

The prospective study of clinico-etiological analysis on patients of papilloedema

Chetan P. Saoji¹, Surendra P. Wadgaonkar^{2,*}, Preeti A. Patil³, Bhimrao S. Kamble⁴

¹Senior Resident, Jawaharlal Nehru Medical College, Maharashtra, ²Associate Professor, ³Assistant Professor, ⁴Professor, Dept. of Ophthalmology, JMF's ACPM Medical College, Dhule, Maharashtra

***Corresponding Author:**

Email: surendrawadgaonkar@gmail.com

Abstract

Background: As the etiologies of papilloedema are important from prognostic point of view, early recognition of its cause & prompt treatment is required for better visual outcome.

Objective: To study the etiology of the papilloedema in concerned patient and to study the optic disc changes by using ophthalmoscope and classify papilloedema.

Materials and Methods: This prospective study was based on patients presenting to our tertiary care hospital. 45 cases were selected amongst the patients attending our hospital during the study duration from July 2012 to July 2014. 45 patients were registered and informed consent was taken prior to study, those patients not giving consent for study were excluded. These participants were interrogated their demographic, investigative and management data were recorded and documented on pre-designed and pre-tested proforma, and analyzed by using SPSS version 17.

Results: Papilloedema occur in a wide range of age group but are more common in the age group of 31-40 years. Overall, males were affected more than females. All the patients had bilateral papilloedema. The common etiological factor for papilloedema was intracranial space occupying lesion. According to stages of papilloedema, early papilloedema was most commonly seen. 57.2% of the patients had normal visual acuity (unaided) in papilloedema and all the patients had normal color vision pattern.

Conclusion: A careful history, general and complete ophthalmological work-up with necessary investigations like CT, MRI/MRV are mandatory to diagnose patients with papilloedema.

Keywords: Etiology, Investigations, Papilloedema, Patients

Access this article online

Website:

www.innovativepublication.com

DOI:

10.5958/2395-1451.2016.00054.8

Introduction

Papilloedema is an ocular manifestation of various central nervous system disorders. These ocular manifestations of neuro-ophthalmic disorders are often features of more dangerous central nervous system or systemic pathology. The vision threatening nature of these disorders requires prompt recognition and diagnosis. A delay of even a few hours could result in a poor outcome. Therefore, the ophthalmologist must be familiar with the manifestations of these disease entities and initiate appropriate testing and treatment without delay.

Papilloedema is nothing but, optic disc edema, which results from increased intracranial pressure. The condition is usually bilateral and can occur over period of hours to weeks. Unilateral presentation is extremely rare. Its key features are blurring of the optic disc margins, anterior extension of the nerve head, venous congestion of arcuate and peripapillary vessels, and hyperemia of the optic nerve head.²

The conditions leading to papilloedema can be disorders associated with generalized brain swelling,

space occupying disorders like tumor or abscess, infections like meningitis and encephalitis, cerebral hemorrhage and trauma.³

As the etiologies of papilloedema are important from prognostic point of view, early recognition of its cause & prompt treatment is required for better visual outcome. Hence the present study was done with the objective to study the etiology of the papilloedema in concerned patient and to study the optic disc changes by using ophthalmoscope and classify papilloedema

Materials and Methods

This prospective study was based on patients presenting to tertiary care hospital. 45 cases were selected amongst the patients attending hospital during the study period from July 2012 to July 2014. Those 45 patients were registered and informed consent was taken from them for study, those patients not giving consent for study were excluded. Ethical clearance was obtained from Institute Ethical Committee. These participants were interrogated their demographic, investigative and management. All this information was collected in a pre-designed and pre-tested Performa. The collected data were analyzed as frequency and percentages.

Inclusion criteria

1. Patients having pupillary reflex normal
2. Both sexes.
3. Patients with normal and altered sensorium

Exclusion criteria

1. Patients with hazy media impairing the visualization of the fundus.
2. Patients in whom dilation of pupil contraindicated.

Demographic information like name, age (in years), sex (male/ female), occupation, address was obtained from each patient. History of symptoms like headache, defective vision for distant and near, double vision, transient visual-obscuration, deviation of eye ball to left or right were noted along with their onset, duration progression. History of DM, Hypertension and any systemic illness along with its duration were noted. History of any drugs use like amiodarone, tetracycline etc. were noted. In female obstetric history was also asked. Examination finding including general vital data like pulse, blood pressure, peripheral pulses are noted, higher function status also noted. Examination of cranial nerves and ENT structures was done.

The presenting distant uncorrected visual acuity (UCVA) of all patients/was recorded using Snellens chart or illiterate E chart of both eyes. If UCVA was less than 6/6 then pinhole improvement was recorded. Autorefractometry was done.

The patients were exposed to Ishihara color plates and asked to read. If the patients could read all plates perfectly, it was recorded that patient had normal color vision

External eye examination was performed for conditions like exophthalmos, bupthalmos etc. any deviation of the eyeball recorded. Extra ocular movements are noted down-both for ductions and versions in all cardinal positions. Details of the anterior segment from the lids to the lens are noted. Pupil size, reaction, any anisocoria noted.

Slit lamp examination was done according to physical status of patient to measure anterior chamber depth to see any opacity in all media like cornea is hazy or not, the lens was examined for evidence of cataract, pseudophakia, aphakia. Anterior vitreous examined for pigment and cells.

Intraocular pressure was measured using schiotz tonometer an average 3 readings were taken. Normal range was considered to have range of 10-21 mm of mercury, sometime digitally also intraocular pressure had been checked.

The pupils of the patients with normal IOP and normal A/C depth on slit lamp, were dilated with Tropicamide 1% eye drop then fundus was examined using examined by using direct ophthalmoscope and indirect ophthalmoscope or 90 D using slit lamp biomicroscope for optic disc changes as seen in papilloedema or any other abnormality noted.

After performing thorough general, neurological, anterior and posterior segments differential diagnosis was made. Then those concerned patients was subjected to further examination like blood tests, radiological imaging, if required fundus fluorescein angiography.

Follow up of concerned patients after 1 month of discharge for any sequel.

Data was collected prospectively and analyzed retrospectively using SPSS 17.0 software.

Results

The data for this prospective study was obtained from 45 cases of papilloedema. The following results were obtained.

Table 1: Age group wise distribution of papilloedema cases

Age group (years)	Number	Percentage
0-10	01	2.2
11-20	09	20
21-30	09	20
31-40	20	44.4
41-50	04	8.9
51-60	02	4.4
Total	45	100

Maximum i.e. 44.4% cases were of 31 to 40 years age group, followed in frequency by 21 to 30 and 11 to 20 years age group having 20% of patients each.

Table 2: Gender wise distribution of papilloedema cases

Gender	Number	Percentage
Male	30	66.7
Female	15	33.3
Total	45	100

Male predominance was noted in cases of papilloedema. Male to female ratio was 2:1.

Table 3: Etiological pattern of papilloedema cases in our hospital

Etiology	Number	Percentage
Space occupying lesion (intracranial)	24	53.3
Intracranial infection (meningitis, encephalitis)	16	35.6
Trauma	02	4.4
Systemic condition (hypertension)	01	2.2
Idiopathic intracranial hypertension	01	2.2
Post surgery	01	2.2
Total	45	100

Out of 45 cases, 24 cases (53.3%) of papilloedema were having space occupying lesion as etiological factor, intracranial infection such as meningitis and encephalitis was the cause in 16 cases (35.6%).

Table 4: Classification of papilloedema in stages (according duration)

Stage	Number	Percentage
Early (incipient) papilloedema	18	40
Established (fully developed) papilloedema	14	31.1
Chronic (long standing)	13	28.9
Atrophic papilloedema	00	00
Total	45	100

Early (incipient) papilloedema was noted in 18 cases (40%), established papilloedema was found in 14 cases (31.1%) and chronic papilloedema was noted in 13 cases.

Table 5: Visual acuity of papilloedema cases (n=45)

Visual acuity	Number	Percentage
6/6	26	57.8
Corrected 6/6	09	20
No improvement	10	22.2
Total	45	100

On examination (57.8%) 26 cases had visual acuity 6/6, (20%) 9 cases had corrected visual acuity of 6/6 and no improvement with glasses seen in 10 patients (approx 22.2%).

Table 6: Stages of papilloedema according to etiology

Etiology	Stage of papilloedema			Total
	Early	Established	Late	
Hypertension	00	00	1 (7.7%)	1 (2.2%)
Idiopathic	1 (5.6%)	00	00	1 (2.2%)
Intracranial infection	00	6 (42.9%)	10 (76.9%)	16 (35.6%)
Post surgery	1 (5.6%)	00	00	1 (2.2%)
Space occupying lesion	14 (77.8%)	8 (57.1%)	2 (15.4%)	24 (53.3%)
Trauma	2 (11.1%)	00	00	2 (4.4%)
Total	18 (100%)	14 (100%)	13 (100%)	45 (100%)

Chi-Square value = 26.893, p value = 0.003

Out of 18 cases of early stage of papilloedema 77.8% was due to SOL, 11.1% caused due to trauma and 5.6% was due to idiopathic intracranial hypertension. 5.6% was also caused by post-surgical etiology. Out of total 14 cases of established papilloedema 57.1% had SOL and 42.9% was due to intracranial infection. Out of 13 cases of chronic papilloedema, 76.9% caused due to intracranial infection, 15.4% had SOL as etiology and

7.7% caused due to systemic hypertension. There was statistically highly significant ($p < 0.01$) difference of the stages of papilloedema according to etiology

Discussion

The majority of patients belonged to 31-40 years of age group (44.4%). Male predominance was noted in this study. Male to female ratio was 2:1. In pseudo tumor cerebri cases females, were more commonly affected. Bilateral papilloedema was seen in all 45 cases i.e. 100%. But Ambika S et al⁴ noted a female preponderance. In their study, they found that 80% of the females were affected.

The causes of papilloedema in the present study were space occupying lesion (53.3%), trauma in 4.4%, systemic condition (2.2%), systemic hypertension (uncontrolled) in one patient, and 2.2% due to post-surgical causes. Kei Lijima et al⁵ reported that increased intracranial pressure was the most common cause in 59%, pseudopapillitis in 16%, uveitis in 8%, hypertensive retinopathy in 5%. Whiting AS et al⁶ listed intracranial tumors, idiopathic intracranial hypertension, and subarachnoid hemorrhage as important causes of papilloedema. Ha Son Nguyen et al⁷ during their routine clinical practice found that two cases of papilloedema had causes like anemia and leukemic infiltration of the central nervous system. They advised that the physician should be careful in their diagnosis as to the cause of papilloedema as it can avoid unnecessary surgical interventions. Davidson DL et al⁸ in their study of three cases of Guillaine Barre Syndrome observed that two cases had papilloedema. They found that this was due to defective re-absorption of CSF due to high protein concentration. Pye IF et al⁹ described a case of papilloedema, the cause of which was respiratory failure. But the patient presented with complaints like headache and visual impairment, which led them to go for complete neurological examination and investigations. But ultimately the cause was found to be respiratory failure.

The female patient was prescribed tab minocycline 100 mg twice daily dosage for 2 months for acne vulgaris treatment. Mochizuki et al¹⁰ has also reported the occurrence of pseudo tumor cerebri with bilateral optic disc swelling after use of minocycline for acne vulgaris. Minocycline is a cause or precipitating factor in pseudo tumor cerebri syndrome.¹¹

In this study, 40% of patients were found to have early (incipient) papilloedema. 31.1% of patients found to have established papilloedema. 28.9% of patients found to have chronic (long standing) papilloedema i.e. 13 cases. None of case had atrophic papilloedema

In this study the visual acuity was tested by using Snellens chart at 6 meter distance and also finger counting. 57.8% of papilloedema belonged to normal visual acuity 6/6. 20% (approx) of papilloedema belonged to 6/6 visual acuity with correction. 22.2% of papilloedema belonged to visual acuity was not

improving with pin hole and glasses, this was seen in 10 patients, out of this 10 patients 2 patients had counting finger 3 meter. Normally patients with early papilloedema are visually asymptomatic, the visual acuity not being affected.

In this study all patients of papilloedema showed normal color vision assessed by using Ishihara charts.

According to our study, early papilloedema was more commonly seen in 31-40 years age group that is 10 cases (55.6%) out of 18 cases. Established papilloedema more commonly seen in 21-30 age group that is 6 cases (42.9%) out of 14 cases. Chronic papilloedema was more commonly seen in 31-40 years age group that is 5 cases (38.5%) out of 13 cases.

According to our study, space occupying lesion is most common etiological factor that is 14 cases (77.8%) out of 18 cases for early stage of papilloedema. Space occupying lesion is most common etiological factor that is 8 cases (57.1%) out of 14 cases (total) for established stage of papilloedema. Intracranial infection is most common etiological factor that is 10 cases (76.9%) out of 13 cases for chronic stage of papilloedema.

Out of 45 cases of papilloedema 28 had turned for follow up while 37.8% i.e. 17 cases lost on follow up.

Conclusion

A careful history, general and complete ophthalmological work-up with necessary investigations like CT, MRI/MRV are mandatory to diagnose patients with papilloedema. Since papilloedema can be manifestations of life threatening condition, Patients may present to ophthalmologist with signs and symptoms of raised intracranial pressure, such as headache, nausea, vomiting, and abducens paresis, or may be referred by another physician or may be detected accidentally during a routine ophthalmic check-up.

References

1. Duke-Elder, Sir Stewart. Neuro Ophthalmology. In: Duke-Elder, Sir Stewart, editors. System of Ophthalmology. Henry Kimpton Publishers; 1976. p. 34-41.
2. Khurana AK. In: Khurana AK, editor. Comprehensive Ophthalmology, 5th ed. Tunbridge Wells, Kent: Anshan Publishers; 2014. p. 596.
3. Sihota R, Tandon R. Diseases of the Optic nerve. In: Sihota R, Tandon R, editors. Parson's Diseases of the Eye, 21st ed. New Delhi: Elsevier; 2011. p. 341-2.
4. Ambika S, Arjundas D, Noronha V et al. Clinical profile, evaluation, management and visual outcome of idiopathic intracranial hypertension in a neuro-ophthalmology clinic of a tertiary referral ophthalmic center in India. Ann Indian Acad Neurol 2010;13(1):37-41.
5. Iijima K, Shimizu K, Ichibe Y. A study of the causes of bilateral optic disc swelling in Japanese patients. Clin Ophthalmol 2014;8:1269-74.
6. Whiting AS, Johnson LN. Papilloedema: clinical clues and differential diagnosis. Am Fam Physician 1992;45(3):1125-34.
7. Ha Son Nguyen, Haider KM, Ackerman LL. Unusual causes of papilloedema: Two illustrative cases. Surg Neurol Int 2013;4:60.
8. Davidson DL, Jellinek EH. Hypertension and papilloedema in the Guillaine Barre Syndrome. J Neurol Neurosurg Psychiatry 1977;40(2):144-8.
9. Pye IF, Blandford RL. Papilloedema associated with respirator failure. Postgrad Med J 1977;53(625):704-9.
10. Mochizuki K, Tkahashi T, Kano M et al. Pseudotumorcerebri induced by minocycline therapy for acne vulgaris. Japanese J Ophthalmol 2002;46:668-72.
11. Monaco F, Agnetti V, Mutani R. Benign intracranial hypertension after minocycline therapy. Eur Neurol 1978;17:48-9.