## Subjective Assessment of Visual Functions InPatients Presenting With Papilledema

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#### Abstract:

**Background:** Papilledema refers to bilateral, passive, non-inflammatory oedema of the opticnerve head due to raised intracranial pressure  $(ICP)^{l}$ . It can occur in any age and sex.Diseases causing Papilledema to include brain tumours, cerebral venous sinus thrombosis, Idiopathic intracranial hypertension (IIH), trauma, meningitis and hydrocephalus. Several theories were postulated to explain the underlying mechanism of papilledema. Of them, mechanical compression theory and an ischemic insult to axons are the most important. The incidence of different causes of papilledema in various studies shows highest due to vascular insult followed by inflammation, idiopathic intracranial hypertension(IIH) and intracranial space occupying lesions. In all cases presenting with papilledema, it is important to assess visual functions and establish an etiological diagnosis for early treatment to prevent blindness. So, the present study is carried out to assess the visual function in patients presenting with papilledema.

AIM: Subjective assessment of visual function in patients with papilledema.

**MATERIALS AND METHODS:** This is hospital-based prospective, observational study. A total of 30 patients both males and females aged between 10-60 yrs were included in this study.

After written and informed consent taken from the patients, a detailed history was taken regarding chief complaints, duration of illness. Clinical examination of the patient included a detailed general physical examination and systemic examination, followed by an ophthalmological examination which includes best corrected visual acquity, colour vision, anterior segment evaluation using slit lamp, posterior segment evaluation using direct ophthalmoscope, 90 D lens and fundus photography, visual field examination using Humphreys visualfield analyser. Results obtained were subjected to statistical analysis.

**RESULTS:** In this study 60 eyes of 30 patients presenting in various stages of papilledema were evaluated for visual functions. Statistically significant difference present among best corrected visual acquity, colour vision, and visual fields with respect to age, duration, stage of papilledema.

**CONCLUSION:** Based on visual function tests assessed in patients with papilledema, one can establish etiological diagnosis, prognosis, early treatment directed to etiology which further prevents blindness. **KEYWORDS:** Visual acquity, colour vision, visual fields, papilledema.

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#### I. Introduction

Papilledema refers to bilateral, passive, non-inflammatory oedema of the opticnerve head due to raised intracranial pressure (ICP)<sup>1</sup>. It can occur in any age and sex.Diseases causing Papilledema to include brain tumors, cerebral venous sinus thrombosis, Idiopathic intracranial hypertension (IIH), trauma, meningitis and hydrocephalus<sup>2</sup>.The optic nerve is surrounded by all three meningeal layers and is continuous with the subarachnoid space of the brain. Intracranial CSF is in this way contiguous with the CSF surrounding the optic nerve allowing elevated ICP to be transmitted directly to the optic nerve. It is assumed that this disrupts the normal pressure gradientacross the intraocular and orbital optic nerve causing retrograde axoplasmic flow across the optic disc, which results in disc oedema and optic neuropathy. Several theories were postulated to explain the underlying mechanism of papilledema. Of them, mechanical compression theory and an ischemic insult to axons are the most important<sup>3</sup>.

The incidence of different causes of papilledema in a study by Sirisha et al<sup>4</sup>, which included 50 cases of papilledema, is as follows - vascular causes in 19 cases (38%), inflammatory causeswere seen in 12 cases (24%). IIH was seen in 10 cases (20%), and intracranial tumours were seen in 9 cases(18%).

In all cases presenting with papilledema, it is important to assess visual functions and establish an etiological diagnosis. Early identification of the defective vision due to papilledema underscore the urgent treatment directed at the aetiology, which will prevent the blindness. So, the present study is carried out to assess the visual function in patients presenting with papilledema.

#### II. Materials And Methods

This is a Prospective observational study which was carried out on patients of department of Ophthalmology, Sri Venkateswara medical college andSri Venkateswara Ram Narayan Ruia Govt General Hospital, Tirupati. from November2019 to September 2021. A total of 30 patients both males and females were included in this study.

Study design: Hospital-based prospective, observational study.

**Study location :** Department of Ophthalmology, Sri Venkateswara medical college andSri Venkateswara Ram Narayan Ruia Govt General Hospital, Tirupati.

**Study Duration:** one year from the date of ethical committee approval from November2019 to September 2021.

Sample size: A total of thirty subjects with papilledema were included in the study.

#### Inclusion criteria:

- 1. Patients with papilledema and willing to participate in the study were included.
- 2. Patients between the age of 10-60 were included

#### **Exclusion criteria:**

- 1. Cases of pseudo-papilledema
- 2. Patients with hazy media impairing the visualization of the fundus.

#### III. Methodology

After obtaining the approval of the institutional ethical committee a written and informed consent was taken from the patients in his/her vernacular language. A detailed history was taken regarding chief complaints, duration of illness was taken. Clinical examination of the patient included a detailed general physical examination and systemic examination, followed by an ophthalmological examination.

• Best corrected visual acquity was assessed with the Snellen chart and scored with logMAR scale.

• Colour vision examination with ISHIHARA chart(pseudoisochromatic plates).

The patient should be tested single eye at a time, distance of 75-100cm at arm'slength with the illumination of 500-600 lux or daylight illumination and observation for period of 3-5 sec per plate. Interpretation is made by Counting the number of plates misread by the patient, excluding the demonstration plate. If the patient misread 4 or fewer plates among 38 plates indicates normal colour vision. If more than eight plates were misread among 38 plates signifies deficient colour vision

• Slit-lamp examination for anterior segment. (Carl zeissMeditec AG0.7740 Jena Germany)

• Fundus examination with direct and indirect ophthalmoscopy, slit-lamp biomicroscopy using 90Dlens for optic disc assessment.

- Disc evaluation with a direct ophthalmoscope and 90 D.Following details were noted -
- i. Cup size, the colour of the disc
- ii. Cup to disc ratio
- iii. The blurring of the disc margins
- iv. Haemorrhages
- v. Tortuosity
- vi. Venous pulsation
- vii. Hard exudates in the peripapillary area
- viii. Exudates in the macula
- ix. Paton's line

- x. Corpora amylase
- xi. General background
- xii. Macula

# • Visual field examination with Humphrey visual field analyser. (ZeissHumphrey systems CTG-00473)FIELD PROTOCOL:

The 30-2 programme on Humphrey's field analyzer with a white-on-white Goldmann size III target was used for visual field examination. All patients underwenta full threshold strategy for visual field examination. The reliability criteria used were fixation losses <20%, false positive and false negative errors <33%. Only fields reliablyperformed were included in the analysis.

#### Statistical analysis:

Data was collected on predefined cases proforma and transcribed into MS- Excel spreadsheets. All the entries were double-checked to minimise the data entry errors. Data were expressed as mean with standard deviation (SD) for continuous variables and frequencies with percentages for categorical variables. All the statical analysis was performed using SPSS v. 20.0 (IBM Corp, Somers, NY, USA). P-value < 0.05 is considered statistically significant.

#### IV. Result

30 patients with papilledema who attended ophthalmology department OPD at SVRR GGH, SVMC Tirupati who met the inclusion and exclusioncriteria were included in the present study, and the results are analyzed and tabulatedas follows.

#### AGE WISE DISTRIBUTION

The age group of the patients included in the study was 10-60 years. Nearly one-third of the cases (n=10; 33.3%) were in third decade (31-40 years) of their age (table 1). Mean age (SD) 32.5(16.4) years. The age range of the participantswas between 11 to 58 years.

Age group (in years)	Frequency	Percentage
10-20	6	20
21-30	7	23.5
31-40	10	30
41-50	4	13.5
51-60	3	10
Total	30	100

#### TABLE 1: DISTRIBUTION OF PATIENTS ACCORDING TO AGE.

#### GENDER DISTRIBUTION

In the study group containing 30 patients, the majority of the participants were females (53.0%) followed by males (47.0%); the maximum number of males were in the 31-40 years, whereas females were 21-30 years age group. Table 2 shows the gender-wise distribution of patients.

#### TABLE 2: GENDER WISE DISTRIBUTION OF PATIENTS.

Gender	Frequency	Percentage
Males	14	47
Females	16	53
Total	30	100

#### AGE-WISE DISTRIBUTION OF GENDER OF PATIENTS

The highest number of male patients were in the age group of 31-40 with 7 (23.5%) whereas in the females it is in the age group of 11-20 and 21-30 with 4 in each decade. Table 3 show the age-wise distribution of the gender of the patients.

	NO. OF PATIENTSn (%)	MALEn (%)	FEMALEn(%)
AGE GROUP 11-20	6(20)	02(6)	04(13.5)
21-30	7(23.5)	01(3)	06(20)
31-40	10(30)	07(23.5)	03(10)
41-50	4(13.5)	02(6)	02(6)
51-60	3(10)	02(6)	01(3)
TOTAL	30(100)	14(47)	16(53)

#### TABLE 3: AGE-WISE DISTRIBUTION OF GENDER OF PATIENTS.

### AETIOLOGICAL DIAGNOSIS OF PATIENT WITH PAPILLEDEMA

Majority of the patients with papilledema had ICSOL 14 (46.6%) followed by IIHin 8 (26.6%), CSVT in 6(20%) AVM in 1 (3.3%) and SDH in 1 (3.3%) case each. Table4 and figure 1 show the aetiological diagnosis of the patients with papilledema.

Aetiology	Number (n=30)	Percentage	
Idiopathic intracranial hypertension (IIH)	8	26.6	
Intra-cranial Space Occupying Lesion (ICSOL)	14	46.6	
Cerebral Sinus Venous Thrombosis (CSVT)	6	20	
Artery Venous Malformation (AVM)	1	3.3	
Sub Dural Hemorrhage	1	3.3	

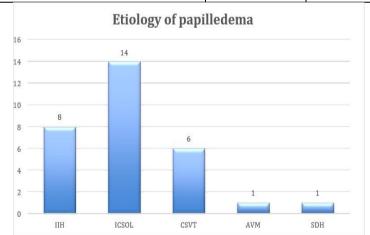


FIGURE 1: SHOWS THE AETIOLOGY OF PATIENTS WITH PAPILLEDEMA

#### BEST-CORRECTED VISUAL ACUITY OF PATIENTS WITH PAPILLEDEMA

Best corrected visual acuity in 26 (43.3%) eyes was 6/6 (log MAR value of 0), 13(21.5%) eyes had 6/12(log MAR value 0.30), 9(15%) eyes had 6/18 (log MAR value0.48),8 (13.3%) eyes had 6/24(log MAR value 0.60) and 4 (6.6%) had 6/36 (log MAR value of 0.78). Table 5 and figure 2 showing the best corrected visual acuity of patients with papilledema.

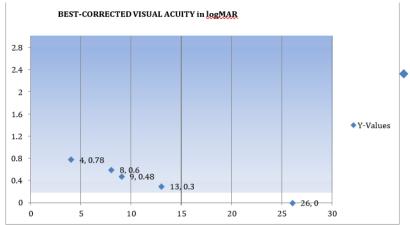


FIGURE 2: BEST-CORRECTED VISUAL ACUITY OF THE PATIENTS.

### TABLE 5: BEST-CORRECTED VISUAL ACUITY IN PATIENTS WITHPAPILLEDEMA.

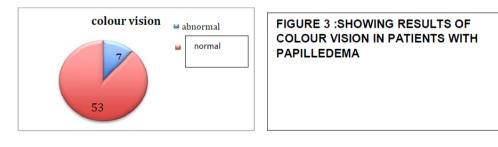
BCVA (SNELLEN)	BCVA (LogMAR)	No. of Eyes (n=60)	Percentage (%)
6/6	0	26	43.3
6/12	0.30	13	21.5
6/18	0.48	9	15
6/24	0.60	8	13.3
6/36	0.78	4	6.6

### COLOUR VISION IN PATIENTS WITH PAPILLEDEMA

Colour vision tested by using the Ishihara chart shows abnormalities in 7(11.5%) out of 60 eyes. Table 6 and figure 3 show the colour vision abnormality in thepapilledema patients.

### TABLE 6: SHOWS COLOUR VISION IN PAPILLEDEMA PATIENTS

Colour vision	No. of eyes	Percentage
Normal	53	88.5
Abnormal	7	11.5
Total	60	100

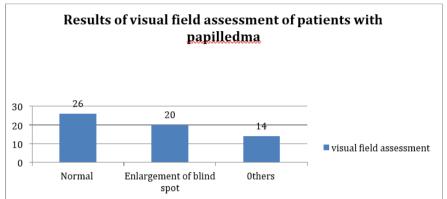


### VISUAL FIELD ASSESSMENT IN PAPILLEDEMA PATIENTS

Visual field assessment was normal in 26 eyes, followed by an enlargement ofblind spot in 20 eyes. Other visual field defects detected in 14 eyes include bitemporalhemianopia, right homonymous hemianopia, and constriction of visual fields. Table 7 and figure 4 show visual fields defects detected. Table 8 showing the other visual field defects detected in patients with papilledema and their etiological diagnosis. Onepatient with recurrent CSVT and another with fulminant IIH had constriction of visual fields. One patient with craniopharyngioma with hydrocephalus and pituitary macroadenoma with hydrocephalus had bitemporal hemianopia. One patient with left parieto-temporal glioma and another patient with left parieto-temporal gaint AVM hadright homonymous hemianopia.

TABLE 7: SHOWS THE VISUAL FIELD CHANGES IN FATIENTS WITHFAFILLEDEWI				
Visual fields	No of eyes	Percentage		
Normal	26	42.5		
Enlargement of blind spot	20	33.5		
Others	14	24		
Total	6	100		





#### FIGURE 4: SHOWING THE RESULTS OF VISUAL FIELD ASSESSMENT OFTHE PATIENT WITH PAPILLEDEMA

#### TABLE 8: SUMMARY OF THE OTHER VISUAL FIELD DEFECTS NOTED INPATIENTS WITH PAPILLEDEMA.

Diagnosis of patient	OTHER VISUAL FIELD DEFECTS
Recurrent CSVT	Constriction of visual fields (BE)
Left parietal-temporal gaint AVM	Right Homonymous Hemianopia (BE)
Fulminant IIH	Constriction of visual fields (BE)
Craniopharyngioma with hydrocephalus	Bitemporal hemianopia
Left parieto - temporal glioma	Right homonymous hemianopia (BE)
Pitutary macroadenoma with	Bitemporal hemianopia
hydrocephalus	
Right parieto-temporal Ganglioglioma	Left homonymous hemianopia

### STAGING OF PAPILLEDEMA

30 (50%) eyes were found to have early Papilledema followed by 24(40%) eyes established Papilledema, 6(10%) had chronic Papilledema. Table 9 and figure 5 showing the stages of papilledema in the patients.

TABLE 9 SHOWS STAGES OF PAPILLEDEMA IN THE EXAMINED EYES				
Stage of papilledema	Number of eyes	Percentage		
Early papilledema	30	50		
Established papilledema	24	40		
Chronic papilledema	6	10		

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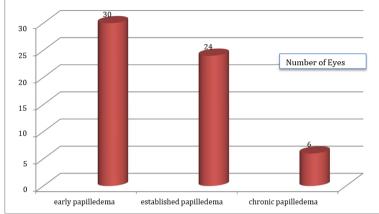


FIGURE 5: SHOWS THE STAGES OF PAPILLEDEMA IN EXAMINED EYES

In Patients with early papilledema 20 out of 30 (66.7%) had BCVA 6/6 whereas 10 out of 30 had >6/6 -6/12 (p-value =0.01). In patients with established papilledema 18(75%) out of 24 had 6/12-6/24 whereas 6(25%) had 6/6 (p- value=0.0006). All patients i.e., 6(100%) with chronic papilledema had BCVA 6-24/6-36 (p-value =0.0009). Table 10 showing stages of papilledema and BCVA.

#### TABLE 10: SHOWING STAGE OF PAPILLEDEMA AND BEST-CORRECTED VISUAL ACUITY

Stages of Papilledema	Best-corrected Visual acuity	t-corrected Visual acuity	
1 apineuenia	Normal	Normal Abnormal	
Early(n=30)	20 (66.7%)	10 (33.3%)	0.01(SS)
Established (n=24)	6(25%)	18(75%)	0.0006(SS)
Chronic (n=6)	0	6(100%)	0.0009(SS)
Total (n=60)	26	34	

S S: Statistically significant

Colour vision in all patients 30 (100%) with early papilledema was normal (p- value <0.0001). In patients with established papilledema 23 (95.8%) out of 24 had normal colour vision whereas 1(4.2%) had abnormal colour vision (p-value<0.0001). All patients 6(100%)/6 with chronic papilledema had abnormal colour vision (p-value =0.0009). Table 11 showing stages of papilledema and colour vision.

Stages of	Color	ır vision	
Papilledema	Normal	Abnormal	P-value
Early(n=30)	30 (100%)	0	<0.0001(SS)
Established (n=24)	23 (95.8%)	1 (4.2%)	<0.0001(SS)
Chronic (n=6)	0	6 (100%)	0.0009(SS)
Total (n=60)	53 (88.5%)	7 (11.5%)	

#### TABLE 11: SHOWING STAGE OF PAPILLEDEMA AND COLOUR VISION

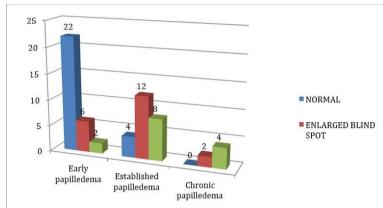
SS: statistically significant

SS: statistically significant

Out of the 30 eyes with early papilledema, 22 (73.3%) eyes had normal visual fields,6 (20%) had enlarged blind spot, and 2 (7.7%) had other field defects. Out of 24eyes with established papilledema, 4 (16.7%) had a normal visual field, 12 (50%) hadenlargement of blind spot, and 8 (33.3%) had other field defects. Four eyes (67%) eyes with chronic papilledema had other field defects and 2(33%) had enlargement of blind spot. Summary of the stages of papilledema and corresponding visual field findings are shown in table 12 and figure 6.

	VISUAL FILEDS ASSESSMENT			
STAGING OF PAPILLEDMA	NORMAL		OTHERFIELD DEFECTS	TOTAL
Early(n=30)	22(73.3%)	6 (20%)	2(7.7%)	30
Established(n=24)	4 (16.7%)	12(50%)	8(33.3%)	24
Chronic(n=6)	0	2(33%)	4(67%)	6
TOTAL(n=60)	26	20	14	60





#### FIGURE 6: SHOWING STAGES OF PAPILLEDEMA AND VISUAL FIELD DEFECTS.

Visual field abnormalities were found in 26.7% (p=0.0003) in early papilledema,83.3% in established papilledema (p<0.0001) and 100% in chronic papilledema (p=0.0009) which was statistically significant. Table 13 showing visual field defects and stages of papilledema.

#### TABLE 13: STAGES OF PAPILLEDEMA AND VISUAL FIELD ASSESSMENTRESULTS WITH STATISTICAL ANALYSIS

Stages of	Visual filed assessment	P-value	
Papilledema	Normal	Abnormal	
Early(n=30)	22 (73.3%)	8 (26.7%)	0.0003(SS)
Established (n=24)	4(16.7%)	20(83.3%)	<0.0001(SS)
Chronic (n=6)	0	6(100%)	0.0009(SS)
Total (n=60)	26	34	

SS: Statistically significant

#### V. Discussion

Papilledema is defined as passive bilateral optic disc oedema (ODE) due to raised intracranial pressure (ICP). It can be an alarming sign for disease entities that cause elevated ICPs such as brain tumours, cerebrospinal inflammation and IIH. Papilledema due to intracranial hypertension may develop at any age, in either of thesex and in any racial or ethnic group. Most often, it results from IIH, of which the annualincidence per one lakh has been estimated to be 0.9 in the general population of the United States of America (USA). The present study to assess the visual function in papilledema is done by subjective methods. The subjective assessmentis done by visual acuity, colour vision, and visual fields.

Most of the patients belong to the 31-40-year age group, which constituted 30 percent of the cases, followed by the 11-20 years age group with 23.5 percent of cases. Only three patients belong to the age group of 51-60. The mean age group of thepatients is 32.5 years. A comparison of the most common age group in various studies shown in table 14. Present study findings are comparable to Agrawal et al<sup>5</sup> study.

ABLE 1	ABLE 14: SHOWS THE COMPARISON OF THE AGE GROUP OF PATIENTS IN VARIOUS STUDIES. Most common agegroup Percentage of			
Most common agegroup Percentage of				
	Study name		09505	

Study name	Most common agegroup	Cases Cases
Present study	31-40	30
Sivakalai et al <sup>6</sup>	21-30	44
Agrawal et al <sup>5</sup>	35-44	21.4

In our study, 53 per cent of the patients are female, with male to female ratio 1:1.2 comparison of male to female patients in various studies is shown in table 15. Most of the previous studies reported female preponderance as the present study. Agrawal et  $al^5$  study showed male preponderance.

# TABLE 15: SHOWS THE MALE TO FEMALE RATIO OF STUDY POPULATION INVARIOUS STUDIES.

Study name	Male to female ratio
Present study	1:1.2
Agrawal et al <sup>5</sup>	2:1
D solanki et al <sup>7</sup>	3:4.1
Ambika et al <sup>8</sup>	1:4
Sivakalai et al <sup>6</sup>	1:2

In the present study most of the patients with papilledema had ICSOL 14 (46.6%) followed by IIH in 8 (26.6%), CSVT in 6(20%), AVM in 1 (3.3%) and SDH in 1(3.3%) case each. Previously reported studies by D Solanki et al<sup>7</sup>, Sivakalai et al<sup>6</sup> reported ICSOL as the most common cause with 24% and 28.9%, respectively, which is comparable to the present study. Paton reported ICSOL in 77.9% of cases<sup>9</sup>.CrumOM et al4 reported ICSOL in 11.6% of patients.

In the present study IIH was reported in 26.6% of cases. Crum OM et al<sup>10</sup> reported IIH as the most common cause of papilledema in 67.4% of cases whereas sivakalai et al<sup>6</sup> reported in 15.5% and D Solanki et al<sup>7</sup> reported 2% of cases. In the present study, CSVT was noted in 20% of the cases. Sivakalai et al<sup>6</sup> reported CSVT 8.8%. Crum OM et al<sup>10</sup> reported CSVT in 2.3%. SDH was noted in 3.3% in the presentstudy. Siva kalai et al<sup>6</sup> reported SDH in 4.4%, Agarwal et al<sup>5</sup> reported SDH in 3.57% which are comparable to the present study. Table 16 shows comparison of aetiology of papilledema withother reported studies.

#### TABLE 16: SHOWS COMPARISON OF THE AETIOLOGY OF PAPILLEDEMA INVARIOUS STUDIES

	Aetiology of papilledema in various studies						
Study name	ICSOL	IIH	CSVT	SDH			
Present study (n=30)	46.6%	26.6%	20%	3.3%			
Sivakalai et <sub>al</sub> 45(n=45)							
	28.9%	15.5%	8.9%	4.4%			
Agarwal et al <sup>46</sup> (n=56)	42.86%	-	16.7%	3.57%			
Solanki et al <sup>48</sup> (n=17)	64.7%	-	11.6%	-			
Crum OM etal <sup>4</sup>	11.6%						
(n=86)	11.070	67.4%	2.3%	-			

In the present study best-corrected visual acuity of 65% of the tested eyes is between 6/6 and 6/12 followed by 35% of the patients between 6/18 and 6/36. Best- corrected Visual acuity findings in various studies on papilledema are shown in table 17. Our findings are comparable to the Agarwal et al.<sup>5</sup> study.

# TABLE 17: SHOWS THE BEST-CORRECTED VISUAL ACUITY FINDINGS INPATIENTS WITH PAPILLEDEMA IN VARIOUS STUDIES.

	ë <b>.</b>	Percentage of eyes		
Study name		between 6/18-6/36		
Present study	65	35		
Agrawal R et al <sup>5</sup>	66	34		
Ambika et al <sup>8</sup>	41.2	20		

Colour vision tested with Ishihara charts showed abnormalities in 11.5% of theeyes tested. Reported Colour vision abnormalities in papilledema in the previous studies are shown in table 18. The present study results are comparable to Wall  $M^{11}$ study.

# TABLE 18: SHOWS COLOUR VISION ABNORMALITIES OF PATIENTS WITHPAPILLEDEMAIN VARIOUS STUDIES

Study name	Percentage
Present study	11.5
Ambika et al <sup>8</sup>	41.6
Wall M <sup>11</sup>	16
Agrawal et al <sup>5</sup>	3.5

RAPD was seen in 8(13.3%) out of 60 eyes in our study. Corbett  $JJ^{12}$  et al.reported RAPD in 4(14.3%)/28 eyes in their study, which are comparable to our study.

Visual field assessment was normal in 43.5 per cent of the eyes tested, enlargedblind spot was found in 33.5 percent and defective visual field was found in 24 percent. A comparison of visual field defects in various previously done studies on papilledemais shown in table 19. Our findings are comparable to the study done by Sivakalai et al<sup>6</sup>.

# TABLE 19: VISUAL FIELD DEFECTS IN PATIENTS WITH PAPILLEDEMA INVARIOUSSTUDIES.

Study	Normal visual field (%)	Enlarged blind spot (%)	Defective Visual field (%)	
Present study	43.5	33.5	24	
Sivakalai et al <sup>6</sup>	44.4	28.9	13.3	
Sirisha et al <sup>4</sup>	34	56	10	
Agrawal et al <sup>5</sup>	61.70	27.65	10.63	

#### DEFECTIVE VISUAL FIELDS IN THE PATIENTS WITH PAPILLEDEMA

Visual field defects other than enlargement of blind-spot was noted in 14(24%)eyes. Details of the visual field defect and their aetiological diagnosis of the patients are shown in table 20. Among them one patient with fulminant IIH and another with recurrent CSVT had constriction of the visual fields with chronic papilledema. Rowe et al<sup>13</sup> also reported global constriction of visual fields in patients with papilledema. One patient with craniopharyngioma with hydrocephalus and pituitary macroadenoma withhydrocephalus had bitemporal hemianopia. A study by Kennedy HB et al<sup>14</sup> reported bitemporal hemianopia in 27% of patients with craniopharyngiomas. One patient withleft parieto- temporal glioma and another patient with left parieto-temporal gaint AVM had right homonymous hemianopia. One patient with right parieto-temporal ganglioglioma had left homonymous hemianopia. In these patients the observed visualfield defects were due to location of the lesion in the visual pathway rather than papilledema.

#### TABLE 20: DETAILS OF OTHER VISUAL FIELD DEFECTS NOTED IN THESTUDY

Diagnosis of patient	OTHER VISUAL FIELD DEFECTS		
Recurrent CSVT	Constriction of visual fields (BE)		
Left parieto-temporal gaint AVM	Right Homonymous Hemianopia		
Fulminant IIH	Constriction of visual fields (BE)		
Craniopharyngioma with hydrocephalus	Bitemporal hemianopia		

Left parieto - temporal glioma	Right homonymous hemianopia
Pituitary macroadenoma with	Bitemporal hemianopia
hydrocephalus	
Right parieto-temporal ganglioglioma	Left homonymous hemianopia
with hydrocephalus	

# A REVIEW OF VISUAL FIELD DEFECTS IN PATIENTS WITH PAPILLEDEMA DUE TO IIH DESCRIBED IN THE IDIOPATHIC INTRACRANIAL HYPERTENSION TREATMENT TRIAL (IIHTT) AND OTHER STUDIES<sup>15</sup>.

The most common visual field defect observed in papilledema is enlarged blindspot. Prevalence of enlargement of blind-spot in various previously reported studies ranged from 33 to 93%. Enlargement of blind spot indicates only optic disc swelling, nothing about the integrity of optic nerve. Peripheral field testing is a better indicator of optic nerve integrity. Accumulation of peripapillary subretinal fluid was the proposed mechanism underlying the development of enlargement of blind-spot. Uncertainty exists regarding the source of subretinal fluid in papilledema; some of the possible sources are the vitreous humor, CSF, and the choroid plexus.

Other field defects noted in papilledema due to IIH includeGeneralized constriction,

Loss of the nasal visual fields (inferonasal)Inferioraltitudinal loss,

Superonasal and superotemporal lossArcuatedefects

Scotomas (central, cecocentral, and paracentral).

# TABLE 21: SHOWING SUMMARY OF VISUAL FUNCTION ASSESSMENT USING SUBJECTIVE METHODSAMONG THE STAGES OF PAPILLEDEMA WITH STATSITICAL ANALYSIS.

	Early (n=30)		Established (n=24)			Chronic (n=6)			
	Normal	Abnormal	p-value	Normal	Abnormal	p-value	Normal	Abnormal	p- value
BEST CORRECTED Visual acuity	20 (66.7%)	10 (33.3%)	0.01*	6 (25%)	18 (75%)	0.0006*	0 (0%)	6 (100%)	0.0009 *
Color Vision	30 (100%)	0 (0%)	<0.0001*	23 (95.8%)	1 (4.2%)	<0.0001*	0 (0%)	6 (100%)	0.0009 *
Visual field	22	8 (26.7%)	0.0003*	4	20	<0.0001*	0 (0%)	6 (100%)	0.0009
	(73.3%)			(16.7%)	(83.3%)				*

\*statistically significant

#### VI. Conclusion

• In the present study, 60 eyes of 30 patients presenting in various stages of papilledema were evaluated for visual functions. The subjective testing included visual acuity, colour vision and visual field recording. The mean age of the subjects was 32.5 years +/- 16.5 SD (Range 11-58) withmaximum distribution in the age group of 31-40 Years .The majority of the subjects were females, 53%, and males were 47%.Early Papilledema was found in 50% of eyes, established papilledema in 40% and chronic Papilledema in 10%.The most common aetiology causing the papilledema in patients included in the study was ICSOL in

46.6 %, followed by IIH in 26.6 % CSVT in 20% and SDH in 3.3%.Best-corrected visual acuity was 6/6 in 66.7% in early papilledema and 25% inestablished Papilledema.Visual acuity was normal in the majority of the patients (66.7%) with early Papilledema (p=0.01), was reduced in the majority(75%) of the patients with established papilledema (p=0.0006) and in all patients (100%) with chronic Papilledema (p=0.0009). Visual acuity decreased as the stage of Papilledema progressed to the chronic stage, which was statistically significant. Colour vision assessment was normal in all (100%) patients in early papilledema (p<0.0001) and the majority (97.8%) of patients with established papilledema p < 0.0001 and chronic p=0.0009). The most common visual field defect observed was the enlargement of the blind spot (33.5%). Abnormal visual fields were recorded in all stages of Papilledema with a p- value that was statistically significant (early papilledema p=0.0003, established papilledema p < 0.0001 and chronic p=0.0009).

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