

# To evaluate the profile of patients with disc edema/ papilledema and their presenting pattern

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## Abstract

**Background:** Raised intracranial pressure (ICP) is a potentially life threatening condition, which can also lead to visual loss and blindness. One of the only objective signs accessible during physical examination to confirm a suspicion of raised ICP is papilledema, or swelling of the optic nerve head (ONH). **Material & Methods:** The present study is a non randomized prospective case series being conducted in the 50 patients with disc edema/papilledema attending OPD and referred from other departments to DEPARTMENT OF OPHTHALMOLOGY, Gandhi Medical College and associated Hamidia Hospital, Bhopal from January 2013- December 2014. 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), follow up examination was compared with fundal photography. **Results:** In our study out of 50 patients, Most commonly affected age group was between 21-30 years in which 32% cases were observed, 42% affected were males and 58% females. Papilledema in 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. Among systemic causes, 24% cases of ICSOL 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, pseudotumor cerebri, anaemia, encephalopathy and head injury. **Conclusion:** ophthalmic evaluation and subjective grading of papilledema by FUNDAL examination has the potential of being a low cost objectively and quantitatively, and assess progression and efficacy of treatment directed toward lowering ICP and thus improve clinical decision-making regarding its diagnosis and treatment papilledema.

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

## Introduction

Papilledema the acquired bilateral, occasionally asymmetrical, ONH swelling is due to increased intracranial pressure [1]. Papilledema often produces brief episodes of monocular or binocular visual loss, called transient visual obscurations (TVOs) [2,3]. TVOs are often precipitated by postural changes and Valsalva-like maneuvers, with vision rapidly recovering back to baseline between episodes. TVOs are thought to occur due to transient ischemia of the swollen optic nerve head and, although dramatic, are not a sign of impending permanent visual loss. Rarely, IHH patients with papilledema can report gaze-evoked amaurosis, which is transient visual loss precipitated by movement of the eye into an eccentric gaze position [4].

Untreated papilledema can result in progressive irreversible visual loss and secondary optic atrophy in up to 31% of patients [2,5,6-8]. However, sustained visual loss is less commonly reported than TVOs at the time of presentation [2]. Raised intracranial pressure (ICP) is a potentially life threatening condition, which can also lead to visual loss and blindness [9]. One of the only objective signs accessible during physical examination to confirm a suspicion of raised ICP is papilledema, or swelling of the optic nerve head (ONH). A relatively common cause of papilledema is idiopathic intracranial hypertension, which affects approximately 1 in 100,000 people in the United States and is more prevalent in women with a high body-mass index [10]. Visual field tests may be normal in papilledema, and while medical tests such as lumbar

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puncture and neuroimaging can help in establishing elevated intracranial pressure, they can also give false-positive and false-negative results[11]. Different imaging modalities have been used to evaluate the topography of ONH in patients with papilledema [12,13]. From monocular color fundus photographs, papilledema can be quantified subjectively using the Frisén scale [14] by taking into account visual features of the optic disc margin and the appearance of discontinuity of blood vessels as they course over the ONH. Stereo disc photography may increase the accuracy of the assessment of papilledema. However, its interpretation is subjective and qualitative and thus depends on the observer's expertise and bias [15]. Early or subtle changes related to papilledema cannot be assessed precisely and objectively owing to the ordinal character of the Frisén scale, which remains one of its limitations, although it is in widespread clinical use. Thus, reliable techniques to assess and quantify papilledema are of high interest and may help the diagnosis and management of disorders causing raised ICP, especially when expertise in ophthalmologic evaluation of the ONH may not be readily available [11].

**Pathogenesis of Papilledema-** Because all CSF spaces communicate freely, the pressure and composition of the CSF is thought to be the same throughout the CNS. Consequently, researchers have based their theories and experiments on the assumption that the pressure in the SAS surrounding the ON is the same as that in the cerebral and spinal SAS. However, this assumption has not been proved, mainly due to the difficulty in obtaining accurate pressure measurements in the SAS of the ON in vivo. In the SAS of the ON, CSF flows from the chiasmatic cistern through the canicular portion and into the intraorbital portion of the ON, a space that becomes a cul de sac at the back of the globe [16]. Diagnosis of papilledema is made by direct or indirect ophthalmoscopic examination of the optic nerve. Hyperemia of the nerve head, blurring of the optic disc margin, peripapillary flame shaped hemorrhage, dilatation of the capillaries on the surface of nerve & absence of spontaneous venous pulsation are all clinical signs of papilledema. Nerve fiber layer haemorrhages are the most common finding & often indicate an acute process [17]. In the present study we will be evaluating the clinical profile of the patients of disc edema with special reference to Papilledema to highlight the role and significance of fundus evaluation at various stages and its clinical correlation.

## Material & Methods

The present study is a non randomized prospective case series being conducted in the patients with disc edema/papilledema attending OPD and referred from other departments to RIO, DEPARTMENT OF OPHTHALMOLOGY, Gandhi Medical College and associated Hamidia Hospital, Bhopal from January 2013- December 2014.

### Inclusion Criteria:

- Proven case of disc edema
- Proven case of papilledema

### Exclusion Criteria:

- Cases of pseudopapilledema

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 Frisén'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
3. Intra ocular pressure-recorded with Schiottz indentation tonometer
  4. Slit lamp examination.
  5. 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.

- a) Hyperemia of the disc
- b) Blurring of the disc margin
- c) Apparent forward protrusion of disc
- d) Blurring of the physiological cup
- e) Overfilling of the vein
- f) 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 distribution**

Age	No of Patient (n=50)	% of Patient
0-10	06	12
11-20	12	24
21-30	16	32
31-40	8	16
41-50	5	10
51-60	3	6
<b>Total</b>	<b>50</b>	<b>100</b>

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: Sex distribution**

Sex	No of Case(n=50)	% of Case
Male	21	42
Female	29	58
<b>Total</b>	<b>50</b>	<b>100</b>

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

**Table-3: Laterality**

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

In this study 70% cases of bilateral (papilloedema) and 30% case of unilateral disc edema were observed.

**Table-4: Local Causes**

	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
<b>Total</b>		<b>15</b>	<b>30</b>

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**

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	
	<b>Total</b>		<b>35</b>	<b>70</b>

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 C`ases	% 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**

Papilloedema simply means oedema of the opticdisc, without reference to its underlying cause. Recognition of papilloedema is of great clinicalimportance because

it is the classic and mostimportant clinical sign of raised intracranialpressure. Modern diagnostic methods haveadvanced to such an extent that the recognitionand treatment of raised intracranial pressure isusually

undertaken before papilloedema appears [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 sella turcica, 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 fundoscopic 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 fundoscopic examination, 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- $\alpha$ -2-glycoprotein, immunoglobulin heavy constant  $\alpha$ -1,  $\alpha$ -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 IIHWOP 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 readings 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 IIHWOP 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 IIHWOP 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, pseudotumor cerebri, anaemia, encephalopathy and head injury, similarly Agrawal 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 Bidot S 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. Our study does not speak to the clinical profile and treatment of papilledema in all

cases; evidence-based treatments of these patients await future randomized clinical trials.

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