropathy. Following optic neuropathy were 5% cases of AION in patients between the ages of 51 and 60, and 3% cases of BRAO in patients between the ages of 40 and 50 [Table 1].

**Table 1: Local causes of Papilloedoma**

<b>Causes</b>	<b>No. of Patients</b>	<b>Percentage of Patients</b>
<b>Optic neuropathy</b>	30	23%
<b>AION</b>	12	5%
<b>BRAO</b>	9	3%
<b>TOTAL</b>	<b>51</b>	<b>31%</b>

Of 100 patients, Meningitis (11%), malignant hypertension (9%), drug use (7%), malaria (5%) and each of diabetes, pseudotumor cerebri, anemia, encephalopathy, and head injury (1%), were the other systemic reasons that contributed to 23% of cases.

Headache was the most frequent presenting symptom in 71% of patients, followed by DOV in 51%, nausea, and vomiting in 46%, and LR palsy with diplopia in 3%. [Table 2].

**Table 2: Symptoms Causes**

<b>Symptoms</b>	<b>Percentage</b>
<b>Headache</b>	71%
<b>DOV</b>	51%
<b>nausea and vomiting</b>	46%
<b>LR palsy with diplopia</b>	3%

## **Discussion**

Without mentioning its underlying aetiology, papilloedema simply refers to oedema of the optic disc. Because papilloedema is the most well-known and significant clinical indicator of elevated intracranial pressure, its recognition is crucial from a clinical standpoint. Since the development of modern diagnostic techniques, elevated intracranial pressure is now frequently identified and treated before papilloedema manifests [8]. In our study and a related study [9], headache was found to be the most prevalent symptom. IIH is a rare disorder marked by increased intracranial pressure with no radiological or laboratory evidence of intracranial pathology other than empty sella turcica, an optic nerve sheath with filled cerebrospinal fluid spaces, and smooth-walled nonflow-related venous sinus stenosis or colloid stenosis.

Obese women are often affected by this illness. With the prevalence of obesity rising, IIH is becoming more common. The most typical symptom is a constant headache. A significant complication that patients might not be aware of is visual impairment. The clinical signs, difficulties with the diagnosis, and available therapies for IIH in adults are discussed in this study. The effectiveness of various imaging modalities for the detection of IIH and papilloedema has been investigated. New investigations on the medicinal, surgical, and interventional therapy of this illness are also included in this review [10].

Two leading explanations for the papilloedema brought on by high ICP in IIH are optic nerve compression and optic nerve ischemia [11]. One of the symptoms used to diagnose IIH is papilloedema. Although papilloedema is typically symmetric or just minimally asymmetric, significant

asymmetry may be present in certain cases, which can be explained by differences in the size of the bone optic canals or the trabecular meshwork in the subarachnoid space surrounding the optic discs [12,13]. Similar to our work, unilateral papilledema is not frequently observed [14,15].

A fundoscopic examination is crucial to assessing this problem. The severity of papilledema can be graded according to Frisén's [14] criteria. It is crucial to distinguish between real papilledema and pseudoedema, which is the physiologic variant or benign alterations of the optic disc. Hemopexin, angiotensinogen, vitamin-D-binding protein, and transthyretin were four proteins that were downregulated in IHH. The first protein to be validated in the study was angiotensinogen, and it was discovered that down-regulating angiotensinogen may help to produce IHH by increasing CSF production. The investigation of other proteins may yield more information about the novel biomarkers for IHH diagnosis. These proteins might also be the focus of therapeutic intervention [16].

As anticipated and in line with earlier investigations [17,18], the majority of patients in this group complained of headaches. About half of the patients in both groups had depression; this finding is also consistent with earlier research [19]. Both IHHWP (48%) and IHHWOP (33%) patients reported having pulsatile tinnitus as a prevalent symptom; this was in line with earlier research [20, 21].

In this study, out of 100 patients, 31% had local causes, of which 21% had optic neuropathy, followed by 5% cases of AION in the 50–60 year age range and 1% cases of BRAO in the 40–50 year range. Agrawal et al. [18] found that systemic causes are more prevalent than local causes, with ICSOL being the most common among all causes in 23% of cases of systemic causes, followed by 13% cases of meningitis, 11% cases of malignant hypertension, 7% cases of drug history, 5% cases of malaria, and

1% cases of diabetes, pseudotumor cerebri, anaemia, encephalopathy, and head injury.

In this study, 70% cases of bilateral (papilloedema) and 30% cases of unilateral disc edoema were observed, which is consistent with many studies [2-4]. In contrast, Bidot S et al [21] studied asymmetric papilledema in idiopathic intracranial hypertension (IIH) and discovered that there were no significant differences in the incidence of the condition. 20 (3.6%; 95%CI: 2.3-5.6%) of the 559 adult patients with definite IHH at the time of the initial evaluation showed extremely asymmetric papilledema. They differed from patients with symmetric papilledema in that they were older (39 vs 30 years;  $p=0.001$ ), had a lower cerebrospinal opening pressure (35.5 versus 36 cm of water;  $p=0.03$ ), and were more likely to be asymptomatic (27% versus 3%;  $p=0.001$ ). [22] The side with the highest grade of papilledema had worse visual fields ( $p=0.02$ ). The bone optic canal was smaller on the side of the lowest-grade edoema in all 8 individuals (100%) in whom the imaging was sufficient for accurate measurements ( $p=0.008$ )

## Conclusion

The estimation of ONH shape and quantification of optic disc swelling from stereo fundus images using three-dimensional (3-D) image analysis methodology may increase repeatability and reliability of the assessment of papilledema and hence improve therapeutic outcomes because ophthalmoscopic evaluation and subjective grading of papilledema can exhibit significant variability among observers and require specialised clinical expertise. This study cannot generalize to the clinical profile and management of papilledema; evidence-based care for these individuals will be studied in clinical studies in the future.

## References

1. Sharma O, GMC B. To evaluate the profile of patients with disc

- edema/papilledema and their presenting pattern.
2. Wall M, GEORGE D. Idiopathic intracranial hypertension: a prospective study of 50 patients. *Brain*. 1991 Feb 1;114(1):155-80.
  3. Corbett JJ. Familial idiopathic intracranial hypertension. *Journal of Neuro-Ophthalmology*. 2008 Dec 1;28(4):337-47.
  4. O'Duffy D, James B, Elston J. Idiopathic intracranial hypertension presenting with gaze-evoked amaurosis. *Acta Ophthalmologica Scandinavica*. 1998 Feb 1;76(1):119-20.
  5. Corbett JJ, Savino PJ, Thompson HS, Kansu T, Schatz NJ, Orr LS, Hopson D. Visual loss in pseudotumor cerebri: follow-up of 57 patients from five to 41 years and a profile of 14 patients with permanent severe visual loss. *Archives of neurology*. 1982 Aug 1;39(8):461-74.
  6. Orcutt JC, Page NG, Sanders MD. Factors affecting visual loss in benign intracranial hypertension. *Ophthalmology*. 1984 Jan 1;91(11):1303-12.
  7. Radhakrishnan K, Thacker AK, Bohlaga NH, Maloo JC, Gerryo SE. Epidemiology of idiopathic intracranial hypertension: a prospective and case-control study. *Journal of the neurological sciences*. 1993 May 1;116(1):18-28
  8. Rowe FJ, Sarkies NJ. Assessment of visual function in idiopathic intracranial hypertension: a prospective study. *Eye*. 1998 Jan;12(1):111-8.
  9. Cameron AJ. Marked papilloedema in pulmonary emphysema. *The British journal of ophthalmology*. 1933 Mar; 17(3):167.
  10. Binder DK, Horton JC, Lawton MT, McDermott MW. Idiopathic intracranial hypertension. *Neurosurgery*. 2004 Mar 1;54(3):538-52.
  11. Menke MN, Feke GT, Trempe CL. OCT measurements in patients with optic disc edema. *Investigative ophthalmology & visual science*. 2005 Oct 1;46(10):3807-11.
  12. Trick GL, Vesti E, Tawansy K, Skarf B, Gartner J. Quantitative evaluation of papilledema in pseudotumor cerebri. *Investigative ophthalmology & visual science*. 1998 Sep 1;39(10):1964-71.
  13. Rebolleda G, Munoz-Negrete FJ. Follow-up of mild papilledema in idiopathic intracranial hypertension with optical coherence tomography. *Investigative ophthalmology & visual science*. 2009 Nov 1;50(11):5197-200.
  14. Frisén L. Swelling of the optic nerve head: a staging scheme. *Journal of Neurology, Neurosurgery & Psychiatry*. 1982 Jan 1;45(1):13-8.
  15. Scott CJ, Kardon RH, Lee AG, Frisén L, Wall M. Diagnosis and grading of papilledema in patients with raised intracranial pressure using optical coherence tomography vs clinical expert assessment using a clinical staging scale. *Archives of Ophthalmology*. 2010 Jun 1;128(6):705-11.
  16. Killer HE, Jaggi GP, Miller NR. Papilledema revisited: is its pathophysiology really understood? *Clinical & experimental ophthalmology*. 2009 Jul;37(5):444-7.
  17. Miller NR, Walsh FB, Hoyt WF, editors. *Walsh and Hoyt's clinical neuro-ophthalmology*. Lippincott Williams & Wilkins; 2005.
  18. Agarwal, A, Yadav P. Papilledema (choked disc) *Journal, Indian Academy of Clinical Medicine*. October-December 2000; 1(3).
  19. Julayanont P, Karukote A, Ruthirago D, Panikkath D, Panikkath R. Idiopathic intracranial hypertension: ongoing clinical challenges and future prospects. *Journal of Pain Research*. 2016 Feb 19:87-99.
  20. Passi N, Degnan AJ, Levy LM. MR imaging of papilledema and visual

- pathways: effects of increased intracranial pressure and pathophysiologic mechanisms. *American Journal of Neuroradiology*. 2013 May 1;34(5):919-24.
21. Bidot S, Bruce BB, Saindane AM, Newman NJ, Biousse V. Asymmetric papilledema in idiopathic intracranial hypertension. *Journal of Neuro-Ophthalmology*. 2015 Mar 1;35(1):31-6.
22. Yeganeh. Studying the effect of spironolactone treatment on right ventricular function in patients with pulmonary hypertension group 1. *Journal of Medical Research and Health Sciences*, 2023; 6(2): 2450–2456.