International Journal Dental and Medical Sciences Research
Volume 5, Issue 1, Jan-Feb 2023 pp 245-249 www.ijdmsrjournal.com ISSN: 2582-6018

Clinical Profile of Idiopathic Intracranial Hypertension in a Tertiary Eye Care Centre in South India

Chitra Govindan
Assistant Professor, Department of Ophthalmology, Government Dharmapuri Medical College and Hospital, Dharmapuri.

Submitted: 12-01-2023 Accepted: 24-01-2023

ABSTRACT:
BACKGROUND: Idiopathic intracranial hypertension(IIH) is a syndrome characterised by elevated intracranial pressure without ventriculomegaly or mass lesion, and with normal cerebrospinal fluid composition that usually occurs in obese women of child-bearing age. The aim was to study the behaviour and natural course of IIH, its associated neuroimaging features and to evaluate the response to treatment and visual outcome.
METHODS: A prospective study of a series of 61 patients with clinically and radiologically proven diagnosis of IIH were included in our study. The mean age of presentation was 29.97 years and obese females of child-bearing age were more commonly affected. Anaemia was a significant risk factor in our study population. Most of the patients had visual acuity of 6/6. Most common false localising sign was sixth nerve paresis. Most of the patients presented with established papilledema. Most common visual field defect was an enlarged blind spot. In MRI and CT brain, thickening of optic nerve sheath and empty sella were the most common findings in our patients. In MRV study, 30.2%(16) patients had congenital hypoplasia or stenosis of transverse sinus or sigmoid sinus or both. Patients were treated with acetazolamide, iron supplements, lumbar puncture and optic nerve sheath decompression depending upon the stage and severity of presentation. Follow up at one month and at three months showed resolving papilledema following treatment in most cases.
CONCLUSION: IIH is a neurological disorder common in obese women of childbearing age presenting with varied clinical features. Neuroimaging shows hypoplasia or stenosis of transverse venous sinuses in a large number of IIH patients. Whether these venous abnormalities are cause or consequence of increased intracranial pressure is still under debate.

KEYWORDS: Idiopathic intracranial hypertension, obesity, papilledema, transverse sinus stenosis.

I. INTRODUCTION

Idiopathic intracranial hypertension is a syndrome characterised by elevated intracranial pressure without ventriculomegaly or mass lesion, andwith normal cerebrospinal fluid composition that usually occurs in obese women of child-bearing age. It is a disorder of elevated CSF pressure. It is often referred to as "Pseudotumour cerebri"

The underlying cause is reduced absorption of cerebrospinal fluid. The ventricular system is normal without any deformity and obstruction. The neuroimaging is normal except for raised CSF pressure. The CSF pressure is above 200mm of water in the non-obese and above 250mm of water in the obese.

The peak incidence is in the 3rd decade of life, especially obese females in the reproductive age group. The most common presenting symptoms of raised intracranial pressure are headache, pulsatile tinnitus, transient obscuration of vision, pain behind the eyes, double vision and loss of vision. Signs are diplopia due to abducent nerve paresis and papilledema resulting in loss of sensory visual function. Most Common visual field defect is an enlarged blind spot.This satisfies the modified Dandy criteria for IIH. Neuroimaging signs include empty sella syndrome, lateral or transverse sinus stenosis, flattening of globe and unfolding of optic nerve sheaths.

CRITERIA FOR DIAGNOSIS (MODIFIED DANDY CRITERIA)

1. "Signs and symptoms of increased intracranial pressure headaches, nausea, vomiting, transient obscurations of vision,

papilledema

2. No localising, focal neurologic signs, except unilateral or bilateral sixth nerve paresis.
3. Cerebrospinal fluid opening pressure ≥ 25 cm, but without cytologic or chemical abnormalities.
4. Normal neuroimaging results adequate to exclude cerebral venous thrombosis, i.e., magnetic resonance imaging of the

brain, often with additional sequences (computed tomography or magnetic resonance

International Journal Dental and Medical Sciences Research
Volume 5, Issue 1, Jan-Feb 2023 pp 245-249 www.ijdmsrjournal.com ISSN: 2582-6018

venography)

The purpose of the study was
1. To study the clinical profile of patients with idiopathic intracranial hypertension.
2. To study the behaviour and the natural course of idiopathic intracranial hypertension.
3. To study the neuroimaging features and to look for hypoplasia and stenoses of transverse and sigmoid sinuses.
4.To analyse the response to treatment and study the visual outcome.

II. MATERIALS AND METHODS

A prospective, hospital based study was carried out in our hospital in the Department of Ophthalmology from December 2021 to May 2022 (6 months). 61 Patients who were proven to have idiopathic intracranial hypertension clinically and radiologically were enrolled for the study.

Inclusion criteria
It includes all patients with any of the following symptoms and signs with radiologically proven idiopathic intracranial hypertension

- Signs and symptoms of idiopathic intracranial hypertension such as headache, transient obscuration of vision, defective vision, vomiting, neck pain, giddiness, field defects and diplopia.
- No localising focal neurological signs except unilateral or bilateral sixth nerve paresis.
- Normal neuroimaging results adequate to exclude cerebral venous thrombosis and intracranial tumours i.e magnetic resonance imaging of the brain, often with additional sequences (computed tomography or magnetic resonance venography)
- Reproducible visual field defects

Exclusion criteria
1.Abnormalities on neurological examination aside from papilledema and its related visual loss or sixth nerve paresis.
2.Abnormal neuroimaging except for an empty sella, thickening of optic nerve sheath complex, stenoses of transverse and sigmoid sinuses and widening of peri-optic subarachnoid space

Clinical evaluation

A series of 61 patients who presented to our Ophthalmology department with clinically and radiologically proven diagnosis of idiopathic intracranial hypertension were included in our study. All these patients underwent a thorough ophthalmological and neurological evaluation.

A detailed history of each and every symptom of the patient was taken such as the onset, duration, progression, associated factors, aggravating an 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.

1. Visual acuity by Snellen's chart
2. Refraction
3. Pupillary reaction for normal pupil, sluggish pupil or RAPD.
4. General ophthalmic examination with torch light and slit lamp biomicroscopy
5. Intraocular pressure measurement by non-contact tonometry
6. Fundus examination by direct ophthalmoscope and slit lamp biomicroscopy using 90 Dioptre lens and indirect ophthalmoscopy.
7. Extraocular movement examination using torch light
8. Colour vision evaluated by Pseudo-isochromatic Ishiharas chart
9. Central fields by Bjerrums screen
10. Visual fields by Automated perimetry (HFA 30-2)
11. A complete neurological evaluation was done to every patient including general consciousness, cranial nerve examination, motor system evaluation and sensory system evaluation were done.
12. Neuroimaging was done in all patients either CT Brain or MRI/MRV with or without contrast depending upon the need and affordability of the individual patients.
13. During follow up visual acuity, pupil reaction, colour vision, fields and fundus examination were done at one month and three months.
14. Vitals such as pulse rate and blood pressure were recorded
15. Weight, height and body mass index were done
16. Systemic examination of cardiovascular, respiratory, central nervous system were done.
17. Investigations such as haemoglobin percentage, random blood sugar, lipid profile and thyroid profile were done.
18. Fundus photographs were taken for every patient
19. During follow up weight, visual acuity, pupillary reaction, colour vision, central fields and fundus was done at one month and three months.

III. DISCUSSION

In our prospective study, we included 61 patients. The mean age group is 29.97 years which

International Journal Dental and Medical Sciences Research
Volume 5, Issue 1, Jan-Feb 2023 pp 245-249 www.ijdmsrjournal.com ISSN: 2582-6018

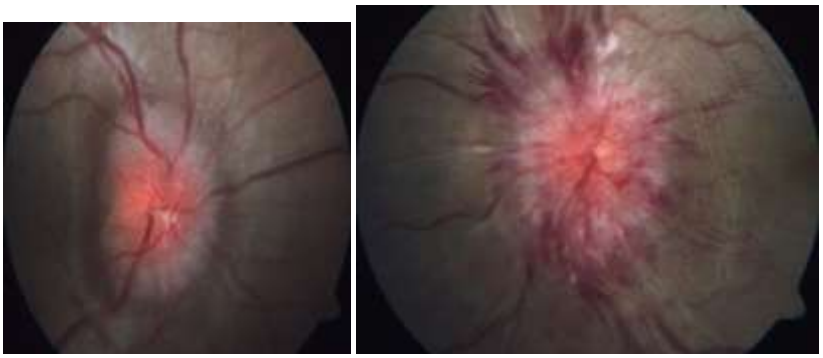
ranges between 16 years to 49 years. Out of 61 patients 56(91.8%) were females and 5(8.2%) were males. The most common complaints in our study patients were headache(88.5%), transient obscuration of vision(41.0%), vomiting(37.7%), defective vision(29.5%), neck pain(22.9%), diplopia(14.8%), giddiness(8.2%) and field defect(1.6%). Among our study patients, 9 patients (14.7%) had systemic illness history such diabetes, hypertension, dyslipidemia, pregnancy induced hypertension and polycystic ovarian disease. Remaining 52 patients (85.3%) had no systemic illness**.** In our study, 2 patients gave history of treatment with oral contraceptive pills for menstrual irregularities, 1 patient took treatment for acne with oral doxycycline and vitamin A capsules and 3 patients had treatment history with thyroxine for hypothyroidism.

The body mass index was calculated as weight in kilograms divided by height in meters squared in our study and 47.5% were overweight, 23% were healthy, 18% were obese and 11.5% were severely obese. On presentation, Best corrected visual acuity was found to range from 6/6 to Hand movements in our study population. Out of 122 eyes, 121 eyes had visual acuity between 6/6 to 6/60 and one eye had visual acuity of hand movements on presentation.

Pupillary examination was normal in 104 eyes(85.2%),sluggish in 16 eyes(13.1%) and Relative Afferent Pupillary defect in 2 eyes(1.6%).Extraocular movements were normal in 92.6% and abnormal in 7.4%.Most common involvement in our study group was the sixth nerve. Horizontal diplopia was reported due to sixth nerve paresis which is a false localising sign in IIH.

Fundus examination of our patients showed early papilledema in 52 eyes(42.6%), established papilledema in 66 eyes(54.1%) and chronic papilledema in 4 eyes(3.3%). One patient progressed to optic atrophy as a result of chronic papilledema. Papilledema was bilateral in 59 patients but asymmetric in 2 patients with one eye early papilledema and one eye established papilledema.

EARLY PAPILLEDEMA **ESTABLISHED PAPILLEDEMA**



Colour vision was defective in 7 eyes(5.7%) and normal in 115 eyes(94.3%)Central fields were normal in 104 patients(85.2%) and defective in 18 patients(14.8%) in our study population. Most common defect was an enlarged blind spot in the affected patients. Fields by Automated perimetry were normal in 30 patients(49.2%) and abnormal in 31 patients(50.8%). Most common defects were enlarged blind spot and generalized constriction of fields.

Blood investigations such as haemoglobin, random blood sugar, fasting lipid profile and fasting thyroid profile were done for all the patients. Haemoglobin values were normal in 34 patients(55.7%) and below normal in 27 patients(44.3%). Random blood sugar values were normal in all the patients(100%). Lipid profile was normal in 56 patients(93.3%) and abnormal in 4 patients(6.7%). Thyroid profile was normal in 55 patients(90.2%) and altered in 6 patients(9.8%).Our study highlights the possible association between iron deficiency anemia and IIH evidenced by resolution of papilledema after correction of iron deficiency.

In neuroimaging, 38 Patients showed both thickening of optic nerve sheath with widening of perioptic subarachnoid space and partial empty sella. 23 patients showed only thickening of optic nerve sheath with widening of perioptic subarachnoid space alone. Out of the 53 patients who underwent MRI and MRV, 23 patients had either congenital hypoplasia or stenosis of transverse sinus or sigmoid sinus or both. In the other 30 patients, MRV was normal without stenoses or hypoplasia. Out of our 23 patients, 16

(30.2%) had hypoplasia of left transverse sinus stenosis and hypoplasia of left sigmoid sinus stenosis, 2 patients had hypoplasia of both left transverse and left sigmoid sinus stenoses with right transverse sinus and right sigmoid sinus stenoses, 1 patient had bilateral transverse sinus stenoses, 1 patient had hypoplasia of both right transverse and right sigmoid sinuses with left transverse sinus stenosis, 1 patient had left transverse sinus stenosis alone, 1 patient had right transverse sinus stenosis alone and 1 patient had both left transverse and sigmoid sinus stenoses.

Out of the 61 patients, 24 with high BMI were treated with Acetozolamide 250mg BD and were advised Weight reduction, 15 patients who had coexisting Anaemia and with high BMI were treated with acetazolamide tablets and iron supplements along with Weight reduction, 13 whose BMI was normal were treated with acetazolamide alone, 1 patient underwent lumbar puncture for both diagnostic and therapeutic purposes, one patient was initially treated with intravenous Mannitol and maintained with acetazolamide in the subsequent follow ups and 2 patients underwent surgical decompression by unilateral Optic Nerve Sheath Decompression. Two patients developed intolerance to acetazolamide and were treated with Topiramate tablets. Weight reduction should be recommended for all obese and overweight IIH patients. Weight control improves the overall quality of life.

IV. CONCLUSION

Idiopathic intracranial hypertension is characterized by elevated CSF pressure without apparent cause. Obese women of child-bearing age are more commonly involved than males. Most common complaint was headache in our study patients. Most of our patients presented within one month duration. Commonly associated risk factors are thyroid dysfunction, anemia, pregnancy, hypertension, exposure to exogenous drugs like tetracyclines, oral contraceptives, vitamin A supplements and thyroid replacement therapy. Anemia was a significant risk factor in our study population. High body mass index and recent weight gain is associated with IIH. Visual acuity was 6/6 in most eyes in our study. Sixth nerve paresis occurs as a false localizing sign in IIH. Most of the patients presented with established papilledema. Most common visual field defect is an enlarged blind spot in both Central fields test by Bjerrums Screen and Automated perimetry. In MRI and CT brain, thickening of optic nerve sheath and empty sella were the most common findings in our patients. In MRV study, 30.2%(16) patients had hypoplasia of left transverse sinus stenosis and hypoplasia of left sigmoid sinus stenosis. Patients who had high BMI were treated with Acetazolamide 250mg BD with Weight reduction. Patients who had coexisting Anaemia were treated with iron supplements along with acetazolamide. One patient was treated by lumbar puncture and two patients were treated with unilateral Optic Nerve Sheath Fenestration. Follow up at one month and at three months showed resolving papilledema in most cases.

REFERENCES

[1]. Ambika S, Arjundas D, Noronha V, Anshuman null. 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 Jan;13(1):37–41.

[2]. Friedman. I. Deborah, Papilledema In: Walsh and Hoyt's clinical Neuroophthalmology. Vol. 1. 6th ed. Philadelphia: Lippincott Williams and Wilkins; 2005. p. 237 to 291

[3]. Szewka AJ, Bruce BB, Newman NJ, Biousse V. Idiopathic intracranial hypertension: relation between obesity and visual outcomes. J Neuroophthalmol. 2013 Mar;33(1):4–8.

[4]. Jack.J.Kanski, Clinical Ophthalmology A systematic Approach. 7th ed.London: Elsevier Saunders; 2011. p. 334 – 338 , 801- 802

[5]. Riggeal BD, Bruce BB, Saindane AM, Ridha MA, Kelly LP, Newman NJ, BiousseV.Clinical course of idiopathic intracranial hypertension with transverse sinus stenosis.Neurology. 2013 Jan 15;80(3):289-95

[6]. Kaul B, Ramanarayanan S, R S, Mahapatra H, Sethi TK, Ahlawat R. Iron deficiency masquerading as idiopathic intracranial hypertension. BMJ Case Rep. 2009. doi: 10.1136/bcr.06.2008.0346. Epub 2009 Mar 17.

[7]. Woodall MN, Nguyen KD, Alleyne CH, Macomson SD. Bilateral transverse sinus stenosis causing intracranial hypertension. BMJ Case Rep. 2013;2013.

[8]. Dr.Mahesh Kumar. S, Neuro Ophthalmology. 4th ed. Madurai: Aravind Eye Hospital; 2007. p. 113 – 121, 122 – 127

[9]. Ibrahim YA, Mironov O, Deif, Mangla R, Almast J. Idiopathic Intracranial Hypertension: Diagnostic Accuracy of the Transverse Dural Venous Sinus Attenuation on CT Scans. Neuro radiol J December 2014 volume 27 665-670.

[10]. Dave SB, Subramanian PS. Pseudotumorcerebri: an update on treatment options. Indian J Opthalmol. 2014 Oct;62(10):996-8.