A STUDY ON CLINICAL ANALYSIS OF PAPILLOEDEMA

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ABSTRACT

BACKGROUND

"Papilloedema is defined as a passive, non-inflammatory oedema of the optic nerve head due to raised intracranial pressure, which is almost always bilateral and without visual deficit". Papilloedema is one of the true neuro-ophthalmic emergencies. We wanted to study the etiological pattern, involvement of sixth cranial Nerve, visual acuity pattern, visual field and colour vision pattern in papilloedema.

METHODS

The cases studied were the patients with papilloedema who presented to the department of neuro-ophthalmology at the Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College, Chennai. This is a cross-sectional, descriptive, non-interventional, hospital-based study. The period of study was from September 2008 to October 2009.

RESULTS

The common etiological factor was space occupying lesion, 44.4% of the patients had sixth cranial nerve paresis; more than three forth of patients had normal visual acuity; less than half of the patients had visual field involvement; all patients had normal colour vision pattern in papilloedema.

CONCLUSIONS

Papilloedema can be a manifestation of life-threatening conditions. So, the ophthalmologist should be able to detect early papilloedema and refer them immediately.

HOW TO CITE THIS ARTICLE: Sivakalai R, Sivakami M. A study on clinical analysis of papilloedema. J. Evid. Based Med. Healthc. 2019; 6(19), 1443-1447. DOI: 10.18410/jebmh/2019/294

BACKGROUND

"Papilloedema is defined as a passive, non-inflammatory oedema of the optic nerve head due to raised intracranial pressure, which is almost always bilateral and without visual deficit."[1] Papilloedema is one of the true neuro-ophthalmic emergencies.[2]

Etiologies of papilloedema are the following; 1- Causes of raised intracranial pressure[3] are space occupying lesion (Neoplasm, Abscess, Inflammatory mass, Hemorrhage, Infarction, Arteriovenous malformation), Focal or diffuse cerebral edema(Trauma, Toxic, Anoxia), Reduction in the size of cranial vault (Craniosynostosis, Thickening of the skull), Blockage of cerebro spinal flow(Non communicating hydrocephalus), Reduction in CSF resorption (Communicating hydrocephalus, Meningeal processes (infectious meningitis, inflammatory (aseptic) meningitis), carcinomatous meningitis, elevated CSF protein, Elevated venous pressure), 2- increased CSF production, 3- idiopathic intracranial hypertension. Pseudo tumour cerebri is diagnosed by following criteria; Normal head imaging scan, increased intracranial tension as measured on lumbar puncture,[4] Normal cerebro spinal fluid composition, and No evidence of intracranial mass lesion or cerebral venous thrombosis.

Stages of Papilloedema[5] (according to duration of papilloedema) are early papilloedema, Established papilloedema, chronic papilloedema, and Atrophic stage (Secondary Optic Atrophy). Differential diagnosis 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 papillopathy (oedema of optic nerve head in the absence of significant visual dysfunction), Thyroid related optic neuropathy, and Amiodarone toxicity.

Treatment of papilloedema: According to the cause medical and surgical treatments are available. Medical treatment**:** Anti oedema measures like IV Mannitol, Tablet. Furosemide (40 mg), injection Decadron (8 mg), Tablet. Acetazolamide (250 mg). Surgical treatment: Ventriculo peritoneal shunt in case of intractable headache, optic nerve sheath decompression if vision is threatened are the indication for surgical treatment.

Papilloedema serves as an important indicator and warning signal of intracranial pathology. It can also help in finding the severity and management of systemic disease

Financial or Other, Competing Interest: None.
Submission 01-03-2019, Peer Review 10-03-2019,
Acceptance 16-03-2019, Published 13-05-2019.
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DOI: 10.18410/jebmh/2019/294

such as hypertension and preeclampsia and preventing further vascular crisis in other organs.

METHODS

Patients presenting to RIOGOH, Chennai with signs and symptoms of raised intracranial pressure, such as headache, nausea, vomiting, and double vision, or may be referred by another physician or may be detected accidently during a routine ophthalmic checkup were included in this study. All the age groups and both sexes were included. A complete ophthalmological workup was done.

Inclusion Criteria

All cases of papilloedema.

Exclusion Criteria

All cases of post papilloedema optic atrophy, papilloedema due to grade IV hypertensive retinopathy, and papilloedema due to grade IV hypertensive retinopathy in pregnancy patients were excluded.

Ocular Examination

Head posture, facial symmetry is noted. Any deviation of eyeball is recorded. Under slit lamp, details of the anterior segment from the lids to the lens are noted. Extraocular movements are noted -both ductions and versions in all cardinal positions. Pupil size, reaction, any anisocoria is noted. A dilated fundus examination and refraction is done. Diplopia charting – is done in a dark room. Patient is asked to wear goggles with red in front of the right eye and green before the left eye.

A torch light with a stenopaic slit is used. The patient is asked to look at this torch held 120 cm away and then the torch is moved to various positions. The patient is asked to describe the position of the images. The false image is usually the fainter and farther one. If Sixth Cranial Nerve palsy is suspected restriction of abduction is noted. If a superior oblique palsy is suspected, Parks Bielschowsky's 3 step head tilt test is done. A forced duction test is performed in doubtful cases to rule out restrictive aetiology.

Neurological Examination

Examination of other cranial nerves, Motor, sensory, and cerebellar symptoms and signs are noted.

Examination of Thyroid- Any neck swelling is noted.

Examination of spine & back is noted.

To look for congenital anomalies and neuro cutaneous markers are done.

Examination of ENT structure was done.

Investigations

Both eyes were evaluated for all cases.

Vision - Uncorrected (Using Snellen's charts at 6 meters) visual acuity and Best corrected (after Retinoscopy) visual acuity was done. Intraocular pressure was measured with applanation tonometer after topical anaesthesia. Detailed slit lamp examination was done. Fundus examination- Papilloedema, and any abnormalities were noted. Diplopia charting was done. Measurement of deviation- Primary & secondary deviation, cover uncover test in various gaze positions, both for near and distance was done. Hess charting, Gonioscopy, Visual field examination and Colour vision was done.

Haematology (in all cases)

Total count, Differential count, Erythrocyte sedimentation rat, Hemoglobin%, RBC count / platelet count, Blood sugar- Fasting, and Postprandial, Mantoux intradermal test, Blood VDRL / ELISA, CSF analysis (if any), Urine – albumin/ sugar, Motion- ova/ cyst was done.

Radiology

X ray skull lateral view / orbit, X ray chest – To rule out tuberculosis, X ray PNS – were done.

Orbital USG (In indicated cases)

Optical Coherence Tomography[8]- to rule out Disc oedema done in indicated cases. NEURO IMAGING: Emergency MRI with gadolinium and magnetic resonance venography (MRV) of the head was preferred. CT scan was done if MRI was not available.[9] Fundus Fluorescin Angiography was done in doubtful cases. Neurophysician /Neurosurgeon opinion was obtained.

RESULTS

A Retrospective study of 45 cases of papilloedema was examined. The following results were obtained.

Age Distribution

Age Group	Total Cases
0-10	2
11-20	9
21-30	20
31-40	9
41-50	4
51-60	1
	45

Table 1. Age Distribution in Papilloedema Patients

Regarding the age distribution, considering all the papilloedema cases in total, the maximum number of patients belonged to 21-30 years age group (44.4%) followed in frequency by 11-20 and 31-40 years age group both with 20% of patients, 41-50 years age group with 8.9% patients and 0-10 year's age group with 4.4% patients. The least number was seen in the age group of 51-60 years (2.2%).

Sex Distribution

Male	Female	Total
15	30	45

Table 2. Sex Distribution in Papilloedema Patients

In this study there was a gender difference, out of 45 patients 30 patients (66.7%) were in the female group. Out of 45 patients 15 patients (33.3%) were in the male group. Male to female ratio was 1: 2.

Laterality

Unilateral Papilloedema	Bilateral Papilloedema	Total
2	43	45

Table 3. Laterality in Papilloedema

Regarding laterality bilateral involvement was most common, 43 patients out of 45 patients had bilateral papilloedema (95.6%). Unilateral papilloedema was found in 2 cases out of 45 cases (4.4%) (Foster Kennedy syndrome-ipsilateral optic atrophy and contra lateral papilloedema).

Etiological Pattern in Papilloedema

Aetiology	No. of Cases
Space Occupying Lesion (SOL)	13
Meningitis (MTIS)	6
Thrombosis (TSIS)	4
Trauma (TMA)	2
Post-Surgical (PS)	2
IICT	7
Systemic Condition (SC)	1
CT Normal (CTN)	7
Not Turn Off (NTO)	3

Table 4. Etiological Pattern in Papilloedema Patients

In the study we observed 13 cases (28.9%) of papilloedema to have space occupying lesion. 6 cases (13.3%) of papilloedema to have meningitis. 4 cases (8.9%) of papilloedema to have thrombosis. 2 cases (4.4%) of papilloedema to have trauma. 2 cases (4.4%) of papilloedema to have post-surgical causes. 7 cases (15.5%) of papilloedema to have idiopathic intracranial hypertension. 1 case (2.2%) of papilloedema to have systemic condition-leukemia. 7 cases (15.5%) of papilloedema to have normal CT brain. 3 cases (6.7%) of papilloedema were not turn off.

Sixth Cranial Nerve Palsy in Papilloedema

Below chart shows sixth cranial nerve palsy and normal sixth cranial nerve in papilloedema patients.

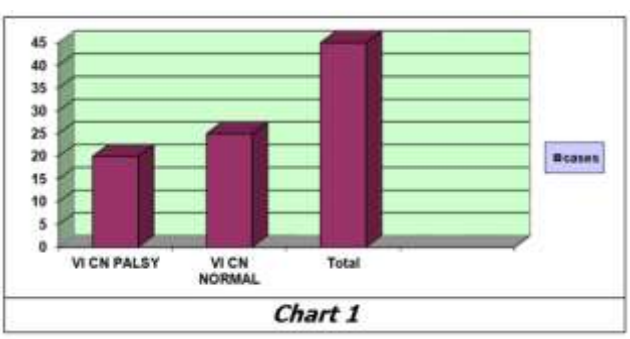

Chart 1

In this study 20 cases (44.4%) of papilloedema had sixth cranial nerve palsy. VI cranial nerve was normal in 25 cases (55.6%) of papilloedema.

Visual Acuity in Papilloedema

The following chart shows Visual acuity in papilloedema.

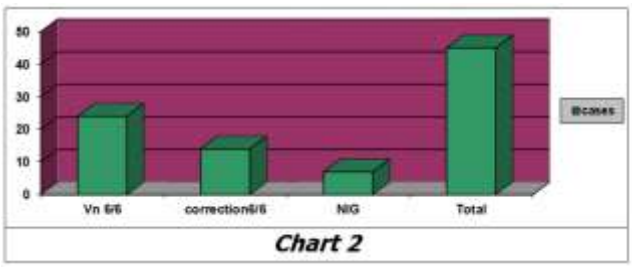

Chart 2

In this study 24 cases (53.3%) of papilloedema had normal visual acuity 6/6. 14 cases (31.1%) of papilloedema had 6/6 visual acuity with correction. 7 cases (15.6%) of papilloedema had not improving with glasses.

Visual Fields in Papilloedema

WNL	B/S Enlarged	Defective	Not co-op	Total
20	13	6	6	45

Table 5. Visual Fields Pattern in Papilloedema Patients

In this study the visual field pattern was assessed, 20 cases (44.4%) of papilloedema had normal visual field pattern. 13 cases (28.9%) of papilloedema had only blind spot enlargement. Compression, detachment and lateral displacement of the peripapillary retina[10] appear to be major reasons that blind spot increase in size in patient with papilloedema. 6 cases (13.3%) of papilloedema had defective visual field pattern. 6 cases (13.3%) of papilloedema had not cooperative for visual field testing.

All cases of papilloedema had normal colour vision assessed by using Ichihara charts.

DISCUSSION

In this study 45 cases of papilloedema were examined. The majority of patients belonged to 21-30 years of age group. But usually in case of idiopathic intracranial hypertension the most common age group affected was 30 years.

In this study there was a gender difference, out of 45 patients 30 patients (66.7%) were in the female group. Out of 45 patients 15 patients (33.3%) were in the male group. Male to female ratio was 1:2. Usually in idiopathic intracranial hypertension females are more commonly affected.

In this study bilateral papilloedema was most commonly (95.6%) seen, whereas unilateral papilloedema (Foster Kennedy syndrome) was least common (4.4%).

In this study 28.9% of papilloedema belonged to space occupying lesion. Comparing with the study by Gowers, 1904; Paton, 1909; 77.9% in 1,239 cases of papilloedema belonged to tumours of brain.

13.3% (6 patients) of papilloedema were due to meningitis. Comparing with the study by Blagojevic and Armbasic, 1956, they found 23% of cases belonged to tuberculous meningitis. In this study out of 6 patients, 5 patients had TB meningitis, 1 patient had HIV positive with disseminated TB with cryptococcal meningitis. 8.9% (4 patients) of papilloedema belonged to thrombosis. Out of 4 patients, 3 patients belonged to cerebral venous sinus thrombosis; one patient belonged to anti phospholipid antibody IgM positive. 4.4% (2 patients) of papilloedema belonged to trauma. Out of 2 patients one patient had post traumatic bilateral SDH (sub dural hematoma); one patient had post traumatic bilateral frontal lobe contusion. 4.4% (2 patients) of papilloedema belonged to post-surgical causes. Out of 2 patient's one patient developed papilloedema after modified radical neck dissection for papillary carcinoma thyroid, another patient developed papilloedema after decompression for Arnold-Chiari malformation.[11] 15.5% (7 patients) of papilloedema belonged to idiopathic intracranial hypertension. Out of 7 patients one patient had papilloedema due to Vitamin A over dosage. One patient had papilloedema with empty sella. CT and MRI/MRV was normal for remaining 5 patients. LP was done in one patient showed the opening pressure 32 cm H2O. 2.2% of papilloedema belonged to systemic condition. In this study one patient developed papilloedema due to acute myeloblastic leukemia. 15.5% of papilloedema belonged to CT brain normal study group. In this group MRI was not done. According to Rev Prat 2001 Dec 15;51 (20):2210-4 isolated bilateral papilloedema requires MRI, looking for tumour, hydrocephalus, cerebral venous thrombosis, if MRI is normal and does not show any tonsillar herniation, lumbar puncture has to be done with CSF pressure evaluation. Intracranial hypertension without any intracranial lesion (mass lesion, arteriovenous shunt, and venous thrombosis) is pseudotumour cerebri syndrome.[12]

In this study involvement of sixth cranial nerve palsy was assessed clinically by abduction restriction and diplopia charting. 44.4% of papilloedema had sixth cranial nerve palsy. VI cranial nerve was normal in 55.6% of papilloedema.

Damage may be unilateral or bilateral, the mechanism being compression or stretching of the abducens nerve at the base of the skull.

In this study the visual acuity was tested by using Snellen's chart at 6-meter distance. 53.3% of papilloedema belonged to normal visual acuity 6/6. 31.1% of papilloedema belonged to 6/6 visual acuity with correction. 15.6% of papilloedema belonged to visual acuity was not improving with glasses. Normally patients with early and even fully developed papilloedema are visually asymptomatic, the visual acuity not being affected. In this study 7 patients' visual acuity were not improving with glasses. Out of 7 patients 3 patients' visual acuity were not improving due to macular edema. 2 patients' visual acuity were not improving due to cataract. 2 patients' visual acuity were not improving due to pallor of disc on the affected side of SOL (Foster Kennedy syndrome), fellow eye vision was improving to 6/6.

In this study the visual field pattern was assessed by automated perimetry and Bjerrum screen. 44.4% of papilloedema showed normal visual field pattern. 28.9% of papilloedema showed blind spot enlargement. 13.3% of papilloedema showed defective visual field pattern. Out of 6 patients 2 patients showed typical of contracted visual field defect, 3 patients showed defective field and one patient had homonymous hemianopia with papilloedema due to empty sella. 13.3% of papilloedema were not cooperative for visual field testing. Normally in early stage of papilloedema – there is no field defect. In established stage, there is enlargement of blind spot. In chronic stage, there is associated with peripheral constriction of the visual field with appearance of nerve fiber layer bundle defects. Finally, there is total loss of visual field.

In this study all patients of papilloedema showed normal colour vision pattern.

CONCLUSIONS

Papilloedema occurs in a wide range of age, but is more common in the age group of 21-30 years. Overall, females were affected more than males. Bilateral papilloedema was most common than unilateral papilloedema like Foster Kennedy syndrome. The common etiological factor for papilloedema was space occupying lesion. 44.4% of the patients had sixth cranial nerve paresis in papilloedema. More than three forth of patients had normal visual acuity in papilloedema. Less than half of the patients had visual field involvement in papilloedema. All the patients had normal colour vision pattern in papilloedema. A careful history, general and complete ophthalmological workup with necessary investigations like CT, MRI/MRV is mandatory to diagnose patients with papilloedema. Since papilloedema can be a manifestation of life-threatening conditions, the ophthalmologist should be able to detect early papilloedema and refer them immediately.

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