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A clinical study of Etiological Factors and Visual Field Changes in Patients with Papilledema in a Teaching Hospital.

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Abstract
Introduction: *Papilledema is defined as edema of the optic disc or optic nerve head due to increased intracranial pressure. The aim of this study was to evaluate Etiological Factors and Visual Field Defects in Patients with papilledema.*
Material and methods: *Prospective study of 50 patients with papilledema was done between 2021 to 2022 in the Department of Ophthalmology NRI Medical College and General Hospital, Chinakakani,Guntur. The visual acuity is done by Snellen's chart. Color vision done using Ishihara pseudo isochromatic plates. Fundus examination using Heine direct ophthalmoscope, indirect ophthalmoscope & Slit lamp biomicroscopy using volk +90D lens was done. Neuroimaging was done in all the cases. Visual fields evaluation was done with Humphrey's automated perimeter.*
Results: *Among 50 patients 33 were females and 17 were males. The mean age in the present study was 33.32 years. The most common etiology was idiopathic intra cranial hypertension.The study showed that visual acuity, color vision were less affected in early stages of papilledema as compared to late stages. In the present study, mostcommon visual field defect was enlargement of blind spot.*
Conclusion:*Once papilledema isdiagnosed, neurological workup should be done to know the cause of papilledema. Serial visual field testing's are required to monitor the progression, regression and recurrenceof papilledema and to determine the course of treatment. Visual loss is reversible if treatment is started before the onset of disc changes of late stages of papilledema.*
Keywords: *Papilledema, Color vision , Vision ,Visual Field Defects, Enlargement of blind spot, Idiopathic Intracranial Hypertension (IIH).*

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I. Introduction

Papilledema is defined as edema of optic disc or optic nerve head due to raised intracranial pressure[1]. Optic nerve is covered up to the lamina cribrosa by the meningeal sheaths of brain. Optic nerve is freely continuous with the subarachnoid spaces (SAS) and subdural spaces (SDS) of the brain, any increase in intracranial pressure becomes equally noticeable around the nerve. When this occurs the SAS becomes so distended. As a result, edema develops at optic disc, this is purely hydrostatic, non-inflammatory phenomenon.

Experimentally this was proved in a successful animal model developed by Sohansingh hayreh[2,3], experimentally produced chronic intra cranial tension by placing a balloon in sub arachnoid space in rhesus monkeys and by increasing the size of this balloon slowly. It produced increased cerebrospinal fluid (CSF) pressure and optic disc edema. Hence a rise of cerebrospinal fluid pressure in the optic nerve sheath was the essential for the development of optic disc edema.He also evaluated effect of fenestration on sheath of the optic nerve on the side of optic disc edema. It showed resolution of disc edema on side of the optic nerve sheath fenestration, in spite of high intracranial CSF pressure.

Clinical features of papilledema include headache[4]which is worse in a recumbent position, early morning when the patient wakes up and may improve during the day. Transient obscurations of vision (Amaurosisfugax) precipitated by changes in posture, particularly from sitting to standing or from lying down to sitting or standing.In early stages vision and color vision are normal .Othersinclude nausea and vomiting, poor color perception, flashing sensation and double vision.

Fundus findings of papilledema[5] are Blurring of optic disc margins, filling of the physiologic cup, optic disc elevation, edema of the peripapillary Nerve Fiber Layer, Retinal folds / choroidal folds,Hyperemia of the

Optic Nerve Head, Venous congestion (venous dilatation and tortuosity),Peripapillary hemorrhages and retinal hemorrhages, Hard exudates over the disc or peripapillary area, Nerve Fiber Layer infarcts.

The most common cause of papilledema are caused by brain tumors. Inpatients having infratentorial tumorsPapilledema is more prevalent when compared with supratentorialtumors[4].Papilledema may be induced by Intracranial tumors in any position with the exception of medulla oblongata, the highest percentage being found in tumors of the midbrain, parieto-occipital region and cerebellum. Papilledema is rare and late in tumors of the anterior fossa.

Idiopathic Intracranial Hypertension (IIH) is also known as Pseudo tumor cerebri (PTC). It causes raised intra cranial pressure (ICP) in the absence of intracranial space occupying lesion. Lumbar puncture shows normal CSF and high opening pressure. IIH is usually idiopathic, obese young women are the most commonly affected. Drugs such as contraceptive pills, Tetracycline,minocycline, Nitroglycerin,Nalidixic acid, Vitamin A, Steroids and may produce IIH[6].

Enlargement of the blind spot is the most common visual field defect seen in the patients with papilledema[7]. Enlargement of the blind spot is due to hyperopia and detachment of peripapillary retina and choroidal folds. Constriction of the visual field seen in late stages which occurs in chronic papilledema as it progresses to optic atrophy.

Medical treatment consists of diuretics, like carbonic anhydrase(CA) inhibitors and, in cases of IIH, weight reduction and repeated lumbar punctures are needed. Optic nerve sheath decompression in IIH or lumboperitoneal shunt in hydrocephalus is done in cases where papilledema is not controlled by medical management[8].

II. Materials And Methods

The present study "A clinical study of papilledema'' was conducted between study period of March 2021 to June2022, in the department of Ophthalmology, NRI medical college and general hospital chinakakani, Guntur district. The study was approved by the Institutional Ethics Committee (IEC)

Study design: Hospital based prospective study.

Study location: This was a tertiary care teaching hospital based study done in the department of Ophthalmology, NRI medical college and general hospital chinakakani Guntur district.

Study Duration: March 2021 to June 2022

Study sample: 50 patients

Data of the following patients were included in the study:

Outpatients, inpatients of ophthalmology department & outpatients, inpatients referred from other departments, NRI medical college, and general hospital chinakakani, Gunturdistrict. A total of 50 cases of papilledema were included in the present study.

Inclusion criteria

All patients presenting with papilledema.

Patients who are willing to give informed consent.

Exclusion criteria

All patients presenting with disc edema due to other causes (i.e. non cerebral causes).

All patients having blurring of disc margins but due to pseudopapilledema.

Severe morbid patients who are not co-operating for visual field testing.

Methods of study

An informed consent will be obtained in every case.

A standard case protocol is maintained which includes a complete detailedhistory and thorough clinical examination. A detailed history is obtained from the patient regarding ocular and general complaints. A detailed examination of the anterior segment is done using slit lamp biomicroscopy. The visual acuity is recorded by Snellen's chart. Color vision is recorded using Ishihara pseudo isochromatic plates. Diplopia charting isdone using red green glasses for patients with double vision. Visual field recorded using Humphrey's automated perimeter. Fundus examination is done using Heine direct ophthalmoscope, indirect ophthalmoscope & Slit lamp biomicroscopy using volk +90D lens. A detailed systemic examination was done with special emphasis on CNS examination.

III. Results

In the present study, females were 33 and males were 17 cases.

Table no 1 shows most patients with vascular causes, inflammatory causes, IIH belonged to third decade. Most of the patients with intracranial tumors belonged to fifth decade.

Table no 1 Etiology with respect to different age groups

Age group (in years)	Intra cranial tumors	Vascular	Inflammatory	IIH	Total
Less than 20	0	2	2	4	8
21 – 30	1	3	3	12	19
31 – 40	2	1	0	3	6
41 – 50	7	1	0	5	13
51 – 60	2	0	0	0	2
> 60	2	0	0	0	2
Total	14	7	5	24	50

Vision is graded by Categories as per WH0, Eleventh Revision of international classification of Diseases (ICD) (May 2019).Table no 2 shows 34 cases (68%) has normal vision (6/ 6 to 6/ 12), 4 cases (8%) had Mild vision impairment, 9 cases (18%) has moderate vision impairment , 1 (2%) case has Severe vision impairment and 2 cases (4%) has Blindness. Out of 50 cases 14 cases had 6/6 vision.

Table no 2 Visual acuity

Category of visual impairment	Number of cases	Percentage (%)
Normal vision: 0 (6/ 6 to 6/ 12)	34	68
Mild vision impairment: 1 (Less than 6/ 12 to 6/ 18)	4	8
Moderate vision impairment: 2(Less than 6/ 18 to 6/60)	9	18
Severe vision impairment: 2 (Less than 6/60 to 3/60)	1	2
Blindness: 3(Less than 3/60 (FC at 3 m)	2	4

Table no 3 shows color vision is normal in 48 cases (96%), defective in 2 cases (4%) out of total 50 cases. Color vision is recorded in all 50 cases but in two cases color vision is unable to record in one eye and recorded in other eye, as patient had very low vision in one eye. In these two cases color vision is normal in other eye.

Table no 3 Color vision

Normal	Abnormal
48(96%)	2 (4%)

Among 50 cases, 48 cases has symmetrical papilledema. 2 cases had asymmetrical papilledema, in which 1 case had RE atrophic papilledema LE established papilledema, other case had RE established papilledema and LE chronic papilledema.

Table no 4 shows Staging of papilledema in symmetrical papilledema showed early stage of papilledema in 27 cases (56.25%), established stage of papilledema was seen in 20 cases (41.66%).The chronic papilledema was seen in 1 case (2.08%) and atrophic stage of papilledema was not seen any patient (0%).

Table no 4 Staging of papilledema in symmetrical papilledema

Staging of Papilledema in symmetrical papilledema	Number of cases	Percentage (%)
Early papilledema	27	56.25
Established papilledema	20	41.66
Chronic papilledema	1	2.08
Atrophic papilledema	0	0
Total	48	100

Among 50 cases, 20 cases (40%) had normal visual fields,30 cases had Visual field changes.Table no 5 shows the most common visual field change seen was enlargement of blind spot constituting 44%.

Table no 5 Visual field changes in papilledema

Visual field defect	Number of cases	Percentage (%)
Enlargement of blind spot	22	44
concentric constriction of field	3	6
Inferior quadrantanopia	3	6
Homonymous hemianopia	1	2
paracentral scotoma	1	2

IV. Discussion

Out of the 50 cases 16% occurred in the first 2 decades, 38% in the 3rd decade, 12% in 4th decade and 4% beyond 5th decade of life. These results are similar to the study by Subramaniamet. al. who reported 52% in the 3rd decade[9].In the present study 34 cases (68%) has normal vision(6/ 6 to 6/ 12).Fall of vision from 6/18 – 6/60 was noted in 18% and <6/60 in 6% . Out of 50 cases 14 cases had 6/6 vision.In study conducted by Rohit Agrawal[10] et al. in 201966.07% patients had vision between 6/12-6/6, 26.79% patients had vision between 6/36-6/18, 4 patients had vision ≤6/60. In the present study Color vision is defective in 2 cases (4%) out of total 50 cases. In study conducted by Ambika[11] et al on 50 patients of papilledema, color vision was done in 24 patients, out which 58.33% i.e. 14 patients had normal color vision and 41.67% i.e. 10 patients had abnormal color vision.In study conducted by Rohit Agrawal[10] et al. in 2019color vision was abnormal in 2 cases (3.57%) out of 56 cases.

In present study Staging of papilledema in symmetrical papilledema showed early stage of papilledema in 27 cases (56.25%), established stage of papilledema was seen in 20 cases (41.66%) The chronic papilledema was seen in 1 case (2.08%) and atrophic stage of papilledema was not seen any patient (0%).Study conducted by Rohit Agrawal[10] et al. in 2019Early papilledema was noted in 32 cases (57.14%), established papilledema was found in 22 cases (39.29%) only 1(1.79%) case had chronic papilledema and 1 (1.79%) had atrophic papilledema.

In the present study, 20 cases (40%) had normal visual fields,the most common visual field change seen was enlargement of blind spot constituting 44% of the visual field defects. Inferior quadrant defect was reported in 3 cases. Concentric constriction of the peripheral visual field was reported in 3 cases. Homonymous hemianopia was seen in only one case.Paracentral scotoma was seen in one case. In two cases visual fields are not recorded in one eye, as patient had very low vision in that eye. Sivakalai[12] et al in their study found that 20 cases (44.4%) of papilledema had normal visual field pattern. 13 cases (28.9%) of papilledema had only blind spot enlargement. 6 cases (13.3%) of papilledema had defective visual field pattern.Sirisha, et al[13] in their study found that Enlargement of blind spot 28 cases (56%) is the most common visual field defect in patients of papilledema.

Limitation

Very small sample size.

V. Conclusion

The ophthalmologist, neurologist and neurosurgeon have to work as a team in diagnosis and management of cases with papilledema.Once papilledema is diagnosed, neurological workup should be done to know the cause of papilledema. Serial visual field testing's are required to monitor the progression, regression and recurrence of papilledema and to determine the course of treatment. Visual loss is reversible if treatment is started before the onset of disc changes associated with chronic papilledema. By early diagnosis and treatment of papilledema we can prevent sight threatening complications and can save life of the patient.

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