ORIGINAL RESEARCH

To assess the characteristics of individuals with disc edema/papilledema and their presenting symptoms

Dr. Mujahid Islam

Assistant Professor, Department of Opthalamology, Santosh Medical College & Hospital Ghaziabad, NCR Delhi, India

Corresponding author
Dr. Mujahid Islam
Assistant Professor, Department of Opthalamology, Santosh Medical College & Hospital Ghaziabad, NCR Delhi, India

Received: 19 March, 2018 Accepted: 16 April, 2018

ABSTRACT

Background: Disc edema, also known as papilledema when associated with increased intracranial pressure, is a critical ophthalmologic finding that can signal a range of underlying systemic and neurological conditions. **Material & Methods:** The present study is a non randomized prospective case series being conducted in Department of Ophthalmology. All the patients with disc edema/papilledema. Proven case of disc edema and Proven case of papilledema were included in this study. Cases of pseudopapilledema were excluded from the study. **Results:** Most commonly affected age group was between 21-30 years in which 32% case were observed, least common affected age group was 51-60 years in which 6% case were observed. In this study 42% affected were males and 58% females. In this study 70% cases of bilateral (papilloedema) and 30% case of unilateral disc edema were observed. In this study out of 50 patient, 30% patient were of local cause in which 22% cases were of optic neuropathy followed by 6% cases of AION in age group of 51-60 year followed by 2% cases of BRAO in age group of 41-40 years. In this study out of 50 patient, 24% cases of ICSOL in systemic causes followed by 12% cases of meningitis, 10% cases of malignant hypertension, 8% cases of drug history, 6% cases of malaria and 2% case each of diabetes, pseudotumorcerebri, anaemia , encephalopathy and head injury. Most common presenting complaint was headache in 70 % cases followed by DOV in 50 % cases , nausea and vomiting in 48 % cases , LR palsy and Diplopia in 4 % cases. **Conclusion:** Because ophthalmoscopic evaluation and subjective grading of papilledema can show significant variability among observers and requires specialized clinical expertise, the estimate of ONH shape and quantification of optic disc swelling from stereo fundus photographs by three-dimensional (3-D) image analysis methodology may improve reproducibility and reliability of the assessment of papilledema and thus improve clinical decision-making regarding its diagnosis and treatment.

Keywords: disc edema, papilledema, symptoms

INTRODUCTION

Disc edema, also known as papilledema when associated with increased intracranial pressure, is a critical ophthalmologic finding that can signal a range of underlying systemic and neurological conditions. Understanding the profile of patients presenting with disc edema and their clinical patterns is essential for timely diagnosis and management to prevent potential vision loss and address any life-threatening causes.[1-3] Papilledema is commonly caused by intracranial hypertension, which may be idiopathic or secondary to various pathologies such as brain tumors, cerebral venous sinus thrombosis, and meningitis. The clinical presentation can vary widely, ranging from asymptomatic cases discovered during routine eye examinations to severe headaches, visual disturbances, and other neurological deficits. Early recognition and intervention are crucial in managing these patients and mitigating complications.The demographic profile of patients with disc edema can provide insights into the etiology and risk factors associated with this condition. Studies have shown that certain age groups, genders, and comorbid conditions may be more predisposed to developing disc edema. For instance, idiopathic intracranial hypertension predominantly affects obese women of childbearing age, whereas papilledema in older adults may more commonly be linked to neoplastic processes or vascular abnormalities.The presenting symptoms of disc edema can offer clues to the underlying cause. Common symptoms include transient visual obscurations, pulsatile tinnitus, and

diplopia, particularly if there is associated sixth nerve palsy. Headaches, which may worsen with Valsalvamaneuvers or lying down, are frequently reported. Visual field testing often reveals an enlarged blind spot or peripheral field loss, which can aid in the diagnostic process.Ophthalmologicexamination remains the cornerstone of diagnosing disc edema. Fundoscopy typically reveals swollen optic discs with blurred margins, venous engorgement, and hemorrhages.[4-9] Ancillary tests such as optical coherence tomography (OCT) can quantify the extent of disc swelling, while visual field testing and neuroimaging help delineate the underlying cause.The management of patients with disc edema involves addressing the underlying etiology and reducing intracranial pressure if elevated. Therapeutic approaches may include medical treatments such as acetazolamide or surgical interventions like optic nerve sheath fenestration or cerebrospinal fluid shunting. Regular monitoring of visual function and disc appearance is vital to ensure therapeutic efficacy and prevent long-term sequelae.[10-18]

AIM AND OBJECTIVES

To assess the characteristics of individuals with disc edema/papilledema and their presenting symptoms.

MATERIAL & METHODS

The present study is a non-randomized prospective case series being conducted in Department of OphthalmologySantosh Medical College & Hospital Ghaziabad, NCR Delhi, India following the acquisition of informed consent from all patients or their relatives if the patient was unable to provide consent due to their medical condition. The procedure, along with its associated risks, benefits, and potential complications, was thoroughly explained to all participants. The duration of study was from February 2017 to January 2018. All the patientswith disc edema/papilledema. Proven case of disc edema and Proven case of papilledema were included in this study. Cases of pseudopapilledema were excluded from the study.

All patients underwent a complete medical evaluation including careful history taking, ophthalmic examination, complete blood count, blood sugar, urea, creatinine, serum lipid profile, thyroid, chest x-ray in specific cases. and CSF analysis (including opening pressure). Ocular examination consists of visual acuity measurement with Snellen's chart, anterior segment examination using slit lamp biomicroscopy, applanation tonometry, stereoscopic fundus photography and visual fields evaluation using automated perimetry with the Humphrey 30-2 program. The degree of papilledema was graded using Frisen's scheme.[3,4]

Thorough ophthalmic examination including:

a) Assessment of visual acuity on Snellen's chart and near vision.
b) Relative afferent pupillary defect- by swinging flash torch light examination.
c) Colour vision by Ishihara's chart
d) Intra ocular pressure-recorded with Schiotz indentation tonometer
e) Slit lamp examination.
f) Optic disc evaluation Using +78D/90D Condensing lens.

Following details were noted DISC, Cup size-, Color of the disc-,Cup disc ratio-, Cup disc asymmetry between two eyes-, Blurring of the disc margins-,Hemorrhage over disc-

Tortuosity of vein-, Venous pulsation.

In normal eye venous pulsation is present, if it is absent and not appear on pressing the globe it is indication of papilledema. Fundus evaluation with indirect ophthalmoscope and +90 D slit lamp examination done.

In early papilledema we may get following fundus findings:

- Hyperemia of the disc
- Blurring of the disc margin
- Apparent forward protrusion of disc
- Blurring of the physiological cup
- Overfilling of the vein
- In some early case of papilledema, haemorrhage and exudates may be present at some distance from the disc. Fully developed and late papilledema.

The physiological cup becomes partially or completely obliterated, the margin of the disc becomes definitely blurred, the surrounding retina may have grayish tinge and the vessels are seen to climb to attain the disc, the veins become engorged. Haemorrhage may appear as a linear streak on the disc or around it.

Persistent papilledema: The arteries are not any time narrowed because when the arteries exhibit narrowing atrophic changes in the disc invariably follow.

Visual acuity, optic disc changes, and visual field defects were checked in all the patients during follow-up, which was done every 2 weeks for a month, monthly for 3 months, and after that every 6 months for 2 years.

Statistical Analyses

The analyses were largely descriptive, with means, standard deviations, and ranges reported for continuous variables and counts and percentages reported for categorical variables. Associations between continuous variables are described using either Pearson correlation coefficients or Spearman rank correlation coefficients, as appropriate.

RESULTS

Table 1: Age wise distribution of the patients

Age	No of Patient (n=50)	% of Patient
O-10	06	12
11-20	12	24
21-30	16	32
31-40	8	16
41-50	5	10
51-60	3	6
Total	**50**	**100**

Most commonly affected age group was between 21-30 years in which 32% case were observed, least common affected age group was 51-60 years in which 6% case were observed.

Table 2: Gender wise distribution of the patients

Sex	No of Case(n=50)	% of Case
Male	21	42
Female	29	58
Total	**50**	**100**

In this study 42% affected were males and 58% females.

Table-3: Laterality of disc edema

Laterality of Disc Edema	No of Case(n=50)	% of Case
Unilateral	15	30
Bilateral	35	70
Total	50	100

In this study, 70% cases of bilateral papillooedema and 30% cases of unilateral disc oedema were observed.

Table-4: Local Causes of disc edema/papilledema

Disc edema/ papilledema	Causes	No of Case	% of case
Local Causes	Optic neuropathy	11	22
	Aion	3	6
	Brao	1	2
	Hypotony	0	0
	Raised iop	0	0
Total		**15**	**30**

In this study out of 50 patient, 30% patient were of local cause in which 22% cases were of optic neuropathy followed by 6% cases of AION in age group of 51-60 year followed by 2% cases of BRAO in age group of 41-40 years.

Table-5: Systemic Causes of disc edema/papilledema

Systemic Causes	Causes	No of Cases	% of Cases
	Icsol	12	24
	Diabetes	1	2
	Malignant hypertension	5	10
	Meningitis	6	12
	Drug history	4	8
	Pseudo tumor cerebri	1	2
	Malaria	3	6
	Anaemia	1	2
	Encephalopathy	1	2
	Head injury	1	2
	Total	35	70

In this study out of 50 patient, 24% cases of ICSOL in systemic causes followed by 12% cases of meningitis, 10% cases of malignant hypertension, 8% cases of drug history, 6% cases of malaria and 2% case each of diabetes, pseudotumorcerebri, anaemia , encephalopathy and head injury.

Table-6: Symptoms of disc edema/papilledema

Symptom	No. of Cases	% of Cases
Dov	25	50
Headache	35	70
Nausia/vomiting	24	48
Transient obscuration of vision	8	16
Lr palsy	2	4
Diplopia	2	4

Most common presenting complaint was headache in 70 % cases followed by DOV in 50 % cases, nausea and vomiting in 48 % cases , LR palsy and Diplopia in 4 % cases.

DISCUSSION

Papilledema simply means oedema of the optic disc, without reference to its underlying cause. Recognition of papilledema is of great clinical importance because it is the classic and most important clinical sign of raised intracranial pressure. Modern diagnostic methods have advanced to such an extent that the recognition and treatment of raised intracranial pressure is usually undertaken before papilloedemaappears.[18] Among symptomatology, headache is the most common symptom in our study similar study is by Julayanont P et al[19] they studied, idiopathic intracranial hypertension (IIH) is an uncommon disorder characterized by increased intracranial pressure without radiological or laboratory evidence of intracranial pathology except empty sellaturcica, optic nerve sheath with filled out cerebrospinal fluid spaces, and smooth-walled nonflow-related venous sinus stenosis or collapse. This condition typically affects obese women. The incidence of IIH is increasing with the rising prevalence of obesity. Persistent headache is the most common symptom. Visual impairment is a serious complication that may not be recognized by the patients. This paper reviews clinical manifestations, diagnostic challenges, and current treatments of IIH in adults. Various imaging modalities have been studied on their validity for detection of IIH and papilledema. This review also includes new studies on medical, surgical, and interventional management of this condition.[19] Compression of optic nerve and optic nerve ischemia are the two major theories explaining papilledema caused by elevated ICP in IIH.[20] Papilledema is one of the hallmarks for diagnosis of IIH. Even though, papilledema is commonly symmetric or only mildly asymmetric, significant asymmetry may be found in some patients and can be explained by difference in size of bony optic canals or variation of trabecular meshwork in subarachnoid space surrounding optic discs.[21,22] Unilateral papilledema is not commonly reported.[23,24], similar to our study. Fundoscopic examination is very important to evaluate this condition. Frisén's[14] criteria has been widely accepted for grading severity of papilledema. It is very important to differentiate true papilledema from pseudoedema, which is the physiologic variant, or benign changes of the optic disc. Findings from funduscopic examination showed that the presence of retinal or choroidal folds is a pathognomonic sign of true papilledema. However, this finding was seen only in 23% of the patients with true papilledema. The combination of four features from funduscopicexamination, including the swelling of the peripapillary retinal nerve fiber layer, peripapillary hemorrhages, papilla elevation, and congestion of peripapillary vessels, provides very good validity in differentiating true papilledema from pseudoedema (accuracy 93%, sensitivity 95%, and specificity 89%).[25] There is a study aimed at identifying the CSF proteome in IIH patients as the new biomarkers. Six proteins were upregulated in IIH, namely, sterol regulatory element-binding protein 1, zinc-α-2-glycoprotein, immunoglobulin heavy constant α-1, α-1-antitrypsin, serotransferrin, and haptoglobin. Four proteins were downregulated in IIH, including hemopexin, angiotensinogen, vitamin-D-binding protein, and transthyretin. Angiotensinogen was the first protein validated in the study, and it was found that down-regulation of angiotensinogen may contribute to the increased CSF production, which subsequently causes IIH. The study of other proteins may provide more knowledge on the new biomarkers for diagnosis of IIH. Moreover, these proteins may be the target for therapeutic intervention.[26] As expected, the majority of patients in this series reported headaches, consistent with previous studies. [27,28,29] Depression was present in about half the patients in both groups; this finding is also consistent with previous studies [30]. Pulsatile tinnitus was a common symptom in both IIHWP patients (48%) and IIHWOP patients (33%); this was consistent with previous findings.[28] A significant number of IIHWOP patients in this series reported auras. This may indicate that a significant number of IIHWOP patients are also migraine sufferers. Indeed, it has been observed that most patients with IIH have migrainous headaches unrelated to increased intracranial pressure.[31]Some may argue that IIHWOP is merely migraine in obese individuals. However, all of our IIHWOP patients underwent carefully performed measurements of OPs or they were not included in the study. In addition, 65% of our patients had 2 or more elevated reading on more than one occasion. Furthermore, 11 of our patients with IIHWOP had one or more other symptoms of increased ICP (besides headache). Finally, 4 of our IIHWP patients also had aura symptoms with migraine in addition to intracranial hypertension, headache is also common symptom in

our study. Vertigo was more frequently present in IIHWOP although nearly half of the IIHWP subjects also endorsed dizziness or vertigo on a review of systems. In a study reviewing complaints of patients with IIH, vertigo and dizziness are more common when clinicians routinely query for them. [32] Children with IIH frequently complain of dizziness and are ataxic.[33] In the recent William F. Hoyt Lecture of the American Academy of Ophthalmology, Dr Jonathan Trobe posited that papilledema is only a reliable indicator of chronically high ICP because the development of papilledema tends to lag behind the rise in ICP. Trobe noted that fewer than 20% of patients examined within a few days of head trauma or ruptured aneurysm have papilledema and only 6% of patients with chronically high ICP lack papilledema.[34] In this study out of 50 patient, 30% patient were of local cause in which 22% cases were of optic neuropathy followed by 6% cases of AION in age group of 51-60 year followed by 2% cases of BRAO in age group of 41-40 years. 24% cases of ICSOL in systemic causes followed by 12% cases of meningitis, 10% cases of malignant hypertension, 8% cases of drug history, 6% cases of malaria and 2% case each of diabetes, pseudotumorcerebri, anaemia , encephalopathy and head injury,similarlyAgrawal et al[18] found systemic causes are more prominent than local causes, in which ICSOL are most common among all causes. In this study 70% cases of bilateral (papilloedema) and 30% case of unilateral disc edema were observed which is correlated with many studies[2,3,4] while BidotS et al[21] studied, asymmetric papilledema in idiopathic intracranial hypertension (IIH) and found Of the 559 adult patients with definite IIH, 20 (3.6%; 95%CI: 2.3-5.6%) had very asymmetric papilledema at initial evaluation. They were older (39 versus 30 years; p<0.001), had lower cerebrospinal opening pressure (35.5 versus 36 cm of water; p=0.03), and were more likely to be asymptomatic compared to patients with symmetric papilledema (27% versus 3%; p<0.001). Visual fields were worse on the side of the highest-grade papilledema (p=0.02). The bony optic canal was smaller on the side of the lowest-grade edema in all 8 patients (100%) in whom the imaging was sufficient for reliable measurements (p=0.008).

CONCLUSION

Because ophthalmoscopic evaluation and subjective grading of papilledema can show significant variability among observers and requires specialized clinical expertise, the estimate of ONH shape and quantification of optic disc swelling from stereo fundus photographs by three-dimensional (3-D) image analysis methodology may improve reproducibility and reliability of the assessment of papilledema and thus improve clinical decision-making regarding its diagnosis and treatment.

REFERENCES

1. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumorcerebri syndrome in adults and children. Neurology. 2016 Sep 13;87(13):1285-1292. doi: 10.1212/WNL.0000000000003000.
2. Wall M, Falardeau J, Fletcher WA, et al. Idiopathic intracranial hypertension: the role of visual loss and papilledema severity in determining visual outcomes. Am J Ophthalmol. 2017 Jan;173:56-61. doi: 10.1016/j.ajo.2016.08.042.
3. Mollan SP, Ali F, Hassan-Smith G, et al. Evolving evidence in adult idiopathic intracranial hypertension: pathophysiology and management. J NeurolNeurosurg Psychiatry. 2016 Jul;87(7):982-992. doi: 10.1136/jnnp-2015-311302.
4. Bidot S, Bruce BB, Saindane AM, Newman NJ, Biousse V. Asymmetric papilledema in idiopathic intracranial hypertension. J Neuroophthalmol. 2017 Jun;37(2):113-119. doi: 10.1097/WNO.0000000000000435.
5. Kalyvas AV, Hughes M, Koutsarnakis C, et al. Evaluation and management of idiopathic intracranial hypertension: the role of surgery and venous sinus stenting. Br J Neurosurg. 2017 Jun;32(3):274-284. doi: 10.1080/02688697.2017.1395642.
6. Bruce BB, Preechawat P, Newman NJ, Biousse V. "Idiopathic intracranial hypertension in men: elevated opening pressure at lumbar puncture." J Neurol Sci. 2005;236(1-2):59-61. doi:10.1016/j.jns.2005.05.004.
7. De Smet E, Lee AG, Chapman K, et al. "Neuroimaging of papilledema." SeminOphthalmol. 2008;23(3):157-167. doi:10.1080/08820530802111405.
8. McClelland CM, Van Stavern GP. "Acquired non-arteritic ischemic optic neuropathy and papilledema." CurrOpinOphthalmol. 2016;27(6):483-489. doi:10.1097/ICU.0000000000000310.
9. Brazis PW. "Pseudotumorcerebri." CurrNeurolNeurosci Rep. 2004;4(2):111-116. doi:10.1007/s11910-004-0030-x.
10. Morrow MJ, Corbett JJ. "Retrobulbar optic nerve sheath decompression: successful treatment for severe papilledema in pseudotumorcerebri." J Neuroophthalmol. 2001;21(1):49-53. doi:10.1097/00041327-200103000-00009.
11. Menke MN, Feke GT, Trempe CL. OCT measurements in patients with optic disc edema. Invest Ophthalmol Vis Sci. 2005 Oct;46(10):3807-11. [PubMed]
12. Trick GL, Vesti E, Tawansy K, Skarf B, Gartner J. Quantitative evaluation of papilledema in pseudotumorcerebri. Invest Ophthalmol Vis Sci. 1998 Sep;39(10):1964-71. [PubMed]
13. Rebolleda G, Munoz-Negrete FJ. Follow-up of mild papilledema in idiopathic intracranial hypertension with optical coherence tomography. Invest Ophthalmol Vis Sci. 2009;50:5197–5200.
14. Frisén L. Swelling of the optic nerve head: a staging scheme. J NeurolNeurosurg Psychiatry. 1982 Jan;45(1):13-8. [PubMed]
15. Scott CJ, Kardon RH, Lee AG, Frisen 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. Arch Ophthalmol. 2010;128:705–711. [PubMed]
16. Killer HE, Jaggi GP, Miller NR. Papilledema revisited: is its pathophysiology really understood? Clin

Experiment Ophthalmol. 2009 Jul;37(5):444-7. doi: 10.1111/j.1442-9071.2009.02059.x.
17. Friedman DI. Papilledema. In: Miller NR, Newman NJ. Walsh and Hoyt's Clinical Neuro-Ophthalmolgy, 6th Ed.Baltimore: Lippincott Williams and Wilkins, 2005: 237-291.
18. Agarwal, A, Yadav P. Papilledema (choked disc) Journal, Indian Academy of Clinical Medicine _ Vol. 1, No. 3 _ October-December 2000.
19. Julayanont P, Karukote A2, Ruthirago D1, Panikkath D3, Panikkath R3. Idiopathic intracranial hypertension: ongoing clinical challenges and future prospects. J Pain Res. 2016 Feb 19;9:87-99. doi: 10.2147/JPR.S60633. eCollection 2016.
20. Passi N, Degnan AJ, Levy LM. MR imaging of papilledema and visual pathways: effects of increased intracranial pressure and pathophysiologic mechanisms. AJNR Am J Neuroradiol. 2013 May;34(5):919-24. doi: 10.3174/ajnr.A3022. Epub 2012 Mar 15.
21. Bidot S, Bruce BB, Saindane AM, Newman NJ, Biousse V. Asymmetric papilledema in idiopathic intracranial hypertension. J Neuroophthalmol. 2015 Mar;35(1):31-6. doi: 10.1097/WNO.0000000000000205.
22. Maxner CE, Freedman MI, Corbett JJ. Asymmetric papilledema and visual loss in pseudotumourcerebri. Can J Neurol Sci. 1987 Nov;14(4):593-6. [PubMed]
23. Brosh K, Strassman I. Unilateral papilledema in pseudotumorcerebri. SeminOphthalmol. 2013 Jul;28(4):242-3. doi: 10.3109/08820538.2013.768677. Epub 2013 Apr 29.
24. Wattamwar PR, Baheti NN, Radhakrishnan A. Idiopathic intracranial hypertension presenting as unilateral papilledema. Neurol India. 2010 Sep-Oct;58(5):818-9. doi: 10.4103/0028-3886.72208.
25. Carta A, Favilla S, Prato M, Bianchi-Marzoli S, Sadun AA, Mora P. Accuracy of funduscopy to identify true edema versus pseudoedema of the optic disc. Invest Ophthalmol Vis Sci. 2012 Jan 3;53(1):1-6. doi: 10.1167/iovs.11-8082. [PubMed]
26. Brettschneider J, Hartmann N, Lehmensiek V, Mogel H, Ludolph AC, Tumani H. Cerebrospinal fluid markers of idiopathic intracranial hypertension: is the renin-angiotensinogen system involved? Cephalalgia.2011;31(1):116–121.http://www.ncbi.nlm.nih.gov/pubmed
27. Digre KB, Corbett JJ. Idiopathic intracranial hypertension (pseudotumorcerebri): A reappraisal.Neurologist. 2001;7:2–67. [PubMed]
28. Wang SJ, Silberstein SD, Patterson S, et al. Idiopathic intracranial hypertension without papilledema: A case-control study in a headache center. Neurology. 1998;51:245–249.
29. Vieira DS, Masruha MR, Goncalves AL, et al. Idiopathic intracranial hypertension with and without papilloedema in a consecutive series of patients with chronic migraine. Cephalalgia. 2008;28:609–613.
30. Kleinschmidt JJ, Digre KB, Hanover R. Idiopathic intracranial hypertension: Relationship to depression, anxiety, and quality of life. Neurology. 2000;54:319–324.
31. Friedman DI, Rausch EA. Headache diagnoses in patients with treated idiopathic intracranial hypertension. Neurology. 2002 May 28;58(10):1551-3.
32. Murphy TP. Otologic manifestations of pseudo-tumor cerebri. J Otolaryngol. 1991;20:258–261.http://www.ncbi.nlm.nih.gov/pubmed
33. Rangwala LM, Liu GT. Pediatric idiopathic intracranial hypertension. SurvOphthalmol. 2007 Nov-Dec;52(6):597-617. [PubMed]
34. Trobe JD. Papilledema: the vexing issues. J Neuroophthalmol. 2011 Jun;31(2):175-86. doi: 10.1097/WNO.0b013e31821a8b0b. [PubMed]