Original Article

Assessment Of Ocular, Radiological And Neurological Changes In Papilledema

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Abstract

Background: Papilledema is the passive non-inflammatory swelling of the optic nerve head associated with increased intra cranial pressure. Papilledema is seen in many causes of increased intra cranial pressure. Papilledema is one of the earliest signs in cases of increased intra cranial pressure. It is important to determine the ability to identify papilledema at the earliest and classify it appropriately, which could account for early diagnosis of increased intracranial pressure in patients presenting in hospital with subtle signs and with non-invasive techniques available in a tertiary care setup.

Materials and Methods: A cross-sectional study was done including patients clinically diagnosed with papilledema. History and general examinations will be done which is followed by complete ocular examinations and Neurological examination. OCT will be done to evaluate the thickness of the peripapillary retinal nerve fibre layer. Patients diagnosed with papilledema are subjected to MRI brain with or without MRV. Further LP is advised only for therapeutic or diagnostic purposes as per the advice of a neurologist.

Results: The history suggested that more common presenting symptom of all the patients was a headache. Majority of patients presented with papilledema were diagnosed with idiopathic intracranial hypertension. Based on the study no strong correlation could be assigned on the involvement of the 6th cranial nerve. The association of visual acuity and the clinical classification of papilledema. OCT, RNFL thickness had a good association with the different clinical classifications of papilledema with a p-value less than 0.001. MRI optic nerve sheath diameter didn't show a significant association with the classification of papilledema. Posterior scleral angle and optic nerve tortuosity also didn't show much significant association with the classification of papilledema.

Conclusion: Early and accurate diagnosis of papilledema is always an important aspect in clinical practice, for early initiation of treatment of intracranial pressure, and also to monitor the progression of papilledema. Based on the study, the early assessment of papilledema and the high prevalence of causes based on the statistics causes could be assessed. Based on the study, OCT values showed a strong correlation with diagnosing papilledema. And a strong association was observed with the different classifications of papilledema.

Keywords: Papilledema, OCT RNFL, MRI, Idiopathic Intra Cranial Hypertension.

INTRODUCTION

Papilledema is the passive noninflammatory optic nerve head (ONH) elevation and swelling associated with raised intracranial pressure. Visual field loss can be associated with papilledema. In 1676 Briggs was the first to coin the term "papilla" for the optic disc. The term papilledema was a misnomer considering that the optic disc has a cup in the center. Edema of the optic disc due to elevated intracranial pressure (ICP) was called papilledema while disc edema due to other causes was called optic disc edema.[1]

The commonly presented cause of papilledema is found out to be due to brain tumors.[2] Most of the symptoms with which the patient presents in hospital are due to an increase in intracranial pressure.[3] The most common causes of visual field abnormalities in papilledema are due to blind spot enlargement.[4] The reason for enlargement of the blind spot being the accumulation of peripapillary subretinal fluid that causes the disruption of the intermediary tissue of Kuhunt.

Ocular coherence tomography is used for the diagnosis of optic disc edema, the peripapillary thickness of the NFL, and also the retinal nerve fiber layer thickness(NFLT) are taken into account for the early diagnosis of papilledema.[5] Treatment of papilledema is by identifying the cause of increased ICP and treating the cause and taking measures to reduce the intracranial pressure both medically and surgically.[6] Management of the general hemostasis of the patient, since general conditions should be maintained in his euvolemic and eusosmotic status with proper oxygen tension. Pyrexia should be avoided in these patients since that can cause an increase in intracranial pressure. CSF can be drained because of lowering of intracranial pressure using intraventricular catheters. Head of the bed elevation, diuretics, hyperventilation and decompressive craniectomy are the other available treatment modalities.[7] This study aims to the assessment of ocular, radiological, and neurological changes in papilledema.

Materials and Methods

A cross-sectional study was conducted at a Tertiary level healthcare facility in Chengalpattu District of the Indian state of Tamil Nadu for a period from November 2019 to September 2021. The study consisted of a population of patients diagnosed with papilledema due to increased intracranial pressure, presented in ophthalmology OPD Patients are diagnosed with papilledema clinically from fundus picture. In the diagnosed patients neurologic and radiological assessment was done.

All cases of papilledema were included in this study. Patients with papilledema in malignant hypertension or papilledema in pregnancy-induced hypertension or patients with bilateral optic nerve inflammation mimicking papilledema or optic neuropathy caused by toxins or pseudo papilledema caused by hypermetropia or optic disc anomalies mimicking disc edema were excluded from the study.

The patients were subjected to clinical examination. The age, gender, and the cause of increased intracranial pressure were taken into account. The history of presenting complaints was documented. The best-corrected vision of all the patients was assessed using the Snellen chart.

Fundus examination was done for all patients; papilledema was diagnosed and clinically classified into early, established, chronic stage and atrophic stages. Extraocular muscle function was assessed for all patients diagnosed with papilledema, and 6th, 3rd and 4th cranial nerve functions were assessed. Visual fields of the patients were tested using perimetry and visual field changes were documented for each stage of papilledema. OCT RNFL thickness in the peripapillary area measured in a Spectral-domain OCT (RS 3000 Lite). MRI and MRV were done for all the patients taken for study, MRI ONSD was measured, posterior scleral angle assessed, protrusion of papilla and optic nerve tortuosity is assessed.

Results and Analysis

Table 1: Basic Details descriptions

Basic Details	Mean ± SD || Median (IQR) || Min-Max || Frequency (%)
Age (Years)	34.97 ± 11.73 || 33.00 (26.00-45.00) || 15.00 - 67.00
Age ≤20 Years	5 (6.8%)
21-30 Years	25 (34.2%)
31-40 Years	19 (26.0%)
41-50 Years	16 (21.9%)
51-60 Years	7 (9.6%)
61-70 Years	1 (1.4%)
Gender Male	28 (38.4%)
Female	45 (61.6%)
Diagnosis Early Papilledema	24 (32.9%)
Established Papilledema	44 (60.3%)
Chronic Papilledema	5 (6.8%)
Cause Idiopathic Intracranial hypertension/ Pseudotumor Cerebri	26 (35.6%)
Cerebral Venous Thrombosis	11 (15.1%)
Space Occupying Lesion	11 (15.1%)
Cerebral Hemorrhage	5 (6.8%)
Thrombosis of Other Sinuses	5 (6.8%)
Trauma	4 (5.5%)
Tuberculoma	4 (5.5%)
Acute Lacunar Infarct	3 (4.1%)
Meningitis	3 (4.1%)
Retention Cyst in The Spenoidal Sinus	1 (1.4%)
6th cranial nerve involvement (Yes)	6 (8.2%)

Head Ache (Present)	73 (100.0%)

The study consisted of 73 patients. The participants with a mean age of 34.97 ± 11.73 years. 25 (34.2%) of the participants had ages: 21-30 years of age. majority of the participants were females with 45 (61.6%) and the remaining 38.4%being male. The majority of patients were diagnosed with established papilledema (60.3%) and the most common cause was observed due to idiopathic intracranial hypertension. 8.2% of the study population exhibited 6th cranial nerve involvement. All the patients presented in OPD with complaints of headache.

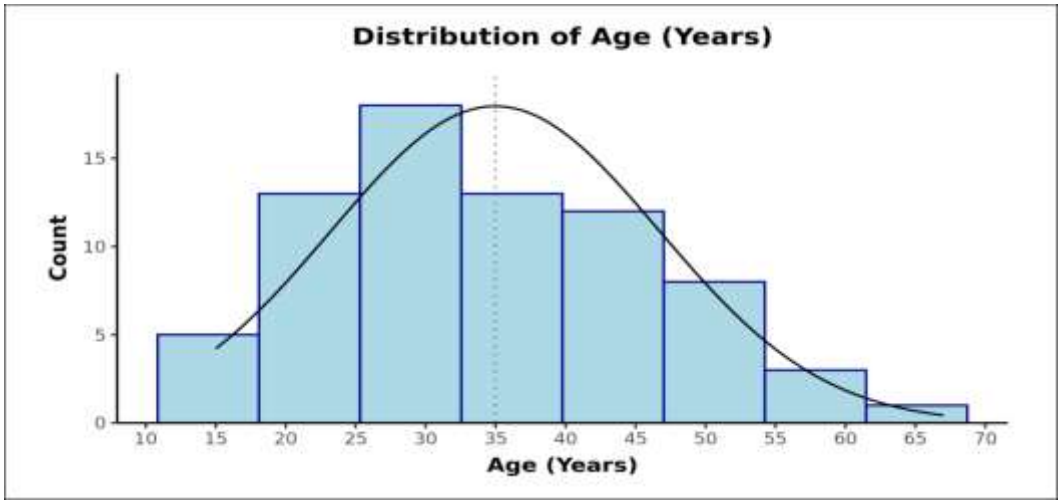

Figure 1: Distribution of study population based on age

The mean (SD) of Age (Years) was 34.97 (11.73). The median (IQR) in the study for Age (Years) was 33.00 (26-45). The Age (Years) ranged from 15 - 67. Age (Years) was not normally distributed based on Shapiro-Wilk Test: p = 0.036.

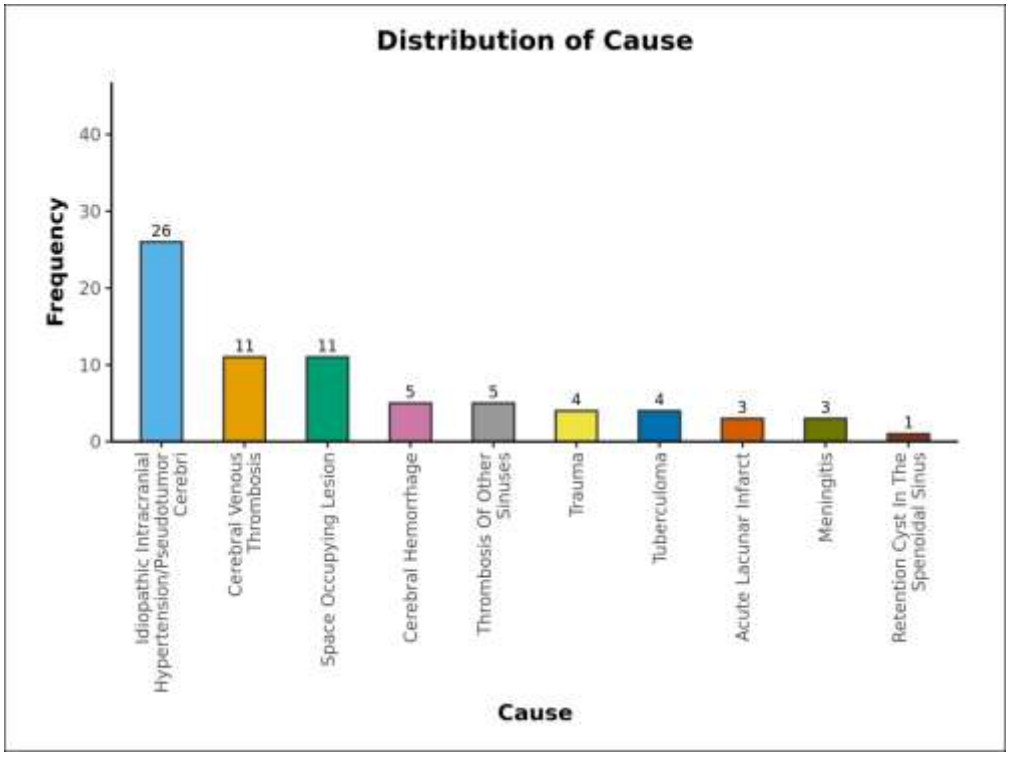

Figure 2: Cause wise distribution of study participants

Based on Fisher's exact test the association between 'Diagnosis' and 'Cause' count of less than 5 was seen in more than 20% of

the total number of cells. The various groups in terms of distribution no difference was observed of Cause ($\chi 2$ =16.004, p = 0.572)

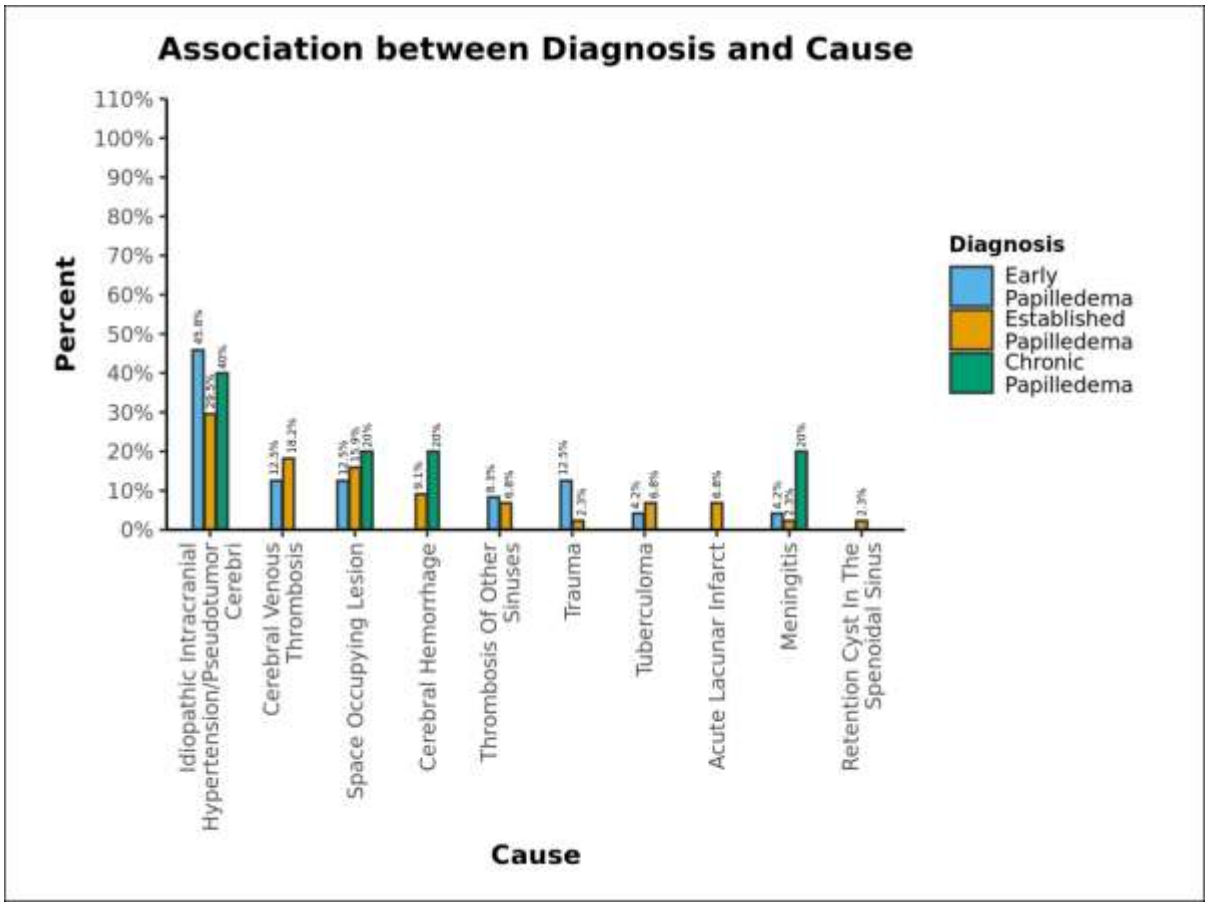

Figure 3: Analysis of the prevalence of papilledema in each cause

Association and correlation between diagnosis and the visual acuity were assessed with the Fisher's exact test was used in the right eye more than 20% of the total number of cells showed an expected count of less than 5. There was a significant difference was observed between the various groups in the distribution of Visual Acuity (Right) ($\chi 2$ = 22.878, p = 0.036). Strength of association between the two variables (Cramer's V) = 0.4 (Moderate Association). The low or minimal association was observed between the two variables (Bias Corrected Cramer's V) = 0.3 (Low Association)

To assess the association between 'Diagnosis' and 'Visual Acuity (Left)' Fisher's exact test was used, in 20% of the total number of cells a count of less than 5. There was a significant difference between the various groups in terms of distribution of Visual Acuity (Left) ($\chi 2$ = 18.965, p = 0.017). Strength of association between the two variables (Cramer's V) = 0.42 (Moderate Association) low to a minimal association between the two variables (Bias Corrected Cramer's V) = 0.29 (Low Association) 83.3% of the participants Diagnosed with Early Papilledema Visual Acuity Left eye of 6\6 was observed. 5.6% of the study population Visual Acuity (Left): 6\12. 11.1% had Visual Acuity (Left): 6\18. 72.7% of the participants Diagnosed with Established Papilledema the Visual Acuity in the Left eye was 6\6. 12.1% of the study population had Visual Acuity in Left eye of 6\12. 3.0% had Visual Acuity in Left eye: 6\18.

6.1% of the participants had Visual Acuity Left eye 6\24. 3.0% had Visual Acuity Left eye 6\36. 3.0% had Visual Acuity Left eye: 3\60. 50.0% of the participants Diagnosed with Chronic Papilledema had Visual Acuity (Left): 6\12. 25.0% had Visual Acuity in Left eye: 6\18]. 25.0% had Visual Acuity Left eye: 6\36.

There was no significant difference was observed between various groups in the distribution of 6th cranial nerve involvement ($\chi 2$ = 1.547, p = 0.622).

The association of 'Diagnosis' with 'Visual Field in Left eye' was assessed with Fisher's exact test as more than 20% of the total number of cells had an expected count of less than 5. No significant difference with a p-value more than 0.05, was observed between the various groups in the distribution of Visual Field (Left) ($\chi 2$ = 3.071, p = 0.291). Low Strength of association was observed between the two variables (Cramer's V) =0.21 (Low Association).

The association of 'Diagnosis' with 'Visual Field changes in Right eye was assessed using Fisher's exact test as more than 20% of the total number of cells had an expected count of less than 5. No significant difference was observed in various groups in terms of distribution of Visual Field (Right) ($\chi2 = 3.071$, $p = 0.291$).

Table 2: Comparison of the 3 subgroups of diagnosis with OCT (RNFL) (Right) (n = 73)

OCT (RNFL) (Right)	Diagnosis			Kruskal Wallis Test	
	Early Papilledema	Established Papilledema	Chronic Papilledema	$\chi2$	p value
Mean (SD)	172.25 (61.94)	241.75 (68.79)	153.20 (10.52)	19.608	<0.001
Median (IQR)	147 (117.25-211.5)	220 (208.75-274.75)	150 (150-152)		
Range	102 - 320	110 - 464	143 - 171		

A significant difference was observed between the clinical classification of papilledema in terms of OCT (RNFL) (Right) ($\chi2 = 19.608$, $p = <0.001$), the median OCT (RNFL) (Right) was seen maximum in the Established Papilledema group. Strength of Association (Kendall's Tau) = 0.21 (Small Effect Size). Same was observed for the left side.

No significant difference was observed between the groups in terms of MRI: ONS (mm) ($\chi2 = 2.927$, $p = 0.231$).

The association between papilledema and 'MRI was tested using Fisher's exact test to explore: Posterior Scleral Angle (Left)' as more than 20% of the total number of cells had an expected count of less than 5. There was a significant difference between the various groups of MRI: Posterior Scleral Angle (Left) ($\chi2 = 9.121$, $p = 0.026$). Low association between the two variables (Cramer's V) = 0.25 (Low Association) Low Association) 54.2% of the participants in the group Diagnosed with Early Papilledema had MRI: Posterior Scleral Angle (Left): No Flattening. 45.8% (Left): Minimal Flattening] 56.8% Diagnosed with Established Papilledema had MRI: Posterior Scleral Angle (Left): No Flattening. 31.8% had (Left): Minimal Flattening. 11.4% of the participants had [MRI: Posterior Scleral Angle (Left): Profound Flattening. 80.0% of the participants in the group Diagnosed Chronic Papilledema had MRI: Posterior Scleral Angle (Left): Minimal Flattening. 20.0% of the participants had MRI: Posterior Scleral Angle (Left): Profound Flattening]. Participants in the group Diagnosis: Established Papilledema had the largest proportion of MRI: Posterior Scleral Angle (Left): No Flattening. Participants in the group Diagnosis: Chronic Papilledema had the largest proportion of MRI: Posterior Scleral Angle (Left): Minimal Flattening. Participants in the group Diagnosis: Chronic Papilledema had the largest proportion of MRI: Posterior Scleral Angle (Left): Profound Flattening.

No significant difference between papilledema classification and Protrusion of the Optic Nerve ($\chi2 = 2.062$, $p = 0.635$). There was no significant difference between the various groups in terms of distribution of Tortuosity of Optic Nerve ($\chi2 = 4.254$, $p = 0.142$).

DISCUSSION

In our study with patients confirmed with papilledema most cases were observed in the age group 21 to 30 years of age group was 34.2%. In Meena et al study[8] 32% of the entire cases the affected age group was between 21-30. In a study done by Vinod dangi et al study[9] done with 100 patients with confirmed diagnoses most common age group with papilledema was in the 2nd to 4th decade.

Based on clinical classification in the study 35.6% of the cases where idiopathic intracranial hypertension was the most common diagnosis with papilledema, 15.1% of the patients were diagnosed with cerebral venous thrombosis. In a study done on 86 patients, Olivia M. Crum, et al[10] in 2019 cross sectional study done in an eye clinic, 87% of cases were diagnosed to be idiopathic intracranial hypertension which was similar to the results from our study. Deschamps R, the et al study[11] also shows a majority of cases in their study also showed idiopathic intracranial hypertension to be the most common cause of papilledema.

The Pattern of Vision Loss Associated with Papilledema

In our study, 27.4% of patients had the best-corrected vision of 6\6, 9.5% patients had vision less than 6\24 had other vision deferring conditions like cataractous changes, choroidal folds. 63.1% of the studied patients had reversible vision loss. John J. Chen; Matthew J. Thurtell; an et al retrospective study[12] was done with 660 patients with IIH reversible vision loss was observed in 46 % of cases (eyes) similar to our study most patients who had associated vision loss unless associated with optic neuropathy was seen to be reversible.

Based on our study there was no significant difference between papilledema classification and the involvement of the 6th cranial nerve ($\chi2 = 1.547$, $p = 0.622$). statistically. Low Association 4.2% of the participants in the group with Early Papilledema was present with 6th cranial nerve involvement 11.4% of the participants in the group with Established Papilledema] had 6th cranial

nerve involvement. In the study done by Pawan Sharma Ravindra Kumar Garg et al 60 patients with tuberculous meningitis was studied, having papilledema, in the study, it was found that 32.3% of patients had sixth cranial nerve involvement.[13] Mathis, Stéphane 1; Le Masson, Gwendal 1 Based on a case report First, radicular neuropathy may be a consequence of ICP ("false localizing sign").[14]

Most commonly documented MRI findings in papilledema are 1) ONS diameter increases, 2) posterior sclera flattening, 3) optic papilla protrusion into the globe, and 4) ON tortuosity.[15]

Based on the optic nerve sheath thickness in my study Group comparison was done using the values documented using the Kruskal Wallis test. papilledema is bilaterally observed, the optic nerve sheath thickness measurement in both eyes was separately measured taking the clinical classification as the basic variable, and was observed that both eyes the mean values of the ONS thickness values were relatable and within the range. The association of the ONS value measured and the clinical grading of papilledema was also assessed and was observed. The median values of the optic nerve sheath thickness values in my above study were in the range of 0.45 -0.58 in the early stages of papilledema, 0.52 – 0.67 in cases of established papilledema, and a range between 0.56 – 0.67 in chronic papilledema. With a p-value of 0.089, no significance was seen based on the different values in different groups of optic nerve sheath thickness in MRI. ($\chi2 = 4.841$, $p = 0.089$). The strength of association is based on Kendall's tau with a significant association. A.C. Rohr, et al study[16] showed ONS thickness widening was the most valid sign indicating increased intracranial pressure with a sensitivity of 94% and 92%. Based on the study done by Heidi Harbison Kimberly et al[17] state that with an increase in intracranial pressure more than 20 cm water, a characteristic curve was found to be Optic nerve sheath thickness of the cut-off value of 5.30 mm had a sensitivity of 100%.

Based on our study not much significant association was seen between the diagnosis and the MRI finding. Not every case of the diagnosed patient had significant flattening of the posterior sclera. No significant difference between papilledema stages and MRI: Posterior Scleral Angle (Right) ($\chi2 = 5.386$, $p = 0.177$). Low association between the variables (Cramer's V) = 0.19 (Low Association) Strength of association between the two variables (Bias Corrected Cramer's V) = 0.1 (Little/No Association). Based on the study done by N. Alperin et al done on 34 eye globes of patients with increased intracranial pressure where the quantitative measure of the globe flattening was done. globe flattening which was the measure of posterior scleral flattening was seen to be negatively correlated with intraocular pressure ($R = -0.75$, $P < .0001$). No significant globe flattening was observed associated with the increase in intraocular pressure.[18] In a study by Bidot et al., done on longstanding idiopathic intracranial hypertension it was observed that the sensitivity of MRI finding posterior globe flattening ranged from 43% to 85% in cases with increased intracranial pressure.[19] P.J. Maralani study done on 43 subjects diagnosed with idiopathic intracranial hypertension with MRI and MRV, flattening of the posterior globes was seen with a specificity 100%, $p < 0.0001$.[20]

In my study, no strong association could be observed with the protrusion of the ONH and the diagnosis of papilledema. No significant difference was seen between groups of papilledema in the distribution of Protrusion of The Optic Nerve (Right) ($\chi2 = 2.062$, $p = 0.635$). Low association between the two variables (Cramer's V) = 0.17 (Low Association). Strength of association between the two variables (Bias Corrected Cramer's V) = 0.02 (Little/No Association). Protrusion of the ONH was visualized in 6.8% of established papilledema cases. There was no strong association of protrusion of optic nerve head associated with increased intracranial pressure. Yu-Cherng C. Chang et al study was done to assess the correlation of MRI and OCT optic nerve protrusion and grades of papilledema it was observed that nerve protrusion length was significantly correlated with the grades of papilledema, P less than 0.0001.[21] Divya R. Hingwala et al studied imaging signs on 21 patients with increased intracranial pressure statistically significant association was observed in conditions where there is an increase in intracranial pressure associated with a tumor.[22]

Papilledema and Optic Nerve Tortuosity in MRI

In the study above all diagnosed patients with papilledema, the tortuosity of the optic nerve was assessed. It was observed that in many cases of established papilledema there was profound tortuosity of the optic nerve. But statistically, no significant difference between the papilledema and Tortousity of Optic Nerve observed (Right) ($\chi2 = 3.467$, $p = 0.147$). Low association between the variables (Cramer's V) = 0.22 (Low Association) Strength of association between the two variables (Bias Corrected Cramer's V) = 0.14 (Low Association). In G.T. Armstrong et al study in 28 cases with optic nerve glioma MRI was assessed with optic nerve tortuosity. the study showed an inter agreement of optic nerve tortuosity between three different people in 75 % of cases. A sensitivity of 89% and observed for the diagnosis of tortuosity.[23] R. Agid et al a retrospective study neuroimaging was done in 30 patients with IIH, all changes in papilledema were assessed with 56 controls. optic nerve tortuosity was significantly associated with IIH ($P<0.05$).[24] In Michael CBrodskyMD et al in a case-control study on 20 cases of pseudotumor cerebri and 20 control cases, a retrospective study was done it was observed 40% of cases had vertical tortuosity of the optic nerve.[25]

CONCLUSION

In my present study, it is observed that along with the clinical diagnosis and classification of the papilledema cases utilization of OCT retinal nerve fiber layer thickness along with the clinical findings would account for a more accurate diagnosis. In view of the chances of interobserver and intraobserver variations which can happen if only clinical findings have relied on the diagnosis. The chances of faulty classification or inter-observer variations in the classification of papilledema are prone to less efficient follow-up of papilledema and intracranial pressure status. A strong statistical association was observed between the clinical classification of papilledema and OCT retinal nerve fiber layer thickness. The study also calls on for acknowledgment of the common etiologies of papilledema. The problem at hand calls for further prospective, longitudinal studies to establish a causal relationship and to have a better understanding of the etiology for developing adequate measures to improve diagnosing and management of papilledema.

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