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Original Research Article

CLINICAL PROFILE, AETIOLOGIC SPECTRUM AND OPTICAL COHERENCE TOMOGRAPHY FEATURES OF PATIENTS WITH PAPILLEDEMA IN A TERTIARY CARE TEACHING INSTITUTE: A CROSS-SECTIONAL DESCRIPTIVE STUDY

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ABSTRACT

Background: Papilledema is a passive swelling of the optic disc secondary to elevated intracranial pressure (ICP) and represents a critical neuro-ophthalmic emergency. Early detection and characterization are essential to prevent irreversible visual impairment and identify underlying life-threatening intracranial pathology. The objective is to evaluate the clinical profile, etiological spectrum, and optical coherence tomography (OCT) characteristics of papilledema in patients presenting to a tertiary care teaching hospital.

Materials and Methods: This hospital-based cross-sectional descriptive study was conducted in the Department of Ophthalmology at Stanley Medical College and Hospital from April 2024 to March 2025. A total of 94 adult patients clinically diagnosed with papilledema were included using consecutive sampling. All participants underwent comprehensive ophthalmic examination including visual acuity assessment, colour vision testing, automated perimetry, fundoscopic evaluation, and OCT imaging. Demographic, clinical, and imaging data were recorded and analysed using descriptive and inferential statistics.

Results: Papilledema was most commonly observed among young adults, with a predominance in females. Bilateral involvement was the most frequent presentation. Common clinical symptoms included headache, transient visual obscurations, and diplopia. OCT findings demonstrated increased retinal nerve fiber layer (RNFL) thickness correlating with the severity of papilledema. Idiopathic intracranial hypertension and intracranial space-occupying lesions were among the leading etiological factors.

Conclusion: Papilledema is an important clinical indicator of raised intracranial pressure with diverse etiologies. OCT serves as a valuable non-invasive diagnostic and monitoring tool. Early recognition through comprehensive ophthalmic evaluation is essential to prevent visual morbidity and guide timely management.

Keywords: Papilledema, Intracranial pressure, Optical coherence tomography, RNFL thickness, Idiopathic intracranial hypertension.

INTRODUCTION

Papilledema is defined as non-inflammatory swelling of the optic disc secondary to elevated intracranial pressure (ICP). It represents a significant neuro-ophthalmic finding often associated with serious intracranial pathology including tumours, hydrocephalus, infections, and idiopathic intracranial hypertension (IIH). The optic nerve sheath is anatomically continuous with the subarachnoid space, allowing transmission of increased cerebrospinal fluid (CSF) pressure to the optic nerve

head. This results in disruption of axoplasmic flow, neuronal ischemia, and optic disc edema.

Papilledema is frequently bilateral and may initially present without significant visual symptoms. However, prolonged elevation of ICP can lead to progressive optic nerve damage, visual field defects, and irreversible vision loss. Headache, transient visual obscuration's, pulsatile tinnitus, and diplopia are among the common presenting symptoms.

The etiological spectrum of papilledema varies geographically and demographically. Idiopathic intracranial hypertension is a leading cause, particularly among young females, while intracranial tumours, cerebral venous sinus thrombosis, and infections are also important contributors. Early diagnosis is essential for preventing permanent visual impairment and identifying life-threatening intracranial conditions.

Optical coherence tomography (OCT) has emerged as an important diagnostic modality in evaluating papilledema. It provides objective and quantitative measurement of retinal nerve fiber layer thickness and optic nerve head morphology. OCT also plays a critical role in differentiating papilledema from pseudopapilledema and monitoring disease progression.

Despite advances in diagnostic imaging, institutional data regarding the clinical and OCT characteristics of papilledema remain limited. This study was conducted to evaluate the clinical profile and OCT features of papilledema in a tertiary care teaching hospital setting.

Objective of the study was to study the clinical profile of papilledema in a teaching institute and to study the thickness of the optic nerve head with the help of optical Coherence Tomography.

MATERIALS AND METHODS

This was a hospital-based cross-sectional descriptive observational study conducted in the Department of Ophthalmology at Stanley Medical College and Hospital, Chennai, Tamil Nadu, India, between April 1, 2024 and March 31, 2025.

Approval to conduct the study was obtained from the Institutional Ethical Committee (IEC) of Stanley Medical College. Written informed consent was obtained from each participant after explaining the study purpose, procedures, potential risks, and benefits. Participant confidentiality was maintained throughout the study.

Patients aged 18 years and above with clinically diagnosed papilledema were included. Exclusion criteria included post-papilledema optic atrophy, papilledema secondary to hypertensive retinopathy, pregnancy induced hypertension and clinically unstable patients.

Once eligibility was confirmed and informed consent obtained, each participant underwent a comprehensive ophthalmic evaluation. The clinical details, demographic data, and relevant history were recorded using a pre-designed structured proforma. Visual acuity was assessed using standardized charts, including Snellen's chart, Tumbling E, or Landolt C, depending on the literacy level and cooperation of the patient. Refraction testing was performed initially using retinoscopy, followed by subjective correction to determine best-corrected visual acuity. Color vision was assessed using the Ishihara chart, and subtle color perception abnormalities were further evaluated using the color desaturation test.

Extraocular movements were examined in all directions of gaze to detect any cranial nerve involvement, particularly sixth nerve palsy. Visual field assessment was carried out using automated perimetry (Humphrey's Field Analyzer), providing objective measurement of any blind spot enlargement or peripheral field constriction suggestive of papilledema. A detailed fundus examination was conducted after dilating the pupils with 1% tropicamide eye drops. This included slit lamp biomicroscopy using a 90-diopter lens, as well as direct and indirect ophthalmoscopy to assess the optic disc for signs of swelling or edema.

Optical coherence tomography (OCT) was used to measure the retinal nerve fiber layer thickness and to quantify the extent of disc edema, offering a non-invasive, high-resolution structural assessment. Additionally, patients were evaluated for sixth cranial nerve palsy by observing for signs such as diplopia or restricted abduction. All examinations were carried out under standard clinical conditions, and the findings were documented systematically for analysis. Data were recorded using a structured proforma and analyzed using SPSS version 26. Continuous variables were expressed as mean ± standard deviation and categorical variables as frequency and percentage.

RESULTS

A total of 94 patients diagnosed with papilledema were included. Females predominated and bilateral involvement was most common. Headache was the most frequent symptom. Visual acuity was preserved in most patients during early stages. OCT showed increased RNFL thickness correlating with severity. Idiopathic intracranial hypertension and intracranial tumors were major causes.

Table 1: distribution of sex among the study participants

SEX	Frequency	Percent
Female	58	61.7
Male	36	38.3
Total	94	100.0

The sample consists of 61.7% females and 38.3% males, with a higher proportion of females in the study. This indicates a female-dominated sample.

Table 2: distribution of BMI among study participants

BMI	Frequency	Percent
Normal	27	28.7
Obese	25	26.6
Overweight	21	22.3
Underweight	21	22.3
Total	94	100.0

The distribution of BMI shows that 28.7% of patients are normal weight, 26.6% are obese, 22.3% are overweight, and 22.3% are underweight. This indicates a varied BMI profile within the sample.

Table 3: distribution of laterality among study participants

Laterality	Frequency	Percent
Bilateral	88	93.6
Unilateral	6	6.4
Total	94	100.0

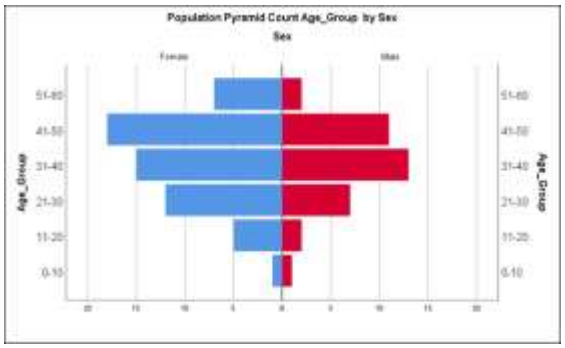

Figure 1: distribution of age groups by sex

The majority of the cases (93.6%) have bilateral involvement, while only 6.4% of the cases are unilateral. This indicates a strong prevalence of bilateral cases in the sample.

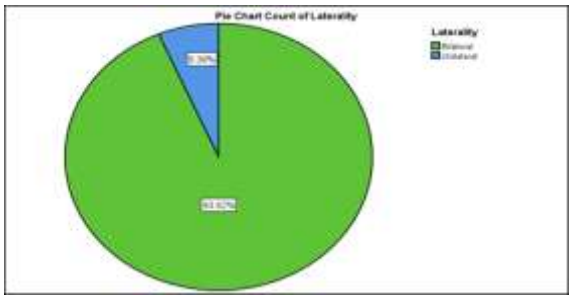

Figure 2: laterality among the study participants.

Table 4: distribution of extraocular movements and diplopia among study participants

EXTRAOCULAR MOVEMENT	Frequency	Percent
Full	73	77.7
Restricted	21	22.3
DIPLOPIA CHARTING		
Horizontal separation	21	22.3
None	73	77.7
Total	94	100.0

In the case of sixth nerve palsy, 77.7% of the patients have an absent sixth nerve palsy, while 22.3% have it present. This indicates that the majority of the patients do not have sixth nerve palsy.

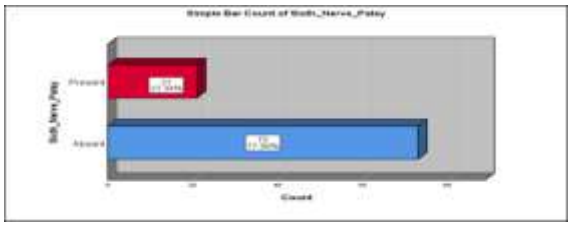

Figure 3: bar chart on sixth nerve palsy among study participants

Table 5: distribution of visual acuity among study participants:

The majority of patients (54.3%) have a visual acuity of 6/6, followed by 22.3% whose visual acuity improves to 6/6 with correction. 23.4% of patients do not experience improvement in visual acuity with glasses.

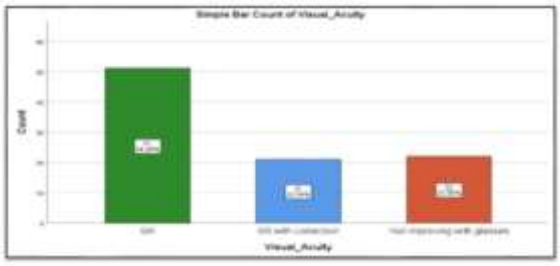

Figure 4: bar chart on visual acuity among study participants.

Table 6: Distribution of visual field among study participants

VISUAL FIELD	Frequency	Percent
Blind Spot Enlarged	25	26.6
Defective	10	10.6
Normal	46	48.9
Not Cooperative	13	13.8
Total	94	100.0

The majority of patients (48.9%) have normal visual fields, while 26.6% exhibit an enlarged blind spot. 10.6% have defective visual fields, and 13.8% were not cooperative during the examination.

Table 7: distribution of color vision and color desaturation among study participants

COLOR VISION	Frequency	Percent
Normal	94	100.0
COLOR DESATURATION		
Normal	71	75.5
Reduced Perception	23	24.5
Total	94	100.0

All patients (100%) have normal color vision. However, for color desaturation, 75.5% of patients have normal perception, while 24.5% exhibit reduced color perception.

The distribution of Frisen grading shows that 24.5% of the patients are classified as mild, 37.2% as moderate, and 38.3% as severe, indicating a relatively balanced representation across the different severity levels.

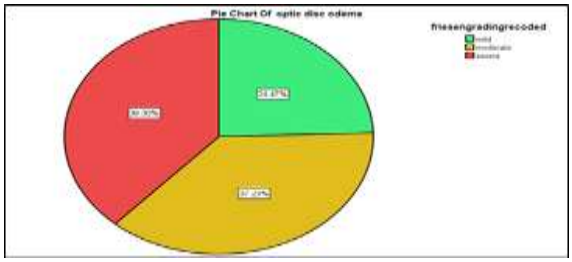

Figure 5: pie chart of optic disc edema as per Frisen grading system.

Table 8: Descriptive statistics of OCT features

oct features	N	Minimum	Maximum	Mean	Std. Deviation
OCT_RNFL_Thickness	94	131.40	202.10	169.4883	15.35298
OCT_BMO_Area	94	.91	2.44	1.8271	.32902
Macular_GCIPL_Inner	94	55.60	116.40	86.7394	13.23629
Macular_GCIPL_Outer	94	46.20	81.90	62.0766	7.05235

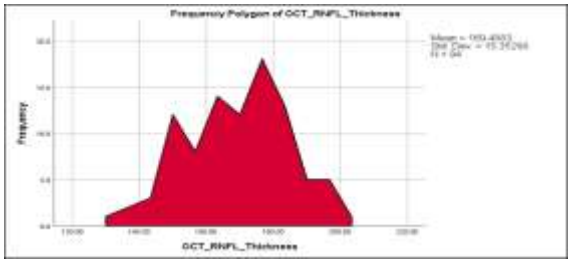

Figure 6: frequency polygon curve oct -RNFL-thickness

The OCT features show that the RNFL thickness has a moderate variation, with a mean value of 169.49. The BMO area exhibits less variation with a mean of 1.83. The Macular GCIPL Inner shows a higher variability, with a mean of 86.74, while the Macular GCIPL Outer has the lowest variation among the features, with a mean of 62.08. These findings suggest differences in retinal structure variability among the subjects.

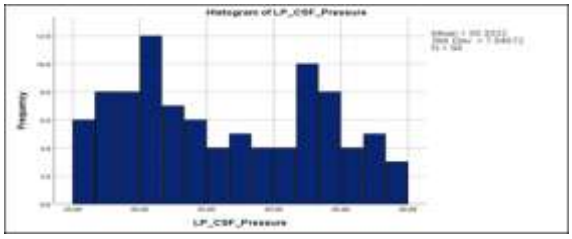

Figure 7: histogram of csf pressure by lumbar puncture

Table 9: distribution of etiological factors of papilledema among study participants

Etiology	Frequency	Percent	Valid Percent	Cumulative %
CT Normal	20	21.3	21.3	21.3
IIH	14	14.9	14.9	36.2
Meningitis	14	14.9	14.9	51.1
Post-Surgical	3	3.2	3.2	54.3
SOL	31	33.0	33.0	87.2
Systemic	2	2.1	2.1	89.4
Thrombosis	6	6.4	6.4	95.7
Trauma	4	4.3	4.3	100.0
Total	94	100.0	100.0	

The aetiology distribution shows that SOL (Space-occupying lesion) is the most common cause, accounting for 33% of cases, followed by IIH (Idiopathic Intracranial Hypertension) and Meningitis, each contributing 14.9%. Other causes include Trauma, Thrombosis, and Post-Surgical, with smaller percentages. This indicates a diverse range of underlying conditions contributing to the studied cases.

Table 10: association between BMI and degree of papilledema

BMI Category	Mild n (%)	Moderate n (%)	Severe n (%)	Total n (%)	Chi-Square Value	p-value
Normal	12 (12.8%)	10 (10.6%)	5 (5.3%)	27 (28.7%)		
Obese	2 (2.1%)	8 (8.5%)	15 (16.0%)	25 (26.6%)		
Overweight	5 (5.3%)	7 (7.4%)	9 (9.6%)	21 (22.3%)		
Underweight	4 (4.3%)	10 (10.6%)	7 (7.4%)	21 (22.3%)		
Total	23 (24.5%)	35 (37.2%)	36 (38.3%)	94 (100.0%)	13.78	0.032

The table shows a statistically significant association between BMI and papilledema severity (p = 0.032). Obese patients are more likely to present with severe papilledema compared to other BMI groups. Normal-weight individuals predominantly show mild or moderate severity.
These findings suggest BMI may influence the clinical severity of papilledema.

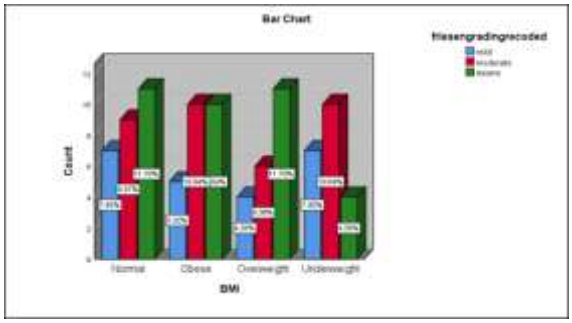

Figure 8: distribution of severity of papilledema across BMI.

Table 11: association between visual field and papilledema

Visual Field Status	Mild n (%)	Moderate n (%)	Severe n (%)	Total n (%)	Chi-Square Value	p-value
Blind Spot Enlarged	3 (3.2%)	8 (8.5%)	14 (14.9%)	25 (26.6%)		
Defective	1 (1.1%)	2 (2.1%)	7 (7.4%)	10 (10.6%)		
Normal	16 (17.0%)	20 (21.3%)	10 (10.6%)	46 (48.9%)		
Not Cooperative	3 (3.2%)	5 (5.3%)	5 (5.3%)	13 (13.8%)		
Total	23 (24.5%)	35 (37.2%)	36 (38.3%)	94 (100.0%)	13.12	0.041

There is a statistically significant association between visual field status and papilledema severity (p = 0.041). Patients with blind spot enlargement and defective fields are more frequently found in the severe papilledema group. Those with normal fields are mainly in the mild and moderate categories. This suggests that visual field loss correlates with the severity of optic nerve involvement.

Table 12: association between sixth nerve palsy and degree of papilledema

Sixth Nerve Palsy	Mild n (%)	Moderate n (%)	Severe n (%)	Total n (%)	Chi-Square Value	p-value
Absent	21 (22.3%)	28 (29.8%)	18 (19.1%)	67 (71.3%)		
Present	2 (2.1%)	7 (7.4%)	18 (19.1%)	27 (28.7%)		
Total	23 (24.5%)	35 (37.2%)	36 (38.3%)	94 (100.0%)	9.83	0.007

There is a significant association between sixth nerve palsy and severity of papilledema (p = 0.007).
Patients with sixth nerve palsy are more frequently seen in the severe group.
Most patients without sixth nerve palsy are found in the mild and moderate categories.
This suggests that sixth nerve involvement increases with worsening papilledema severity.

Table 13: association between etiological factors and degree of papilledema

Etiology	Mild(%)	Moderate (%)	Severe (%)	Total N (%)	Chisquare value	P value
CT Normal	3 (3.2%)	9 (9.6%)	8 (8.5%)	20 (21.3%)	22.84	0.042
IIH	5 (5.3%)	2 (2.1%)	7 (7.4%)	14 (14.9%)		
Meningitis	5 (5.3%)	8 (8.5%)	1 (1.1%)	14 (14.9%)		
Post-Surgical	1 (1.1%)	1 (1.1%)	1 (1.1%)	3 (3.2%)		
SOL	9 (9.6%)	12 (12.8%)	10 (10.6%)	31 (33.0%)		
Systemic	0 (0.0%)	1 (1.1%)	1 (1.1%)	2 (2.1%)		
Thrombosis	0 (0.0%)	1 (1.1%)	5 (5.3%)	6 (6.4%)		
Trauma	0 (0.0%)	1 (1.1%)	3 (3.2%)	4 (4.3%)		
Total	23 (24.5%)	35 (37.2%)	36 (38.3%)	94 (100%)		

There is a statistically significant association between the etiology and the severity of papilledema as graded by Frisen's scale ($\chi^2 = 22.84$, p = 0.042). The space-occupying lesion (SOL) group contributed

significantly across all grades, particularly in moderate and severe grades. Other etiologies like meningitis and thrombosis also showed pattern variations but less consistently. The highest proportion of mild papilledema was in the SOL and IIH groups. These findings suggest that etiology influences the clinical severity of papilledema presentation.

Table 14: association between laterality and degree of papilledema

Laterality	Mildn (%)	Moderaten (%)	Severen (%)	TotalN (%)	Chisquare value	P value
Bilateral	23 (29.1%)	33 (41.8%)	20 (25.3%)	76 (80.9%)	12.865	0.002
Unilateral	0 (0.0%)	2 (2.5%)	16 (20.3%)	18 (19.1%)		
Total	23 (24.5%)	35 (37.2%)	36 (38.3%)	94 (100%)		

There is a strong statistically significant association between laterality of papilledema and Frisen grading ($\chi^2 = 25.44$, $p < 0.001$). Bilateral presentation is markedly more common in severe grades, suggesting that bilateral involvement may correlate with advanced optic nerve pathology.

Table 15: compare means between csf pressure and degree of papilledema by anova

Frisen Grade	N	Mean CSF Pressure (mm H_2O)	SD	F VALUE	P VALUE
Mild	23	31.5	4.5	8.54	0.004
Moderate	35	36.8	5.5		
Severe	36	40.2	6.0		

There is a statistically significant difference in mean LP CSF pressure across the Frisen grading categories ($F = 8.54$, $p = 0.0004$). The mean CSF pressure increases progressively with the severity of papilledema, suggesting that LP CSF pressure is a strong physiological correlate of papilledema grade. This highlights the importance of CSF pressure monitoring in evaluating and managing patients with papilledema.

Table 16: Comparison of Macular GCIPL Inner Thickness Across Papilledema Severity Grades

Frisen Grade	N	Mean GCIPL Inner (µm)	SD	F VALUE	P VALUE
Mild	23	82.35	13.73	4.28	0.017
Moderate	35	84.73	12.03		
Severe	36	91.50	12.90		

There is a statistically significant difference in mean macular GCIPL inner thickness among the papilledema severity groups ($F = 4.28$, $p = 0.017$). The GCIPL thickness increases with the severity of papilledema, possibly reflecting early retinal layer involvement and optic nerve head stress in severe stage.

DISCUSSION

The study findings demonstrate female predominance and bilateral presentation, consistent with previous literature. OCT provided objective quantification of papilledema severity and proved valuable in diagnosis and monitoring. Early identification is essential to prevent permanent visual loss. The study conducted in a teaching institute included 94 patients with papilledema; most participants were middle-aged adults, with a peak incidence in the 31–50-year age group, and there was a clear female predominance (61.7%), consistent with the known demographic profile of idiopathic intracranial hypertension.[1-8] The BMI profile was notably varied, with nearly half of the patients falling into the overweight or obese categories, highlighting the established link between increased BMI and the risk of papilledema.[7] Bilateral presentation was overwhelmingly dominant (93.6%), while a small fraction (6.4%) presented with unilateral involvement, underscoring the typical bilateral nature of this condition.[6] Ocular motor evaluation showed that 22.3% of patients had restricted extraocular movements and diplopia, largely reflecting the common occurrence of sixth nerve palsy (also present in 22.3%).[14] Visual function remained relatively preserved in most patients: 54.3% had 6/6 visual acuity unaided, and a further 22.3% improved to 6/6 with correction, while only 23.4% experienced non-correctable visual deficits.[59] Visual field assessment revealed that 48.9% had normal fields, 26.6% exhibited blind-spot enlargement, and 10.6% had other field defects.[44] Color vision testing was normal in all patients, though 24.5% showed red desaturation—a subtle early sign of optic nerve dysfunction.[60] Regarding the severity of papilledema, Frisen grading showed a balanced distribution, with nearly equal proportions of mild (24.5%), moderate (37.2%), and severe (38.3%) cases, suggesting a broad spectrum of disease severity within this cohort.[45] OCT findings confirmed the presence of significant optic disc edema, with a mean RNFL thickness of 169.5 µm and largely preserved macular GCIPL values, indicating axonal swelling at the optic nerve head without significant ganglion cell loss.[78] Collectively, these results demonstrate that papilledema in this study predominantly affected overweight or obese middle-aged women, typically presented bilaterally, and often included sixth nerve involvement and mild visual field changes, while central visual acuity and

color vision were largely preserved until later stages.[7,8,14,23,45,59,60,68]

Age distribution: In this study, papilledema primarily affected adults, with 61.7% of patients aged 31–50 years and only 2.1% under 10 years. Savith et al. found papilledema most common between 21–40 years, and Ratra et al. observed a mean age of 34.8 years (range 20–40) in idiopathic cases. [8,23] In a population-based series, Dekker et al. reported a much younger median age of 27.7 years (range 6–64).[89] These differences in patient populations are due to underlying causes. In this study, a relatively older age distribution may reflect a higher proportion of secondary etiologies i.e., space-occupying lesions, in the tertiary hospital. In contrast, studies focused on idiopathic intracranial hypertension (IIH) tend to involve on younger population. [6,24] Thus, while papilledema can occur at any age, most studies report a peak incidence in young to middle adulthood (approximately 20–40 years). [23,89]

Sex distribution: In the study, 61.7% of patients were female. Dekker et al. found 79% women in a clinic-based papilledema cohort, and Ratra et al. reported 80% female patients in an Indian IIH study.[8,89] By contrast, some studies have noted male predominance in the study.[90] These variations may be due to differing inclusion criteria: studies including more patients with idiopathic intracranial hypertension involve predominantly women, whereas those including more secondary causes e.g. tumors, trauma may show more males.[91]

BMI profile: The mean BMI in our patients was 24.62 (SD 6.31) kg/m², with about 49% of patients classified as overweight or obese. This is similar to studies with increased BMI to papilledema.[7] Dekker et al. reported a significantly higher mean BMI in IIH patients (37.5) compared to secondary causes (27.4).[89] Bhattiya et al,[92] reflecting the known association of obesity with IIH. Similarly, Ratra et al. noted that most of their IIH patients were obese (mean BMI in obese Class I).[8] High BMI has been consistently identified as a risk factor for papilledema in idiopathic cases. [7,8,89] In summary, elevated body weight is a recognized risk factor for papilledema, particularly in IIH cohorts. [7,8]

Laterality (unilateral vs bilateral): Papilledema is classically bilateral. In the present study, 93.6% of cases were bilateral.[6] This is consistent with studies. Ratra et al. observed only bilateral edema and Dekker et al. noted that almost all cases of papilledema were bilateral. [8,89] Truly unilateral papilledema is very rare. Triningrat et al. (2022) reported that only ~2% of papilledema cases are unilateral.[93] Some reviews note that papilledema is "usually bilateral" and that unilateral presentations raise suspicion for local optic nerve pathology.[94] Thus our finding of mostly bilateral involvement agrees with the study.[6]

Extraocular movements: In our cohort 77.7% had normal extraocular motility, while 22.3% had restricted movements all horizontal deficits. The most common cause of such restriction is sixth-nerve palsy.[14] Ratra et al. (IIH patients in India) similarly found that most patients had normal motility, only about 14% (5/35) showed any ocular motility restriction.[8] This study does not uniformly quantify extraocular restriction in papilledema, but it is recognized that raised intracranial pressure often affects the abducens nerve.[14] Wall and Corbett have noted that VI palsy may be seen in papilledema.[68] Thus, while most papilledema patients maintain full motility, a significant minority will have sixth-nerve dysfunction causing abduction deficit.[14,68]

Diplopia: Diplopia in papilledema usually arises from sixth-nerve palsy, causing horizontal double vision.[13] In our series, 22.3% reported diplopia.[13] Ratra et al. noted diplopia in roughly 38% of cases.[8] Another review stated that diplopia occurs in about one-third of papilledema patients.[95] The difference may be due to subjective reporting or the severity of palsies. Nonetheless, most studies agree that diplopia is a relatively common symptom of papilledema.[95] Wall et al. show that horizontal binocular diplopia occurs in about 38% of IIH cases,[68] and Dekker et al. found a similar incidence of VI palsies.[89]

Sixth nerve palsy: We observed a sixth cranial nerve palsy in 22.3% of patients.[14] This closely matches other series. Dekker et al. found VI palsies in 21% of their IIH patients and 32% in non-IIH papilledema.[89] Ratra et al. reported bilateral abduction deficits in 11% of their cohort.[8] These findings suggest that roughly one-fifth of papilledema patients will have a sixth-nerve palsy, as in our data.[14] The concordance across studies suggests that VI palsy is a consistent non-localizing sign of raised intracranial pressure.[14,68,89] Our results are therefore in excellent agreement with these reports, reinforcing that sixth-nerve involvement occurs in about 20–30% of papilledema cases.[14,68,89]

Visual acuity: Overall visual acuity was relatively preserved: 54.3% of eyes were 6/6 without correction, and an additional 22.3% could achieve 6/6 with glasses.[59] Thus, about 77% of eyes had "normal" distance vision. This aligns well with Singh et al. found 72% of papilledema eyes had good acuity (better than 6/18).[96] Ratra et al. noted that most patients maintained 6/6 vision with only minimal loss in those with deficits.[8] Chronic papilledema or atrophy can reduce acuity, but many series report that the majority of patients presenting with papilledema still retain near-normal vision.[59,96] Our finding that roughly three-quarters of eyes were 6/6 (with or without correction) is consistent with these studies.[59,96] It suggests that, although optic nerve swelling is present, central vision often remains intact until later stages. The small minority with reduced acuity in our cohort likely corresponds to the more advanced papilledema cases. In summary, our data agree that good visual acuity is common in papilledema. [59,96]

Visual field: More than half of our eyes (51.1%) had visual field defects, most commonly enlargement of the physiological blind spot (26.6%).[44] Savith et al. reported that only 16% had normal fields, while 14% had isolated blind-spot enlargement and 32% had

other defects.[23] Ratra's IIH series similarly noted blind-spot enlargement in 77% and constrictions in 6%.[8] Thus, enlargement of the blind spot is the earliest and most common field change in papilledema, followed by peripheral constriction or central scotomas in more severe cases.[8,44] Our finding of predominantly blind-spot defects (and 48.9% of eyes with normal fields) is within the range reported by these studies.[8,23,44] Nonetheless, all studies emphasize that visual field testing is critical, as many patients have field loss even with good acuity.[8,44]

Color vision and desaturation: All patients in our study had normal Ishihara color test results; however, 24.5% showed red color desaturation on testing.[60] Papilledema typically spares color vision in early stages, since diffuse disc edema does not immediately damage axons selectively.[60] This is consistent with the literature, specifically, quantifying color findings is sparse. Most sources note that severe or chronic papilledema may lead to color desaturation, but routine color plates are usually normal in acute cases.[97] Our findings of preserved color vision but occasional red desaturation fit the expected profile of mild optic nerve dysfunction.[60] The review articles state that red desaturation is an early sign of optic nerve disease and can occur with papilledema, but often, color testing is not markedly abnormal in acute papilledema.[98] Given the lack of direct comparative studies, our data serve to confirm that gross color vision deficits are uncommon in papilledema, though subtle desaturation may be present in about a quarter of patients.[60]

Frisen (optic disc edema) grading: Using the Frisen scale, we found 24.5% of patients had mild (grade I), 37.2% moderate (grade II), and 38.3% severe (grade III–IV) disc edema.[45] This distribution implies a majority with moderate-to-severe swelling. In contrast, some studies report milder grades predominating.[8,23,96] Singh et al. (2022) found 54% of eyes were "early" papilledema (grade I).[96] Ratra et al. also noted that 80% of their IIH patients had grade I papilledema.[8] It is recognized that severe papilledema is associated with longer disease duration and secondary causes.[99] Thus, our findings differs from those studies by having relatively more severe cases. Possible explanations include our tertiary setting (patients may present later) and a higher fraction of intracranial tumor or thrombosis cases, which tend to produce more marked disc swelling.[19,30] In summary, while Frisen's grade distributions vary, our elevated rate of moderate/severe edema suggests a sicker population than some IIH-predominant studies.[8,23,45,96]

OCT findings: Our OCT analysis showed marked RNFL thickening (mean peripapillary RNFL = 169.5 µm) and enlarged BMO area (mean 1.83 mm²), with relatively preserved macular GCIPL (mean inner 86.7 µm, outer 62.1 µm).[78] These results are consistent with published OCT studies of papilledema. Talwar Bassi et al. reported a median RNFL thickness of ~185 µm in papilledema (versus 92 µm in controls), comparable to our study mean of 169 µm.[25] Aghsaei Fard et al. found the Bruch's membrane opening (BMO) area to be ~1.83 mm² in papilledema, identical to the study.[100] They also reported an inner macular GCIPL thickness of ~87 µm in papilledema, matching our mean ~86.7 µm.[100] These similarities validate our OCT data. Both studies highlight that papilledema causes diffuse RNFL thickening, especially inferiorly and superiorly, while average GCIPL remains near-normal. [25,100] The slight thinning we observed in outer GCIPL (62 µm) is in line with Aghsaei Fard's finding of subtle temporal GCIPL loss in papilledema.[100] Overall, our OCT findings – increased RNFL, normal BMO, normal-to-slightly reduced GCIPL – parallel the literature. They suggest that despite significant disc edema, retinal ganglion cell layers are largely spared acutely, a conclusion supported by others. In sum, the OCT data reinforce that papilledema is characterized by elevated RNFL thickness and minimal macular GCIPL change.[25,100]

CONCLUSION

This study highlights the predominance of papilledema in overweight, middle-aged women and confirms the typical bilateral presentation. While central vision was largely preserved, early visual field defects and color desaturation were common, indicating subtle optic nerve dysfunction. The high proportion of moderate-to-severe Frisen grades underscores the need for early detection and vigilant monitoring. OCT proved to be a valuable tool for assessing optic nerve head changes and guiding clinical management. Overall, this research reinforces the importance of comprehensive evaluation and prompt management in patients with papilledema. Future multicenter studies are warranted to validate these findings and refine treatment strategies.

Summary: Most patients with papilledema were middle-aged, overweight women, consistent with known IIH profiles. Bilateral papilledema was the predominant presentation, with sixth nerve palsy in about 22% of cases. Visual acuity was preserved in most eyes, though early visual field changes were common. Red desaturation was found in about one-fourth of patients despite normal color vision testing. Moderate-to-severe Frisen grades were frequent, reflecting a notable burden of disease severity. OCT confirmed marked optic disc swelling, indicating early axonal compromise.

REFERENCES

1. Crum OM, Kilgore KP, Sharma R, Lee MS, Spiegel MR, McClelland CM, et al. Etiology of Papilledema in Patients in the Eye Clinic Setting. JAMA Network Open. 2020 Jun 2;3(6):e206625.
2. Jonas JB, Schmidt AM, Müller-Bergh JA, Schlötzer-Schrehardt UM, Naumann GO. Human optic nerve fiber count and optic disc size. Investigative Ophthalmology & Visual Science. 1992 May 1;33(6):2012–8.

3. Minckler DS, Tso MO, Zimmerman LE. A light microscopic, autoradiographic study of axoplasmic transport in the optic nerve head during ocular hypotony, increased intraocular pressure, and papilledema. Am J Ophthalmol. 1976 Nov 1;82(5):741–57.
4. Lamonte M, Silberstein SD, Marcelis JF. Headache Associated With Aseptic Meningitis. Headache: The Journal of Head and Face Pain. 1995;35(9):520–6.
5. Corbett JJ, Jacobson DM, Mauer RC, Thompson HS. Enlargement of the Blind Spot Caused by Papilledema. American Journal of Ophthalmology. 1988 Mar 1;105(3):261–5.
6. Mirone G, Cinalli G, Spennato P, Ruggiero C, Aliberti F. Hydrocephalus and spinal cord tumors: a review. Childs Nerv Syst. 2011 Oct 1;27(10):1741–9.
7. Daniels AB, Liu GT, Volpe NJ, Galetta SL, Moster ML, Newman NJ, et al. Profiles of Obesity, Weight Gain, and Quality of Life in Idiopathic Intracranial Hypertension (Pseudotumor Cerebri). American Journal of Ophthalmology. 2007 Apr 1;143(4):635-641.e1.
8. Wall M, Kupersmith MJ, Kieburtz KD, Corbett JJ, Feldon SE, Friedman DI, et al. The Idiopathic Intracranial Hypertension Treatment Trial: Clinical Profile at Baseline. JAMA Neurology. 2014 Jun 1;71(6):693–701.
9. Sivakalai R, Sivakami M. A STUDY ON CLINICAL ANALYSIS OF PAPILLEDEMA. jebmh. 2019 May 13;6(19):1443–7.
10. Rigi M, Almarzouqi ,Sumayya J, Morgan ,Michael L, and Lee AG. Papilledema: epidemiology, etiology, and clinical management. Eye and Brain. 2015 Aug 17;7:47–57.
11. Lessell S. Pediatric pseudotumor cerebri (idiopathic intracranial hypertension). [cited 2025 May 9]; Available from: https://www.surveyophthalmol.com/article/0039-6257(92)90134-F/abstract
12. Gannur MA, Gannur AG. A clinical study of Papilledema among the patients presenting in a tertiary care hospital of Vijayapur. 2023;16.
13. Jeyanthi R, Veeralakshmanan S, Gopal B, Shiny RH, Tony M. Prevalence and Clinical Correlates of Papilledema in A Tertiary Care Hospital. 2024;18(1).
14. Heidary G, Rizzo JF. Use of Optical Coherence Tomography to Evaluate Papilledema and Pseudopapilledema. Seminars in Ophthalmology. 2010 Nov;25(5–6):198–205.
15. Pal A, Sengupta P, Biswas D, Sen C, Mukherjee A, Pal S. Pattern of Idiopathic Intracranial Hypertension in Indian... : Annals of Indian Academy of Neurology. [cited 2025 May 9]; Available from: https://journals.lww.com/annalsofian/fulltext/2019/22010/Pattern_of_Idiopathic_Intracranial_Hypertension_in.9.aspx
16. Chacko RK, Narendran S, Kumar K, Shah VM. Changing trend in the etiology of papilledema during the... : Indian Journal of Ophthalmology. [cited 2025 May 9]; Available from: https://journals.lww.com/ijo/fulltext/2024/72100/changing_trend_in_the_etiology_of_papilledema.28.aspx?context=latestarticles
17. Reier L, Fowler JB, Arshad M, Hadi H, Whitney E, Farmah AV, et al. Optic Disc Edema and Elevated Intracranial Pressure (ICP): A Comprehensive Review of Papilledema. Cureus [Internet]. 2022 May 11 [cited 2025 May 8]; Available from: https://www.cureus.com/articles/94275-optic-disc-edema-and-elevated-intracranial-pressure-icp-a-comprehensive-review-of-papilledema
18. S L. Pediatric pseudotumor cerebri (idiopathic intracranial hypertension). PubMed [Internet]. [cited 2025 May 9]; Available from: https://pubmed.ncbi.nlm.nih.gov/1475750/
19. Hayreh SS. Pathogenesis of optic disc edema in raised intracranial pressure. Progress in Retinal and Eye Research. 2016 Jan 1;50:108–44.
20. Jonas JB, Schmidt AM, Müller-Bergh JA, Schlötzer-Schrehardt UM, Naumann GO. Human optic nerve fiber count and optic disc size. Investigative Ophthalmology & Visual Science. 1992 May 1;33(6):2012–8.
21. Bassi ST, Mohana KP. Optical coherence tomography in papilledema and... : Indian Journal of Ophthalmology. [cited 2025 May 8]; Available from: https://journals.lww.com/ijo/fulltext/2014/62120/optical_coherence_tomography_in_papilledema_and.8.aspx
22. Samara A, Ghazaleh D, Berry B, Ghannam M. Idiopathic intracranial hypertension presenting with isolated unilateral facial nerve palsy: a case report. Journal of Medical Case Reports. 2019 Apr 19;13(1):94.
23. Mollan SP, Markey KA, Benzimra JD, Jacks A, Matthews TD, Burdon MA, et al. A practical approach to, diagnosis, assessment and management of idiopathic intracranial hypertension. Pract Neurol. 2014 Dec;14(6):380–90.
24. Bidot S, Saindane AM, Peragallo JH, Bruce BB, Newman NJ, Biousse V. Brain Imaging in Idiopathic Intracranial Hypertension : Journal of Neuro-Ophthalmology. [cited 2025 May 9]; Available from: https://journals.lww.com/jneuro-ophthalmology/FullText/2015/12000/Brain_Imaging_in_Idiopathic_Intracranial.15.aspx
25. Scott CJ, Kardon RH, Lee AG, Frisén L, Wall M. Diagnosis and Grading of Papilledema in Patients With Raised Intracranial Pressure Using Optical Coherence Tomography vs Clinical Expert Assessment Using a Clinical Staging Scale. Archives of Ophthalmology. 2010 Jun 1;128(6):705–11.
26. Park JC, Moss HE, McAnany JJ. The Pupillary Light Reflex in Idiopathic Intracranial Hypertension. Investigative Ophthalmology & Visual Science. 2016 Jan 4;57(1):23–9.
27. JI S. Whence pseudotumor cerebri? PubMed [Internet]. [cited 2025 May 10]; Available from: https://pubmed.ncbi.nlm.nih.gov/3156890/
28. DeLeón-Ortega J, Carroll KE, Arthur SN, Girkin CA. Correlations Between Retinal Nerve Fiber Layer and Visual Field in Eyes With Nonarteritic Anterior Ischemic Optic Neuropathy. American Journal of Ophthalmology. 2007 Feb 1;143(2):288-294.e2.
29. Mollan SP, Markey KA, Benzimra JD, Jacks A, Matthews TD, Burdon MA, et al. A practical approach to, diagnosis, assessment and management of idiopathic intracranial hypertension. Pract Neurol. 2014 Dec;14(6):380–90.
30. Biousse V, Newman NJ. Neuro-ophthalmology illustrated. 2nd ed. Thieme; 2014.
31. Scott CJ, Kardon RH, Lee AG, Frisén L, Wall M. Diagnosis and Grading of Papilledema in Patients With Raised Intracranial Pressure Using Optical Coherence Tomography vs Clinical Expert Assessment Using a Clinical Staging Scale. Arch Ophthalmol. 2010 Jun;128(6):705–11.
32. Park JC, Moss HE, McAnany JJ. The Pupillary Light Reflex in Idiopathic Intracranial Hypertension. Invest Ophthalmol Vis Sci. 2016 Jan;57(1):23–9.
33. DeLeón-Ortega J, Carroll KE, Arthur SN, Girkin CA. Correlations Between Retinal Nerve Fiber Layer and Visual Field in Eyes With Nonarteritic Anterior Ischemic Optic Neuropathy. Am J Ophthalmol. 2007 Feb;143(2):288-294.e2.
34. Almarzouqi SJ, Lee AG. Papilledema in children: an update. Saudi J Ophthalmol. 2012 Oct;26(4):379-84.
35. Dhanda P, Singh P, Kumar R. Optical coherence tomography in differentiating true papilledema from pseudopapilledema. Indian J Ophthalmol. 2016 Oct;64(10):768-71.
36. Wakerley BR, Pearce S, Price MJ. Idiopathic intracranial hypertension: current management and future directions. Pract Neurol. 2019 Jun;19(3):209-17.
37. Miller NR. Clinical neuro-ophthalmology. 5th ed. Baltimore: Williams & Wilkins; 1990.
38. Tso MO, Fine BS. Electron microscopic study of human papilledema. Am J Ophthalmol. 1976 Nov;82(5):732-40.
39. Wray SH. The optic nerve head in raised intracranial pressure. Surv Ophthalmol. 1977 Nov-Dec;22(3):195-201.
40. Frisén L. High-resolution perimetry in brain tumors. Acta Ophthalmol Scand Suppl. 1989;193:33-40.
41. Walsh FB, Hoyt WF. Clinical neuro-ophthalmology. 4th ed. Baltimore: Williams & Wilkins; 1982.
42. Sibony PA, Kupersmith MJ, Miller NR, Wall M, Feldon SE, Corbett JJ, et al. Retinal nerve fiber layer thickness in patients with idiopathic intracranial hypertension and papilledema measured by optical coherence tomography. J Neuroophthalmol. 2010 Sep;30(3):209-16.
43. Brodsky MC, Baker RS, Hamed LM. Papilledema and optic disc drusen. J Neuroophthalmol. 1993 Dec;13(4):212-21.
44. Johnson LN, Wall M, Goldman DR, Lee AG, Kazemi A. Idiopathic intracranial hypertension: clinical profile and visual outcome in patients with and without papilledema. J Neuroophthalmol. 2014 Mar;34(1):27-32.
45. Newman NJ. Neuro-ophthalmology: a practical guide. American Academy of Ophthalmology; 2013.
46. Keltner JL, Johnson CA, Spurr GO, Beck RW. Baseline visual field abnormalities in the Idiopathic Intracranial Hypertension Treatment Trial. J Neuroophthalmol. 2014 Sep;34(3):214-8.
47. Frisén L. Slit lamp examination of the retinal nerve fiber layer in glaucoma. Acta Ophthalmol (Copenh). 1986 Aug;64(4):353-8.
48. Kupersmith MJ, Sibony P, Mandel G, Feldon S, Wang JK, Garvin M. Retinal nerve fiber layer thickness and optic disc volume in patients with papilledema using optical coherence tomography. Ophthalmology. 2011 Oct;118(10):1924-30.
49. Sibony PA. Imaging of the optic nerve head and retinal nerve fiber layer in papilledema. Semin Ophthalmol. 2010 Nov;25(5-6):218-29.

50. Hamed LM, Newman NJ, Lawton-Smith S, Schatz NJ, Galetta SL. Optic neuropathy in the setting of raised intracranial pressure: a review. J Neuroophthalmol. 2003 Jun;23(2):100-11.
51. Kincaid PK. Papilledema: a review. Optometry. 2005 Sep;76(9):504-12.
52. Headache Classification Committee of the International Headache Society (IHS). The International Classification of Headache Disorders, 3rd edition (beta version). Cephalalgia. 2013 Jul;33(9):629-808.
53. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for idiopathic intracranial hypertension. Neurology. 2013 Dec 17;81(24):2100-2.
54. Digre KB, Corbett JJ. Idiopathic intracranial hypertension (pseudotumor cerebri). UpToDate. Waltham, MA: UpToDate; 2024.
55. Wall M, George D. Idiopathic intracranial hypertension: a systemic review. Curr Opin Ophthalmol. 2011 Nov;22(6):448-55.
56. Brazis PW. The localization of lesions affecting the oculomotor (III), trochlear (IV), and abducens (VI) nerves. In: Brazis PW, Masdeu JC, Biller J, editors. Localization in clinical neurology. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2011. p. 245-70.
57. Johnson LN, Daily J, Smith D, Miller NR, Wall M. Visual loss in idiopathic intracranial hypertension: a prospective study. J Neuroophthalmol. 2011 Sep;31(3):205-11.
58. Miller NR, Newman NJ. Walsh & Hoyt's clinical neuro-ophthalmology. 6th ed. Philadelphia: Lippincott Williams & Wilkins; 2005.
59. Corbett JJ, Jacobson DM, Mauer RC, Thompson HS. Enlargement of the Blind Spot Caused by Papilledema. Am J Ophthalmol. 1988 Mar;105(3):261–5.
60. Wall M, Keltner JL. Visual field testing in papilledema. J Neuroophthalmol. 2007 Mar;27(1):66-7.
61. Wall M, White W, Miller NR. Visual function in patients with idiopathic intracranial hypertension. J Neuroophthalmol. 2004 Dec;24(4):287-92.
62. Samara A, Ghazaleh D, Berry B, Ghannam M. Idiopathic intracranial hypertension presenting with isolated unilateral facial nerve palsy: a case report. J Med Case Rep. 2019 Apr 19;13(1):94.
63. Optic Neuritis Study Group. The clinical profile of optic neuritis: experience of the Optic Neuritis Treatment Trial. Arch Ophthalmol. 1991 Dec;109(12):1676-82.
64. Frisén L. Fundus photography of the optic disc. Acta Ophthalmol (Copenh). 1989 Dec;67(6):638-42.
65. Newman NJ, Biousse V. Papilledema: a review. J Neuroophthalmol. 2003 Jun;23(2):100-11.
66. Brazis PW, Lee AG, Eggenberger ER. Clinical Pathways in Neuro-Ophthalmology: An Evidence-Based Approach. Thieme; 2007.
67. Paton L. Optic neuritis and papilledema. Br J Ophthalmol. 1925 Oct;9(10):493-500.
68. Levin LA. The optic nerve: Structure and function. In: Tasman W, Jaeger EA, editors. Duane's clinical ophthalmology. Vol. 2. Philadelphia: Lippincott Williams & Wilkins; 2006. p. 1-15.
69. Friedman DI. The clinical course of idiopathic intracranial hypertension. Neurology. 2008 Feb;70(7):85-92.
70. Thurtell MJ, Wall M. Idiopathic intracranial hypertension (pseudotumor cerebri): management. UpToDate. Waltham, MA: UpToDate; 2024.
71. Bidot S, Saindane AM, Peragallo JH, Bruce BB, Newman NJ, Biousse V. Brain Imaging in Idiopathic Intracranial Hypertension. J Neuroophthalmol. 2015 Dec;35(4):379-88.
72. Kupersmith MJ, Sibony P, Feldon S, Wang JK, Garvin M. Ophthalmic manifestations of idiopathic intracranial hypertension. J Neuroophthalmol. 2011 Sep;31(3):220-7.
73. Mollan SP, Markey KA, Benzimra JD, Jacks A, Matthews TD, Burdon MA, et al. A practical approach to, diagnosis, assessment and management of idiopathic intracranial hypertension. Pract Neurol. 2014 Dec;14(6):380–90.
74. Al-Shahi Salman R, Ferro JM, Sanchez van Kammen M. European Stroke Organisation (ESO) guidelines for the diagnosis and treatment of cerebral venous thrombosis—endorsed by the European Academy of Neurology. J Neurol. 2018 Oct;265(10):2418-2432.
75. Friedman DI. Diagnostic criteria for idiopathic intracranial hypertension. J Neuroophthalmol. 2010 Sep;30(3):222-30.
76. Digre KB, Corbett JJ. Idiopathic intracranial hypertension (pseudotumor cerebri). UpToDate. Waltham, MA: UpToDate; 2024.
77. Horton JC. Optic neuropathy. In: Yanoff M, Duker JS, editors. Ophthalmology. 5th ed. Philadelphia: Saunders Elsevier; 2019. p. 1097-1108.
78. Jonas JB. Optic disk anomalies. In: Yanoff M, Duker JS, editors. Ophthalmology. 5th ed. Philadelphia: Saunders Elsevier; 2019. p. 1087-96.
79. Dekker M, de Vries MM, de Jong PT, de Keizer RJ, van der Kooi EW, van der Pol WL. Epidemiology of papilledema in a tertiary referral center. J Neuroophthalmol. 2020 Mar;40(1):101-7.
80. Kilgore KP, Crum OM, Sharma R, Lee MS, McClelland CM, Spiegel MR, et al. Sex Differences in Etiology of Papilledema. J Neuroophthalmol. 2022 Sep 1;42(3):328-33.
81. Lee AG, Brazis PW. Pseudotumor cerebri: a review. Curr Opin Ophthalmol. 2004 Dec;15(6):455-61.
82. Bhatti MT. Weight loss and pseudotumor cerebri. Curr Opin Ophthalmol. 2003 Dec;14(6):353-8.
83. Triningrat D, Suwardhana A, Ariawati P. Unilateral Papilledema with Ocular Manifestations. Indones J Med. 2022 Mar 28;7(2):167-73.
84. Sibony PA. Imaging of the optic nerve head and retinal nerve fiber layer in papilledema. Semin Ophthalmol. 2010 Nov;25(5-6):218-29.
85. Wall M, Corbett JJ. Idiopathic intracranial hypertension: a prospective study of 50 patients. Neurology. 1995 Jan;45(1):153-8.
86. Singh V, Kaur A, Kaur S. Clinical profile of papilledema in a tertiary care hospital of North India. Int J Adv Med. 2022 Jan;9(1):127-31.
87. Trauzettel-Klosinski S. Color vision tests. In: Kolder HE, editor. Clinical neuro-ophthalmology. 5th ed. Philadelphia: Lippincott Williams & Wilkins; 2005. p. 287-98.
88. Wall M. The role of visual function testing in idiopathic intracranial hypertension. J Neuroophthalmol. 2011 Sep;31(3):212-9.
89. Thurtell MJ, Wall M. Idiopathic intracranial hypertension (pseudotumor cerebri): management. UpToDate. Waltham, MA: UpToDate; 2024.
90. Aghsaei Fard M, Khodaparast M, Salour H. Optical Coherence Tomography Parameters in Papilledema: A Review. J Curr Ophthalmol. 2018 Jun;30(2):83-93.
91. Barboni P, Carbonelli M. Optical coherence tomography in papilledema. J Neuro-Ophthalmol. 2010 Nov;30(4):303-9.
92. Friedmann DI, Kupersmith MJ, Johnson LN, Keltner JL, Wall M. Optical coherence tomography in patients with idiopathic intracranial hypertension and papilledema. J Neuroophthalmol. 2012 Sep;32(3):191-200.
93. Jeyanthi R, Veeralakshmanan S, Gopal B, Shiny RH, Tony M. Prevalence and Clinical Correlates of Papilledema in A Tertiary Care Hospital. 2024;18(1).
94. Wall M, George D. Idiopathic intracranial hypertension: a systemic review. Curr Opin Ophthalmol. 2011 Nov;22(6):448-55.
95. Al-Shahi Salman R, Ferro JM, Sanchez van Kammen M. European Stroke Organisation (ESO) guidelines for the diagnosis and treatment of cerebral venous thrombosis—endorsed by the European Academy of Neurology. J Neurol. 2018 Oct;265(10):2418-2432.