ORIGINAL RESEARCH PAPER

Ophthalmology

# CLINICO AETIOLOGICAL STUDY AND DIAGNOSTIC ROLE OF NEUROIMAGING IN PAPILLOEDEMA

**KEY WORDS:** Papilloedema, Neuroimaging Idiopathic Intracranial Hypertension

**L. Ramachandra Murthy** Professor & HOD Ophthalmology, Malla Reddy Institute of Medical Sciences, Hyderabad 500055.

**Renu Shukla Dubey*** Associate Professor of Ophthalmology, Malla Reddy Institute of Medical Sciences, Hyderabad 500055. *Corresponding Author

**ABSTRACT**

**AIM**

1.To clinically evaluate patients presenting with papilloedema and to make possible aetiological diagnosis

2.To evaluate the role of neuroimaging in making aetiological diagnosis of papilloedema

**PURPOSE**

To clinically evaluate patients of papilloedema so as to make anatomical localization and also to make possible aetiological and pathological diagnosis of neurological lesion.Anatomical localization and aetiopathological diagnosis of the neurological lesion will help the attending Neurosurgeon in better management of the case.

**INTRODUCTION**

Papilloedema is the most common and tell tale evidence of raised intra cranial pressure which usually develops within hours to several days from onset othe disease.It can lead to progressive irreversible visual loss and secondary post neuritic optic atrophy in upto 30% of patients,if untreated and also is a potential life threatening condition. The most important diagnostic step in the evaluation of the patient with papilloedema is neuroimaging study either by CT or MRI as well as contrast enhanced CT venography or MR venography.

**MATERIALS AND METHODS**

150 consecutive cases of papilloedema attending the out patient clinic of Department of Ophthalmology malla reddy hospital, malla reddy institute of medical sciences or referred from other speciality clincs were evaluated and investigated thoroughly as a prospective , observational, non interventional hospital study from 1st January 2020 to 31st December 2021. All underwent complete Ophthalmological and general examination along with complimentary investigations and neurological, medical, otorrhinolaryngiological investigations and neuroradiological imaging either CT brain or MRI with MRV depending on the need and affordability of the patients.

**Inclusion Criteria**

1. All patients presented with papilloedema
2. All age groups
3. Both sexes
4. Willing to give consent to undergo investigations

**Exclusion Criteria**

1. Unconscious patients
2. Patients with known neurological disease
3. Malignant Hypertension

**RESULTS**

Results of neuroimaging had shown that nearly 80% of patients had Idiopathic Intracranial Hypertension and 9% had cerebral venous thrombosis and remaining 15% patients had space occupying lesions in the form of Meningioma, Acoustic Neuroma, Neurocysticercosis, Tuberculoma and Colloid cysts of Munro.

**CONCLUSION**

There is a role for Neuroimaging in the early diagnosis of intra cranial lesions in patients with papilloedema which helps in providing better prognosis and saves the life and vision of the patient by early intra cranial intervention.

## INTRODUCTION

Papilloedema is the most common presenting sign of raised intra cranial pressure which develops over a period of few hours to several days from the onset of disease[1, 2] . Papilledema often produces brief episodes of monocular or binocular visual loss, called transient visual obscurations (TVOs) [3,4] . TVOs are thought to occur due to transient ischemia of the swollen optic nerve head and not a sign of impending permanent visual loss[5] . Studies had shown that untreated papilledema can lead to progressive irreversible visual loss and secondary optic atrophy in up to 31% of patients [6] . Raised intracranial pressure (ICP) is a potentially life threatening condition, which can also lead to visual loss and blindness [7] . A relatively common cause of papilledema is idiopathic intracranial hypertension, which affects approximately 1 in 100,000 population and is more among females with a high body-mass index [8] .The first diagnostic step in the evaluation of a patient with papilledema is a neuroimaging study, either by CT or MRI, as well as contrast-enhanced CT venography or MR venography. Contrast-enhanced MR venography is more reliable than the standard flow-related MR venography, which is subject to signal loss unrelated to stenosis or occlusion of venous sinuses [9] . Evidence of an intracranial mass lesion or hydrocephalus should be sought. Imaging findings that are supportive (but not diagnostic) of a diagnosis of idiopathic intracranial hypertension include dilation of the optic nerve sheaths,sinuous intra orbital optic nerve, flattening of the posterior globe, and an empty (or partially empty) pituitary sella [10] . Diagnostic confusion with regard to the MR venogram is based on the fact that narrowing of the transverse venous sinuses is frequently found in patients with idiopathic intracranial hypertension. Demonstration of stenosis resolution after acute therapeutic lowering of intracranial pressure suggests that the stenosis may be a consequence of elevated intracranial pressure [9, 11] . However, the true relationship between increased intracranial pressure and transverse sinus stenosis remains unclear, as persistent stenosis has been demonstrated in some patients following reduction of intracranial pressure and resolution of symptoms [12] . Awareness of this diagnostic confusion with MR venography is essential to avoid unnecessary anticoagulation in idiopathic intracranial hypertension patients. If MRI and MR venography were found to be unremarkable, lumbar

puncture is usually performed keeping 3 goals in mind: (1) Cerebro spinal fluid (CSF) cytology and flow cytometry;(2) Opening pressure of CSF;(3)Determination of improvement of Head ache in 24 to 48 hours fallowing lumbar puncture. If imaging and cerebrospinal fluid contents are normal and elevated opening pressure (greater than 20 cm H20 in a thin patient, 25 cm H20 in an obese patient, or 28 cm H2O in a pediatric patient) is confirmed, a diagnosis of isolated elevated intracranial pressure is established [13] . If causative medications have been excluded, the patient may be diagnosed with idiopathic intracranial hypertension.

**AIM**

1.to clinically evaluate patients presenting with papilloedema and to make possible aetiological diagnosis
2.To evaluate the role of neuroimaging in making aetiological diagnosis of papilloedema

**MATERIALS & METHODS**

A hospital based prospective study was conducted in the ophthalmology department of Malla Reddy hospital, Malla Reddy Institute of Medical sciences for a period of two years between January 2020 and December 2021. A total of 150 patients with clinically diagnosed papilledema presented to our Ophthalmic clinic OPD or refered to us from other speciality clinics, were included in our study.

**Inclusion Criteria**

1. All patients presented with papilloedema
2. All age groups
3. Both sexes
4. Willing To Give Consent To Undergo Investigations

**Exclusion Criteria**

1. Unconscious patients
2. Patients with known neurological disease
3. Malignant Hypertension

The patients particulars like name, age, sex, address were documented in a proforma specially designed for this study and was filled by the examining doctor. A detailed history of each and every symptom of the patient was taken such as onset, duration, progression, associated factors aggravating and relieving factors were documented. The patients were also enquired about the past medical and surgical history, systemic illness, treatment history, personal history and family history. A detailed and complete Ophthalmological examination including a thourough fundus evaluation, papillary reflexes, ocular movements, colour vision and field examination performed. Later on a complete neurological evaluation was done on every patient including general consciousness, cranial nerve examination, motor system and sensory system evaluation. Neuro imaging was done in all patients either CT brain or MRI with MRV depending on the need and affordability of patients. During the follow up visits, visual acuity, pupillary reaction, colour vision, central fields and fundus examination was done at one month and later at three months. All data were entered and analysed using SPSS version 21.

**RESULTS**

The age and gender wise distribution of the study population was shown in table 1. Majority of the study subjects were females (77.5%) and most of them were in the age group between 20- 30 years with a mean age of 31.37 years. The most common presenting complaint among the study subjects was headache (95%) followed by transient visual obscuration (66.7%), field defects was present in 16.7% of the study subjects (table 2). 15% of the patients had cranial nerve abnormality and the most common cranial nerve involved was the 7th nerve, whereas the other cranial nerve which were involved were 3 rd, 4th, 6th and 8th (table 3). The best corrected visual acquity for majority of the patients was between 6/6 – 6/12 and for nearly 10% of the eyes had BCVA in the range of 6/12 – 6/60 and for 3% it was ,<6/60 ( table 4).

For about 60% of the patients the fundus findings had shown a established papilledema and 37% of the eyes had features of early papilledema and 3 of the eyes had features of chronic papilledema and secondary optic atrophy (table 5). Among the 150 patients only 10 patients were advised CT and in that 7 patients had shown thickening of optic nerve sheath complex and the remaining 3 patients had findings of partial empty sella. The remaining 140 patients were advised MRI and their findings were tabulated in table 6 and all these patients had also undergone MRV and in that 68% of the patients had a normal picture and the remaining 32% had certain pathologies reported among which CVT (cerebral venous thrombosis) was found to be more common followed by hypoplasia of left transverse sinus (table 7). The results of neuroimaging had shown that 80% of the patients had idiopathic intracranial hypertension and 9% had cerebral venous thrombosis and the remaining 11% had space occupying lesions in the form of meningioma, acoustic neuroma, astrocytoma,glioma,neurocysticercosis,colloid cyst of Munro and tuberculoma (table 8). In the management aspect all the 16 patients who had space occupying lesions were referred to neurosurgery department and for other patients with IIH and CVT drugs in the form of Diamox, heparin and acitrom was given and all patients with IIH weight reduction measures were advised and during the follow-up period 2 patients underwent lumbar puncture and optic nerve sheath decompression as a mode of treatment. All the patients were followed up for 2 months except the patients who were referred to neurosurgery department and in that 95% of the patients had shown resolving signs of papilledema and 5% had signs of secondary optic atrophy.

**Table 1 : Age And Sex Wise Distribution Of The Study Population.**

| Age group | Gender | |
|---|---|---|
| | Male | Female |
| <20 | 2 (5.8%) | 4 (3.4%) |
| 20 - 30 | 16 (47.05%) | 60 (51.7%) |
| 31 - 40 | 10 (29.4%) | 38 (32.4%) |
| 41 - 50 | 6 (17.6%) | 14 (12%) |
| Total | 34 (100%) | 116(100%) |

**Table 2 : Distribution Of The Study Subjects On The Basis Of Their Presenting Complaints.**

| Complaints | Frequency | Percentage |
|---|---|---|
| Head Ache | 142 | 94.0 |
| Transient obscurations of vision | 100 | 66.0 |
| Field defects | 24 | 15.8 |
| Vomiting | 75 | 49.5 |
| Diplopia | 6 | 3.96 |
| Neck pain | 13 | 8.58 |
| Giddiness | 39 | 25.74 |
| Total | 150 | 100 |

**Table 3 : Distribution Of The Study Population On Their Cranial Nerve Abnormality.**

| Cranial nerve abnormality | Frequency | Percentage |
|---|---|---|
| 3rd Cranial nerve | 7 | 4.6 |
| 4th Cranialnerve | 2 | .66 |
| 6th Cranial nerve | 6 | 2.64 |
| 7th Cranial nerve | 8 | 5.33 |
| 8th Cranial nerve | 2 | 1.32 |
| Nil | 125 | 83 |
| Total | 150 | 100 |

**Table 4 : Distribution Of Study Population As Per BCVA.**

| BCVA | Right eye | Left eye |
|---|---|---|
| <6/60 | 5 (3.3%) | 3 (2%) |
| 6/12 – 6/60 | 15 (9.9%) | 9.2 (9.24%) |
| 6/6 -6/12 | 130 (85.8%) | 133 (87.78%) |

| | | |
|---|---|---|
| Total | 150 (100) | 150 (100) |

**Table 5 : Distribution Of Study Subjects Based On Fundus Examination.**

| Fundus finding | Right eye | Left eye |
|---|---|---|
| Early papilloedema | 58 (38.2%) | 52 (34.32%) |
| Established papilloedema | 88 (58%) | 95 (62.5%) |
| Chronic papilloedema | 2 (1.32%) | 1 (0.82%) |
| Secondary optic atrophy | 2 (1.32%) | 1 (0.82%) |
| Total | 150(100) | 150(100) |

**Table 6 : Distribution Of Of Study Population Based On MRI Findings Among The Study Population.**

| MRI findings | Frequency | Percentage |
|---|---|---|
| Thickening of optic nerve sheath complex | 75 | 53% |
| Partial empty sella | 35 | 25% |
| Space occupying lesion | 16 | 11% |
| Cerebral venous thrombosis | 14 | 10% |
| Total | 140 | 100 |

**Table 7 : Distribution Of The Study Population Based On MRV Findings Among Study Population**

| MRV findings | Frequency | Percentage |
|---|---|---|
| Cerebral venous thrombosis | 14 | 10 |
| Hypoplasia of left sigmoid sinus HLSS | 1 | 1 |
| Hypoplasia of left transverse sinus HLTS | 12 | 9 |
| HLTS ,HLSS | 3 | 2 |
| Hypoplasia of right transverse sinus | 1 | 1 |
| Left transverse sinus stenosis ,Left sigmoid sinus stenosis | 6 | 4 |
| Right transverse sinus stenosis, right sigmoid sinus stenosis | 8 | 6 |
| Normal | 95 | 68 |
| Total | 140 | 100 |

**Table 8 : Distribution Of Study Population According To The Results Of Neuroimaging.**

| Findings | | Frequency | | Percentage |
|---|---|---|---|---|
| Idiopathic Intracranial Hypertension (IIH) | | 120 | | 80 |
| Cerebral venous thrombosis | | 14 | | 9.24 |
| Space ocupying lesions (SOL) | Meningioma | 2 | 16 | 11 |
| | Acoustic neuroma | 2 | | |
| | Astrocytoma | 2 | | |
| | Medulloblastoma | 1 | | |
| | Glioma | 3 | | |
| | Tuberculoma | 3 | | |
| | Neurocysticercosis | 2 | | |
| | Colloid cyst of Munro | 1 | | |
| Total | | 150 | | 100 |

## DISCUSSION

Papilledema can present with varied clinical symptoms, neuro-ophthalmic features and radiological features. It is a disorder of elevated cerebrospinal fluid pressure of various causes. In the present study we found majority of the study subjects were females and were in the age group between 25 and 35 years. A study by Lee et al. [14] was almost in par with the current study. The present study had shown that 80% of the patients with papilledema were due to idiopathic intracranial hypertension and their mean age of presentation was 31.3 years, a similar kind of results was shown in a study done by Ambika S et al[15] and another study done in North America they found the most common age of presentation was between 29 and 30 years [16] . A study done by John chen and Michael Wall had quoted that female gender is a risk factor for IIH since almost 90% of the affected population were obese females[17] . The most common symptoms of IIH include headache, transient obscuration of vision, pulsatile tinnitus, and diplopia. A study done by Timoteo et al. had reported that headache as the sole presentation of cerebral venous thrombosis[18] . Unless there are external constraints (weight limitations, availability), MRI with MRV is currently the study of choice [19] . In the present study only 10 patients were subjected to CT imaging and the remaining 140 patients had underwent MRI and MRV. Using a special technique, three dimensional, gadolinium enhanced MRV appears to be more sensitive than conventional MRV for detecting areas of subtle cerebral venous stenosis. In our study it was found that 69.2% were overweigh and 11% were obese among the patients with IIH and it was supported by the study done by Daniels AB et al. where he had reported that higher BMIs were associated with greater risk of IIH [20] and a other study done by Szewka etal had shown that higher BMI at diagnosis is associated with increased severe visual loss in patients with IIH [21] . A study done by Michael wall reported that horizontal diplopia with sixth nerve paresis were found in 10-20% of cases, which was contradicting to the results of the present study where we found 7th nerve paresis to be more common in our patients. Central fields were normal in 208 eyes (86.6%) defective in 32 eyes (13.3%) of our study population. In this 18 eyes (56.2%) had enlarged blind spots remaining patients had bitemporal hemianopia, homonymous hemianopia, superior and inferior quadrantonopia. Raju K V et al. reported in their study that most of the field defects of neuro-ophthalmic significance are located in central 30 degree field and 56% of the patient in their study showed field defect with enlargement of blind spot which was in par with the present study [22] . Out of 112 patients who underwent MRV isolated thrombosis of superior sagittal sinus involvement was seen in 4 patients, isolated transverse sinus was involved in 2 patients and multiple sinus involvement (superior sagittal sinus, transverse sinus and sigmoid sinus) were seen in 5 patients suggestive of cerebral venous thrombosis in these 11 patients (9.1%), of the total study patients presented with papilledema. Remaining 25 patients had either congenital hypoplasia or stenosis of transverse or sigmoid sinus or both and 76 patients showed normal MRV findings. Our findings are well supported by the study done by AI Hashel JY, in his prospective study of cerebral venous thrombosis he found that superior sagittal sinus thrombosis (54.5%) occur most commonly than transverse sinus thrombosis (52%) [23] , and a retrospective study done by Brodsky MC and Vaphiades M on the patients with pseudotumour cerebri had reported almost similar findings in the MRI [24] . Raju K.V et al. in their study on patients with space occupying lesions reported that 56% of the patients presented had papilledema [23] . Also he mentioned that posterior fossa tumours presented with papilledema earlier but cortical and pituitary tumours present with late papilledema. Miller in his book had mentioned that the etiology is brain tumors in 71% of patients who had presented with bilateral papilledema [25] . Ridha et al. reported that findings suggestive of raised intracranial tension in a patient with IIH like presentation should prompt a careful evaluation of cerebral venous sinus with MR venogram which will be missed with MRI alone [26].

## CONCLUSION

Ocular features are considered as the portal to the brain from which neurological disorders can be diagnosed. Early detection of intracranial space occupying lesions and cerebral venous thrombosis could be possible through ocular examination and it should be confirmed by imaging techniques. Thus neuroimaging helps in early diagnosis of several intracranial lesions in patients with papilledema, which provides better prognosis and saves the life of the patient by early intervention.

## REFERENCES

1. Beri S, Gosalakkal JA, Hussain N, Balky AP, Parepalli S. Idiopathic intracranial hypertension without papilledema. Pediatr Neurol doi:10.1016/j.pediatrneurol,2010;42:56-58.
2. Nazir S, O'Brien M, Qureshi NH, Slape L, Green TJ, Phillips PH et al. Sensitivity

of papilledema as a sign of shunt failure in children. JAAPOS doi:10.1016/j.jaapos,2009;13:63-66.
3. Wall M, George D. Idiopathic intracranial hypertension. A prospective study of 50 patients. Brain. 1991;114(1A):155-80.
4. Corbett JJ. The first Jacobson Lecture. Familial idiopathic intracranial hypertension. J Neuroophthalmol. doi: 10.1097/WNO.0b013e31818f12a2, 2008;28(4):337-47.
5. O'Duffy D, James B, Elston J. Idiopathic intracranial hypertension presenting with gaze-evoked amaurosis. Acta Ophthalmol Scand. 1998;76(1):119-20.
6. Rowe FJ, Sarkies NJ. Assessment of visual function in idiopathic intracranial hypertension: a prospective study. Eye Lond. 1998;12(1):111-8.
7. Cameron AJ. Marked papilloedema in pulmonary emphysema. Br J Ophthalmol. 1933;17(3):167-9.
8. Binder DK, Horton JC, Lawton MT, McDermott MW. Idiopathic intracranial hypertension. Neurosurgery. 2004;54(3):538-51.
9. Farb RI, Vanek I, Scott JN et al. Idiopathic intracranial hypertension: the prevalence and morphology of sinovenous stenosis. Neurology, 2003; 60:1418-24.
10. Brodsky MC, Vaphiades M. Magnetic resonance imaging in pseudotumor cerebri. Ophthalmology. 1998;105(9):1686-93.
11. Higgins JN, Pickard JD. Lateral sinus stenosis in idiopathic intracranial hypertension resolving after CSF diversion. Neurology, 2004;62:1907-8.
12. Bono F, Giliberto C, Mastrandrea C et al. Transverse sinus stenoses persist after normalization of the CSF pressure in IIH. Neurology, 2005;65:1090-3.
13. Avery RA, Licht DJ, Shah SS et al. CSF opening pressure in children with optic nerve head edema. Neurology, 2011;76:1658-61.
14. Lee A, Rigi M, Almarzouqi S, Morgan M. Papilledema: epidemiology, etiology, and clinical management. Eye and Brain, 2015, 47.
15. Ambika S, Deepak Arjundas, Veena Noronha, Anshuman. Clinical profile, evaluation, management and visual outcome of idiopathic intracranial hypertension in a neuro-ophthalmology clinic of a tertiary referral ophthalmic center in India. Ann Indian Acad Neurol, 2010;13(1).
16. Fraser C, Plant GT. The syndrome of pseudotumour cerebri and idiopathic intracranial hypertension: Current Opinion in Neurology. 2011;24(1):12–7.
17. Chen J, Wall M. Epidemiology and Risk Factors for Idiopathic Intracranial Hypertension. Int Ophthalmol Clin Internet. 2014-2016;54(1).
18. Timóteo Â, Inácio N, Machado S, Pinto AA, Parreira E. Headache as the sole presentation of cerebral venous thrombosis: a prospective study. J Headache Pain. 2012;13(6):487-90.
19. Agid R, Farb RI. Neuroimaging in the diagnosis of idiopathic intracranial hypertension. Minerva Med, 2006;97:365-70.
20. Daniels AB, Liu GT, Volpe NJ et al. Profiles of obesity, weight gain, and quality of life in idiopathic intracranial hypertension pseudotumor cerebri Am J Ophthalmol. 2007;143(4):635-641.
21. Szewka AJ, Bruce BB, Newman NJ, Biousse V. Idiopathic intracranial hypertension: relation between obesity and visual outcomes. J Neuro ophthalmol. 2013;33(1):4-8.
22. KV Raju, Anju Abdul Khader. Ocular Manifestations of Intracranial Space Occupying Lesions – A Clinical Study. Kerala Journal of Ophthalmology. 2009; XXI(3).
23. Al-Hashel JY, John JK, Vembu P. Venous thrombosis of the brain. Retrospective review of 110 patients in Kuwait. Neurosciences Riyadh. 2014;19(2):111-7.
24. Brodsky MC, Vaphiades M. Magnetic resonance imaging in pseudotumor cerebri. Ophthalmology. 1998;105(9):1686-93.
25. Miller NR, Walsh FB, Hoyt WF. Walsh and Hoyt's Clinical Neuro-ophthalmology. Lippincott Williams & Wilkins, 2005, 1404. 3
26. Ridha MA, Saindane AM, Bruce BB, Riggeal BD, Kelly LP, Newman NJ et al. MRI findings of elevated intracranial pressure in cerebral venous thrombosis versus idiopathic intracranial hypertension with transverse sinus stenosis. Neuro ophthalmology. 2013;37(1):1-6