ANALYSIS OF CLINICAL PROFILE, LABORATORY INVESTIGATIONS AND MANAGEMENT MODALITIES OF PATIENTS WITH IDIOPATHIC INTRACRANIAL HYPERTENSION

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CERTIFICATE

This is to certify that the dissertation entitled **"ANALYSIS OF CLINICAL PROFILE, LABORATORY INVESTIGATIONS AND MANAGEMENT MODALITIES OF PATIENTS WITH IDIOPATHIC INTRACRANIAL HYPERTENSION" is a bonafide record of work done by DR.J. SENTHIL NATHAN in** the Institute of Neurology, Rajiv Gandhi Government General Hospital & **MADRAS MEDICAL COLLEGE, CHENNAI in partial fulfillment of the** Tamilnadu Dr.MGR Medical **University rules and regulations for the award of D.M. (NEUROLOGY) degree under my direct guidance and supervision during the academic year 2011-2014.**

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DECLARATION

I solemnly declare that this dissertation titled **"ANALYSIS OF CLINICAL PROFILE, LABORATORY INVESTIGATIONS AND MANAGEMENT MODALITIES OF PATIENTS WITH IDIOPATHIC INTRACRANIAL HYPERTENSION"** is done by me in the Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and supervision of **Prof.Dr.Balasubramanian. S M.D., D.M..,** Professor of Neurology, Institute of Neurology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfillment of the university requirements for the award of the degree of D.M. Neurology.

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CONTENTS

INTRODUCTION

Idiopathic Intracranial Hypertension (IIH) is defined as a clinical syndrome characterized by elevated intracranial pressure, in the absence of an identifiable pathology (hydrocephalus or space occupying lesion).

It is characterized by the presence symptoms of raised intracranial pressure viz. head ache, vomiting, blurring of vision, pulsatile tinnitus, double vision, transient visual obscurations and signs of raised intracranial pressure (ICP) , like papilledema, VI th cranial nerve palsy, and investigations revealing increased cerebrospinal fluid (CSF) pressure with normal biochemical and cytologic analysis of CSF.

This disease has been referred by various names since Quincke first described it as “meningitis serosa” as early as 1893. Various terminologies used includes ‘otitic hydrocephalus” ‘Pseudotumour cerebri‘, ‘benign intracranial hypertension’. The term ‘ Benign intracranial hypertension’ which was in use for very long time, since Foley introduced it in 1955, was reconsidered as several reports of visual loss in these patients questioned the benign nature of this illness and hence it was renamed as ‘Idiopathic Intracranial Hypertension’.

IIH is considered as a disease of altered CSF flow dynamics resulting in increased CSF pressure. It is considered in those patients where a detailed neuro-imaging ruled out any structural cause for raised intracranial pressure. Incidence of disease is high among obese, young female patients.

This study tries to analyse the various clinical presentation, investigations of IIH, with the outcome of illness. We also analyse the clinical presentation, investigations of IIH that can predict early surgical intervention in these patients.

AIM OF THE STUDY

- To study the clinical profile of the patients with Idiopathic Intracranial Hypertension
- To study the co–relation between clinical presentation, CSF analysis, management and outcome of patients with Idiopathic Intracranial Hypertension

REVIEW OF LITREATURE

Historical background :

The first report of this condition was published by Heinrich Quincke (1893), a German physician who described it as 'meningitis serosa', [1] though Bouchat had described a similar disease prior to him in 1868

In 1904, Max Nonne, another German neurologist, identified cases that apparently looked like a cerebral tumour but which had more benign course of illness that made him coin the term 'Pseudotumour Cerebri'. In 1930's, Sir Charles Symonds, neurologist from England wrote a series of papers, about pediatric patients, who had raised intracranial pressure. He found an association between raised ICP and middle ear disease and described the disease as 'Otitic Hydrocephalus'.[2]

In the early and mid 20th century, serous meningitis and Pseudotumour cerebri were used extensively for those patients presenting with features of raised ICP with more benign course. Procedures like cerebral Pneumography, Ventriculography and Encepahalography permitted evaluation of live patients and further studies about this condition. Davidoff and Dyke in 1956 published 15 cases with features of raised ICP but normal cerebral pneumography and all showed marked improvement with cranial decompression.[3] The credits for

postulating first diagnostic criteria goes to Walter Dandy, who described 22 cases who had raised ICP but no tumour.[4] His criteria ' Dandy's criteria' was used for long to diagnose , cases of IIh , which was modified by Friedman and Jacobson in 2002.[5]

Foley in 1955 published a study in which he divided patients into two groups , one with ear disease and other with no known cause. He found that the cerebral ventricles were not dilated and hence regarded the term 'Hydrocephalus' in appropriate he introduced the term 'Beningn intracranial hypertension' for non otitic cases.[6] His patients were predominantly young, obese, females. Another important finding in his study was, 'there were variety of proposed numerous etiological agents, which were diverse that made one must suspect that none is a direct cause'. The term he introduced, remained for a very long time before the terminology ' Beningn ' was questioned by several case reports that reported visual loss.

It was in 1989, that Corbets and Thompson proposed the name ' Idiopathic Intracranial Hypertension' a term which was suggested by Buchhiet as early as 1969. The late part of 20th century, saw many papers that postulated the mechanism of IIH, by Johnston (1973,1975),[8,9] Fishman (1979,1984) Rottenberg et al (1980) and Donaldson (1981).[10] All of them favored, a primary pathology in CSF flow dynamics resulting in an imbalance

between formation of CSF and its absorption leading to increase in CSF volume and raised ICP.

DIAGNOSTIC CRITERIA :

Though the disease has been described since 19th century, it was poorly defined. The general rule in the diagnosis of this disease was symptoms and signs of raised intracranial pressure with no hydrocephalus or space occupying lesion. It was in 1937, Walter Dandy, American neuro surgeon proposed a diagnostic criteria for IIH.[4] The criteria proposed by him stayed long and strong untill recently when Friedman and Jacobson in 2002 revised the criteria which are now popularly known as modified Dandy's criteria.[5]

Modified dandy's criteria for Idiopathic Intracranial Hypertension[5]

1). Patient presenting with symptoms of increased intracranial pressure

2). Neurological examination is normal except for papilledema or sixth cranial nerve palsy.

3). Neuro imaging does not show hydrocephalus or space occupying lesions and absence of abnormality of ventricular systems (deformity, obstruction or displacement)

4). CSF analysis shows elevated pressure (>250mm water) but normal bio chemical analysis

5). Conscious and oriented patient

6). Other causes of raised intracranial pressure are not present

EPIDEMIOLOGY

Incidence

The exact incidence of IIH in early days is not clearly known because of absence of uniform consensus in diagnosing the diseases and lack of advanced Neuro imaging. Furthermore the studies to evaluate the incidence of IIH is complicated by the fact that nearly half the patients landed up with ophthalmologist for headache and visual disturbance however later part of 20th century saw many series of cases which were reported and the incidence of IIH calculated. Incidence has been well documented after Friedman and Jacobson postulated modified Dandy criteria

Durcan et al in 1988 at Lowa USA, reported a incidence of 0.9 per 1 Lakh population.[11] In the same year he reported a incidence of 1.07 per 1 lakh population in Louisiana USA.[29] In 1993 Radhakrishnan et al noted a incidence of 2.2/1,00,000 population in Libya[12,15]

In 2000 Yabe et al reported a incidence of 0.03/1,00,000 in Hokkaido Japan.[13] Kaslar & Gadoth reported a incidence of 0.75/1,00,000 in Israel in 2001.[14] However studies on Incidence of IIH among Indian population are lacking.

Gender and Age distribution

IIH is more prevalent in women. Larger studies have reported female to male ratio from 4:3:1 to as high as 15:1[11, 12, 16, 17]

Studies	Female : Male	Age
Durcan et al (1988)	4.3:1	Third Decade
Radhakrishnan et al	15:1	28
Craig et al (2001)	5.7:1	29
Kesler et al (2000)	8:1	-
Galvin&Van stavern(2004)	9.3:1	35
Mezael & Saaldah (2005)	11.5:1	36

It is prevalent in younger age groups. In a study Durcan et al in 1988, 59% of patients were in third decade.[11] Mean ages of onset in various studies are noted in the above table.

Role of other factors in pathogenesis of IIH

Obesity

Dandy's description of typical IIH patient was ' A young obese female patient in her thirties came with complaints of throbbing headache with vomiting and double vision and her examination showing papilledema and lateral rectus palsy while other systems were normal and her brain imaging unremarkable'.[4] Foley's study stated that 14 out of 60 pt were obese. But the prevalence of obesity in IIH is higher in other studies.[6] Geer et al reported 20 cases wherein all were obese. Further weight gain appears to have increased the risk of IIH. Various studies quoting obesity (BMI > 30) and IIH are

Radhakrishnan et al	71%
Galvin et al	88%
Kesler & Gadoth	91%
Wall et al	94%
Benghazi et al	98%

The fact that recent gain in weight leading to IIH and weight reduction relieving symptoms further supports obesity as major risk factor.[19]

Mechanism by which obesity leads to IIH.

1. Raised intra abdominal pressure [20]
2. Release of pro inflammatory cytokinins by adipose tissue (Adipokine Leptin & Adiponectin)[21]

Vitamin A

Vitamin A is known to affect the arachanoid villi structure in dural sinus leading to impaired absorption of CSF into systemic circulation.[22] There are reports of occurence of IIH among patients who have been administered retinol containing compounds for dermatological and other condition. Jacobson et al (1999) showed significant higher serum retinol in 16 IIH patients.[24]

Warnet (2002) and Tabassi (2005) showed elevation of CSF retinol levels in IIH patients compared with controls in their study. There are not enough studies to quote the percentage of prevalence of elevated retinol levels in IIH nor the role of retinol in pathophysiology of IIH.[23]

Medications

The role of drugs in IIH has been extensively studied. More common among them is tetracycline-class antibiotics,[25] corticosteroid therapy,[27] and oral contraceptive pills. Cric et al (1998) reported occurrence of IIH among patients taking tetracycline class antibiotics especially minocycline and doxycycline in 6 out of 34 patients.[25] Patients were on chromic course of drugs for dermatological treatment. Similarly abrupt corticosteroid withdrawal resulted in a clinical syndrome similar to IIH.[26] Further reintroduction of drug showed clinical improvement in these patients. The role of oral contraceptive pills in IIH has been widely debated. Whether OCP directly resulted in IIH or as a result of accompanying cerebral venous thrombosis is not clear. However Glueck et al in 2005 reported a series of IIH cases among 65 women who had normal cerebral venography, which strengthened the hypothesis of OCP directly causing IIH.

Other medications associated with IIH.

Hormonal

- Corticosteroid withdrawl
- Levonorgestral
- Danazol
- Tamoxifen
- Growth hormone
- Anabolic steroids

Antibiotics

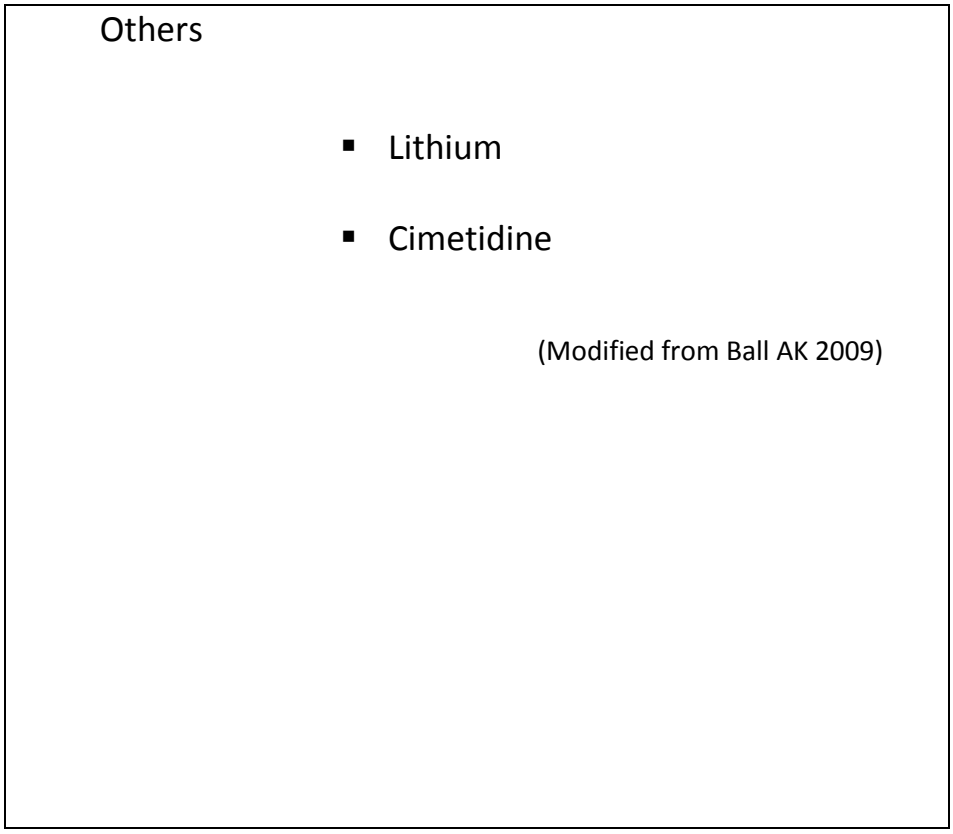
- Tertacycline and derivatives
- Nalidixic acid/
- Nitro furantoin

NSAID'S

- Indomethacin
- Rofecoxib

Vitamin A

- Retinol
- Retinoids



Pregnancy

The role pregnancy in pathogenesis of IIH has been traditionally studied. Grees(1965), reported eight patients with IIH in the first trimester of gestation. He also noted that duration of symptoms was shorter compared to non pregnant women, initially suggesting relationship between changes in estrogen levels to IIH. Later in 1984, Digre et al, after retrospective analysis noted that association of symptoms with age is a typical characteristic of an IIH patient rather than hormonal changes, a fact which was further supported by Durcan et al(1988) and Ireland et al(1990).[28]

Other comorbid factors	Prevalence	Studies
Polycystic ovary syndrome	57%	Glueck et al(2005)[30]
	39%	Glueck et al(2003)[31]
Sleep apnea syndrome	12%	Marcus et al(2001)
	30%	Lee et al (2002)
	4% Female 24% Male	Bruce et al (2009)
Anaemia	9% Female 5% Male	Bruce et al (2009)
	6%	Mollan et al(2009)
Systemic Hypertension	14% 28% 32%	Wall & George (2001) Galvin et al (2004) Ireland et al (1990)
	21% females 24% males	Bruce et al (2009)

Other co-mortid factors associated with IIH.

Diabetes mellitus, Migraine, SLE , Stroke, Hypoparathyroidism, Hepatitis A&E, Leukemia, Renal Transplant, Lysosome Storage Disorders.[32]

CONDITION THAT MIMIC IIH (MICHEAL WALL 2010)

- Reduced flow through arachanoid granulations
    1. Scarring from previous inflammation (meningitis, SAH)
- Obstruction to venous outflow
    1. Venous Sinus Thrombosis
    2. SVC Obstruction
    3. Glomus Tumour
    4. Elevated Right Heart Pressure
- Endocrine Disorders
    1. Hypoparathyroidism
    2. Use of Growth hormone
    3. Steroid withdrawal
    4. Obesity
    5. Addison disease
- Nutritional Disorders
    1. Hypervitaminsosis A
    2. Hyperalimentation in deprivation

- AV malformation and dural shunts
- Drugs
    - Anabolic Steroids
    - Chlordecone
    - Ketoprofen or indomethacin in Bartter syndrome
    - Thyroid replacement
    - Tetracycline and its derivatives
    - Amiodarone
    - Lithium carbonate
    - Nalidixic acid
    - Sulfa antibiotics
    - Oral contraceptives
- Diseases
    - SCE, Via Venous Sinus Thrombosis
    - Sarcoidosis
    - Iron deficiency anemia
    - Hyperthyroidism
    - Menarche
    - Menstrual disturbances
    - Pregnancy

PATHOPHYSIOLOGY OF IIH

Even after 100 yrs since Quincke's first description, the pathogenesis of IIH remains uncertain.[33,34] Any Hypothesis postulated on pathophysiology of IIH should explain few intricacies of IIH namely absence ventriculomegaly, Prelidiction to affect obese young females especially during child bearing age, reduced conductance to the outflow of CSF and absence of histological evidence of cerebral edema.

IIH is basically a disease of altered CSF hemodynamics resulting in raised ICP. Alteration can be either increased CSF production or decreased CSF absorption.[8]

CSF PRODUCTION & CIRCULATION

CSF is produced in choroid plexus of lateral ventricles by ependymal cells. There is a rich vasculature in the ventricles. The rate of CSF production is 500ml in 24 hrs with a volume of 140ml at any point of time and CSF is renewed every 6-8 hrs. From the lateral ventricle, CSF enters into Third ventricle by foramen of Monro and then into fourth ventricle through cerebral aqueduct. Through median and lateral apertures it flows through cerbellomedullary cistern into spinal cord and over cerebral hemispheres.[10]

EXCESSIVE SECRETION

Quincke in 1893 suggested that there could be hyper secretion of CSF leading to raised ICP in IIH.[1] However later studies with isotope cisternognaphy proved it wrong.

Several studies postulated that there is no difference in CSF production between IIH patients and controls and in fact it is less. Hence the hypothesis of increased CSF production in pathogenesis of IIH faded way.[3]

CEREBRAL EDEMA

Earlier study reported diffuse brain edema in IIH as demonstrated in postmortem findings. Later studies using various MRI sequences had showed no significant brain edema. Absence of other clinical features of edema, preservation of neurological function and clinical course all made cerebral edema unlikely in IIH.[35,36]

REDUCED CSF ABSORPTION

The most accepted concepts in pathogenesis of IIH is the reduced CSF absorption. The circulating CSF are absorbed into systemic circulation by arachanoid villi and granulations in dural venous sinuses. An earlier study demonstrated the increased resistance to CSF drainage by intrathecal saline infusion test. In 1974, Johnson And Paterson using isotope Cisternognaphy

demonstrated a delay in circulation of CSF and also holding up in sub arachnoid space.[8] A similar disturbed CSF flow pattern was demosntrated by orefice at el in 1992 in 6 IIH patients who were obese. Gjerrris et all in 1985 used lumbo lumbar perfusion methods to measure CSF outflow resistance, he noticed abnormally low conductance in all of his 14 IIH patients. The following changes were noted in CSF flow Dynamics.[37]

1. Increased CSF pressure
2. Reduced conductance to the CSF Outflow
3. Difference in pressure across various CSF outflow pathways.

Dysfunction of arachanoid villi accounted for the reduced CSF outflow conductance. Whereas in patients who has normal or slightly reduced outflow conductance, high pressure in sagital venous sinuses were recorded. The increase in CSF Pressure is however not associated with consequent ventricular dilatation. This was probably explained by Lenine et all in 2000. In which he suggested that increased CSF pressure is uniformly distributed in ventricles and subarachnoid space and a new equilibrium is reached which prevents ventricular dilation. The conclusion of his study stated that there is raised ICP due to abnormal absorption of CSF into blood stream by abnormalities in archanoid villi

IIH and cerebral venous system

Whether the increased CSF Pressure in IIH is primary or Secondary to high Pressure in venous sinuses is debatable. In earlier days raised ICP due to venous sinus thrombosis were considered as IIH but later criteria by Dandy clearly differentiates Raised ICP Due to venous sinus thrombosis from IIH.[38]

However the raised venous pressure due to anatomical narrowing or generalised venous hypertension, as a cause of IIH is debatable. Farb et al in 2003 did a extensive study among 29 IIH Patients with MRV in which he found 27 of them had substantial venous sinus stenosis leading to anatomic variability of dural venous system. King et al (2005) demonstrated raised pressure, in absence of thrombosis within the superior sagittal and transverse sinuses. Whether raised venous pressure is due to IIH or a cause of IIH is clearly not known.

CLINICAL FEATURES

Symptoms

IIH patients presents with variety of clinical symptoms and signs. Most common symptom is headache. Other common presenting symptoms include transient obscuration of vision, pulsatile tinnitus, photopsia, and retrobular pain. Less common features are diplopia and visual loss.[32] In a prospective

study of 50 patients by Wall et al in 1991, the prevalence of various symptoms are as follows: [39]

Head ache	94%
Transient visual obscuration	68%
Tinnitus	58%
Photopsia	54%
Retrobular pain	44%
Diplopia	38%
Visual loss	30%

Headache:

Headache is the most common presenting feature in majority of IIH patients. The features of IIH headache are daily severe headache, pulsatile quality which is markedly different in severity, intensity and frequency compared to previous headaches.[40] It may awaken patient from sleep or is worse in mornings when patient gets up from sleep, often reported as worst headache ever.[41] It is commonly associated with nausea and sometimes vomiting. It frequently co- exists with other head syndromes like rebound

headache from analgesic or caffeine . Neck and back pain are also prominent features.[39]

Transient visual obscurations

Transient visual obscurations are common among IIH although it is not specific for IIH. It is often a presenting symptom. In study of IIH patients by Push JA (1980), 70% of patients complained of visual obscurations typically lasting for seconds. The pathogenesis of obscuration is probably due to disc edema leading to transient optic nerve head ischemia.

Pulsatile tinnitus

In study by Givseffi et al (1991) 60% of IIH patients presented with tinnitus or intracranial ringing noises described as whooshing or roaring in the ear. The tinnitus was pulsatile with correlation to heart beat. It can be unilateral or bilateral. Tinnitus disappears or decreases after lumbar puncture or jugular venous compression. It is attributed to the transmission of intensified vascular pulsations to the venous sinus wall through high pressure CSF, converting laminar to turbulent flow.[32] Fishman 1992 postulated that tinnitus may be due to transmission of pressure sensation to endolymph of labyrinth through endolympatic duct. Hearing loss have also been described in IIH by Sisman (1985) and Dormati et al (1995).

Visual loss

Visual loss is not a usual symptom of IIH but if present is a worrisome sign. Patients usually report blurred vision or a dark spot in vision, which is probably due to enlargement of blind spot due to papilledema. Some patients have complained of tunnel like vision. However complete visual loss or blindness is seen only in severe cases of IIH. The temporal profile also varies; central visual loss early in the course of illness is a poor prognostic sign.[42] Patients usually presents with black hole in vision due to papilledema and enlargement of blind spot. In early stages the vision remains near normal. Vision loss is expected when the condition is long standing and there is retinal detachment or optic disc edema extends up to macula. Routine Snellen's chart testing can be insensitive to visual loss, which may be picked up only with perimetry. Snellen's acuity testing is also insensitive to worsening of papilledema. Contrast sensitivity is an early and a sensitive indicator of optic nerve dysfunction in IIH. The mechanism of visual loss in IIH has been extensively studied. The field defects in IIH seemed to be optic Disc related. The analysis of visual field defect and histological changes in IIH, points to optic disc (optic nerve head) as the site of damage IIH.

Hayreh SS extensively studied the mechanism of visual loss in IIH. He postulated that there are two major mechanism for damage to optic disc in IIH which are[43,48]

1. Disruption of Axonal transport

2. Intracranial optic nerve ischemia

Increased CSF pressure, low intraocular pressure and a marked decrease in systemic blood pressure leads to stasis of axoplasmic flow. The stasis of axoplasmic flow, intra axonal swelling leading to intra neuronal optic nerve ischemia.[48]

Visual loss which is mild or absent on early course of illness, rapidly progress as the intra neuronal optic nerve ischemia ensues.[50]

Double vision

Double vision or diplopia is noted in 40-60% of IIH patients and one third report it as a presenting complaint. Diplopia is usually binocular with images separated horizontally. It is due to unilateral or bilateral abducens nerve palsy. There are rare instance of monocular diplopia reported which occurs as a result of macular edema or exudates due to severe papilledema. Binocular diplopia usually resolves as treatment is started and as ICP becomes normal.[39]

Other symptoms

Other minor symptoms, that IIH patient may have at presentation includes, neck pain, shoulder arthralgia, facial palsy , radicular pain, neck shiftness, parasthesias and ataxia. Psychological manifestation includes , depression and anxiety. Minor cognitive symptoms like impaired concentration and memory have also been noted.[32]

SIGNS

Papilledema

Papilledema is a hallmark finding in IIH and the diagnosis of IIH has to be reconsidered in absence of papilledema. In IIH papilledema is usually bilateral and asymmetrical but can occur unilaterally.[45] It is usually evident by direct ophthalmoscopy but can be missed in earlier stages of illness during which, it is evident only by indirect ophthalmoscopy.[43] Stereoscopic fundus photography and fluorescein angiography may be used to detect subtle papilledema in earlier stages. Although several studies have shown a definite association between visual loss and degree of papilledema, the appearance of optic disc does not predict visual outcome in IIH patient.[44] Apart from vascular leakage, exudation, axon swelling and degeneration contributes to papilledema in raised ICP.[43,57] Since papilledema is an important sign for diagnosing, assessing severity & grading and influence of treatment it is necessary to have a

standard rating scale. The grading of papilledema is done using Frisen scale, which grades papilledema from Stage 0 to stage 5 (table).

Frisen scale[46]

Stage 0: Normal optic disc

A. Blurring of nasal, superior, and inferior poles in inverse proportion to disc diameter
B. Radial nerve fiber layer (NFL) without NFL tortuosity
C. Rare obscuration of a major vessel, usually on the upper pole

Stage 1: Very early papilledema

A. Obscuration of the nasal border of the disc
B. No elevation of disc borders
C. Disruption of the normal radial NFL arrangement with grayish opacity accentuating nerve fiber bundles
D. Normal temporal disc margin
E. Subtle grayish halo with temporal gap (best seen with indirect ophthalmoscope)
F. Concentric or radial retinochoroidal folds

Stage 2: Early papilledema

A. Obscuration of all borders
B. Elevation of the nasal border
C. Complete peripapillary halo

Stage 3: Moderate papilledema

A. Obscuration of all borders
B. Elevation of all borders
C. Increased diameter of the optic nerve head
D. Obscuration of one or more segments of major blood vessels leaving the disc
E. Peripapillary halo: irregular outer fringe with fingerlike extensions

Stage 4: Marked papilledema

A. Elevation of entire nerve head
B. Obscuration of all borders
C. Peripapillary halo
D. Total obscuration on the disc of a segment of a major blood vessel

Stage 5: Severe papilledema

A. Dome-shaped protrusions, representing anterior expansion of the optic nerve head
B. Peripapillary halo is narrow and smoothly demarcated
C. Total obscuration of a segment of a major blood vessel may or may not be present
D. Obliteration of the optic cup

It is important to exclude other conditions that mimic Papilledema (Pseudopapilledema). Conditions that mimics include the optic disc, optic disc drusen or myelinted nerve fiber layer. But these conditions can be easily differentiated by stereoscopic fundus examination. Pitfalls include absence of definitive papilledema in very early course of disease or when there is raised ICP in a pre – existing optic atrophy. In cases of relapse gliosis of retinal nerve fiber layer may mask the development of papilledema.

Ocular Motility Disturbances

Wall et al (1991) reported ocular disturbances in the form of diplopia, mainly horizontal in one third of IIH patients and common finding was sixth nerve palsy in 10-20%. Both unilateral and bilateral palsies has been noted. IIH typically presents with binocular, horizontal diplopia with esotropia. Other uncommon forms include vertical diplopia due to skew deviation or IV nerve palsy, global opthalmoparesis and LMN type of VII nerve palsies.[47] The common mechanism seems to be the acute bend that these CN make that makes them susceptible to raised ICP. The Disturbances resolves as the Intracranial pressure is lowered. In general, ocular motor palsies other than VI should be viewed with suspicion and other causes should be actively evaluated.

INVESTIGATIONS

Apart from routine investigations to rule out secondary causes of raised ICP, there three important modalities to be tested for all patients suspected for all patients suspected to have IIH.

1. Visual acuity & optic nerve function test
2. Neuro imaging – especially MRI
3. CSF examination

Visual acuity testing is done by Snellen's chart though it carries poor sensitivity it is done first followed by perimetry.

Perimetry

Visual field defects occur in almost all patients with IIH. In a prospective study of 50 IIH patients done by Wall et al in 1991, showed that field defects were found in 96% of patients when Goldman perimetry is used and 92% in automated perimetry.[39] More than 30% of patients, who had mild visual loss, did not even notice visual disturbances. Commonest finding in perimetry includes enlargement of blind spot, inferior nasal step defect, loss of peripheral nasal field of vision followed by arcuate defects.[49,57] At later stages there is gradual depression of entire field which more pronounced peripherally. With

treatment there is significant improvement in field defect as evaluated by perimetry in almost 50% of patients.[52]

Neuroimaging:

A routine plain CT brain in unremarkable in patients with IIH. In fact a normal Imaging forms a criteria for the diagnosis of IIH. However MR imaging of brain may reveal empty Sella, dilated optic nerve sheath especially dilation of perineuronal sub archanoid space, protrusion of optic papilla into posterior globe resulting in flattening sclera may be seen.[53] MR venogram, an elliptic centric 3-D, gadolinium enhanced MRV, may reveal venous sinus stenosis, most commonly transverse sinus. Transverse sinus stenosis reverses with LP or Shunting. Raised ICP in uncommon patients (Men, children & slim women) should be evaluated in detail to find an underlying cause.[38,54]

CSF Examination

CSF Examination is crucial in diagnosis IIH and diagnosis should not be presumptively made without it. The LP should demonstrate a elevated CSF pressure and normal CSF Contents.[55,56]

Significant CSF pressure values
>250mm in adults
>280mm in children
>250mm in children (if not sedated /obese)
201-249 non diagnostic.

If CSF pressure fluctuates or remains normal but there is strong clinical suspicion of IIH, then LP is repeated. Alternatively CSF outflow pressure or R out can be calculated by bolus injection method.[37]

The Pressure must be measured in lateral decubitus position with legs relaxed. A spinal needle of 18-20 Gauge is prefered. The Pressures recorded in sitting / Prone posture can be inaccurate. The base of manometer should be at the revel of right atrium. The CSF should be sent for bio chemical, micro biological analysis and cytology.[8]

Treatment :

The Treatment for IIH is Multi centric , with diet and life style changes to reduce weight, Medications, like carbonic anhydrase inhibitors (acetozolamide), diureties and analegesics and therapeutic LP and CSF drainage and in refractory patients surgical intervention.[9]

Diet and Weight loss:

Weight loss is an important factor in producing remission of IIH. Sugerman et al (1995) in a study noted that 8 morbid obese females who had IIH had undergone gastric surgery and achieved weight loss of mean 55kg had resolution of papilledema and clinical improvement.[20] Kupersmith et all in 1998, made a retrospective study of 58 female IIH patients who showed improvement after losing 2.5 kg over 3 months period; various studies demonstrated a positive correlation between weight loss and improvement in symptoms.[58]

Medications

The Drug that has been extensively studied in management of IIH is Acetazolamide, which is a carbonic anhydrase inhibitor. Carbonic anhydrase is present in choroid plexus of ventricles and plays a major role in secretion of CSF. A study by Tosmark et al in 1988 showed positive improvement in symptoms in 75% of IIH patients. The effective dose is around 1-4gm/day given as divided doses as three to four times a day. It is sulfonamide drug. Adverse reactions includes rashes, aplastic anemia, renal calculus and parasthesia and unpleasant taste with carbonated beverages. Methazloamide another Sulfonamide drug with similar action may be tired if patient cannot tolerate acetazolamide.[59]

Furosemide another loop diuretic may be used, it also reduces CSF secretion from Choroid plexus.[60] Other diuretics (Thiazide, Spironolactone & Triamterene) have been tried with little success and are mainly reserved for those patients who cannot tolerate acetazolamide corticosteroids has been studied extensively in IIH.[27] It shows rapid decrease of ICP and improvement of symptoms, but adverse effects like weight gain, fluid retention, rebound hypertension on tapering makes its use limited. At present corticosteroids are used for those patients who require rapid reduction of ICP like visual loss and Surgery (Liu Gt et al 1994). Headache is symptomatically managed with NSAID'S, TCA and sodium valproate. Topiramate is another promising drug used for headache prevention and also produces weight loss. In a study of 24 IIH patients using Topinamate 15 had weight loss and 10 had improvement a headache. Indomethacin, triptans and dihydroergotamine has also been studied

Repeated Lumbar Puncture

Repeated LP has been tried in those patients who were refractory to medical treatment and not ideal candidates for surgery and in patients who are planning surgery for symptomatic relief. The effect is short lasting and unpredictable duration and more useful in infrequent exacerbations. Relief of headache and minimal improvement in vision has been noted.[9]

SURGICAL TREATMENT

The indications for surgery in IIH are loss of vision or worsening of vision that is attributable to the papilledema in IIH. Earlier subtemporal or subocipital decompression were used in patients with loss of vision and showed long term success, but these procedures are not frequently used now because of its complications line seizures, otorrhea and sub dural hematoma.[58] The Procedures widely used now are optic nerve sheath fenestration (ONSF) and CSF shunt procedures.

ONSF – It involves fenestration of optic nerve sheath or creating a window in edematous optic nerve sheath. It is considered as a treatment of choice in patients with decreasing vision. Mechanism by which it prevents visual loss is still not clear but proposed hypothesis included filtering mechanism, scarring of peneurium and shifting of pressure gradient from lamina cribrosa to myelinated segment of optic nerve. It increases the blood flow to optic nerve, decreases ICP thereby improving vision and decreasing headache.[64]

Some facts about ONSF

1. Procedure done unilaterally improves vision bilaterally
2. More useful for acute papilledema

3. Not useful in chronic papilledema or once papilledema is resolved

4. Sometimes requires revision

5. Complications includes failure, ischemic optic neuropathy transient diplopia and blindness

CSF SHUNTING

Lumbo peritoneal and ventriculo peritoneal shunting are the commonest CSF shutting procedures. Lumbo peritoneal shunting is more preferred over ventirculo peritoneal shunting. Studies showed that shunting procedure show improvement of symptoms in 82% of patients and complete resolution in 29% there was also dramatic improvement in visual field. However both shunting procedures are characterized by high failure rate >50% which requires revision of shunt and other complications, like CSF leak, abdomen pain, infection, hind brain herniation, headache, displacement of catheter also necessitated revision procedures.[63]

<u>OTHER SURGICAL PROCEDURES</u>

Venous sinus stenting

The studies showing the role of transverse sinus stenosis in pathogenesis of IIH had created interest in using endovascular stenting for the treatment of

IIH. Various case series on this procedure shows varying results.[62] Through there is improvement in clinical symptoms, there is high mortality due to complications of procedure which includes sub dural and epidural hematoma, anaphylaxis , hearing loss and death. Hence this procedure is preserved for severe or fulminant IIH and in those patients who have tried all other surgical methods.[58]

BARIATRIC SURGERY

The role of bariatric surgery in management of IIH was studied by sugerman et all (1995) in which he showed positive improvements in 8 morbid obese female IIH patients. However it plays a role in long term management of IIH, rather than acute relief of symptoms and it has no role in improvement of visual function or prevention of visual loss.[61]

Materials and Methods

The study was undertaken at The Institute of Neurology, Madras Medical College And Rajiv Gandhi Government General Hospital, Chennai between December 2012 and December 2013. All patients who presented with headache with or without vision disturbance and who fulfilled modified Dandy's criteria for IIH were taken into study after obtaining consent. All patients who had structural lesions (SOL) in CT or MRI were excluded from study. A detailed clinical examination was done and findings were entered into a proforma (see annexure). Patient underwent imaging studies, MRI brain and after excluding SOL or hydrocephalus, lumbar puncture was done and CSF analysis done. CSF pressure study was done by bolus injection method, advocated by Marmarou and improvised at the Institute Of Neurology (MIN method), after obtaining prior, written consent from patients.

CSF Pressure Study

Procedure

Patient is positioned in right lateral decubitus position and under sterile aseptic precautions, lumbar puncture is performed with 20 G needle and the needle is connected to saline manometer through a 3 way adopter without letting out any CSF. The patient is allowed to relax, extend lower limits and

neck, lying comfortably in right lateral position, and opening pressure Po is noted after saline column stabilizes.

A known volume of saline rV, usually 5ml is injected into subarachnoid space at the rate of 1ml/second through 3 way port. The peak pressure Pp, reached after the bolus injection is noted. The saline column falls gradually after reaching one peak. After a certain time, t (in min), The pressure recording in the manometer, Pt is noted.

The CSF outflow resistance is calculated by the two step Marmarou's formula:

Step 1: Calculation of the pressure – volume index (PVI).

PVI = rV/log (Pp/Po)

Step 2: Calculation of resistance to outflow of CSF (Rout)

Rout = $\frac{t.Po}{Pv1 \ .\log Pt/Pp.(Pt-Po)}$ $\mathrm{CmH_2O/ml/min.}$

This value is divided by 1.36 to express in mmHg/ml/min. about 25 to 30 ml of therapeutic drainage of CSF was done in each case. The procedure was uneventful in all cases, and the patients tolerated procedure well. The details of the patient and observations were recorded in a detailed proforma, tabulated.

ANALYSIS OF RESULTS

Forty patients with features of IIH were treated during December 2012-December 2013 were included in the study.

Age:

Age group	No of patients
16-20	1
21-25	6
26-30	14
31-35	4
36-40	12
41-45	3

Sex :

MALE	FEMALE
5	35

Body mass index:

BMI of patients were calculated using formula

BMI = weight in Kg/(Height in m)^2

Patients were classified according BMI as

BMI	CATEGORY
< 19	under weight
19-24.9	normal weight
25-29.9	overweight
> 30	obesity
Class I – 30-34.9 II – 35-39.9 III - > 40	(NIH 2000)

BMI	NO.OF.PATIENTS
Normal	1
Over weight	11
OBESE	28

Symptoms :

Symptoms like head ache, diplopia, tinnitus were analyzed among 40 patients, head ache was present in all 40 patients and constituted the major symptoms for which patients sought neurological opinion. The head ache was holocranial, compressing to stabbing type and was pulsatile in majority of them. It was associated with vomitting and nausea in half of the patients.

Diplopia was present in 32 patients and in 13 Patients diplopia was a presenting symptom. 29 patients mainly complained of blurring of vision but on probing they were noted to have diplopia. All of them complained of disturbances in distant vision .

15 patients complained of ringing sound in ears, which was unilateral in 8 patients and bilateral in remaining . 6 patients complained of hearing intracranial noises.

Vision disturbances in the form of visual obscurations were complained by 23 patients. Many of them complained as transient darkness and remaining as blurring of margins of images lasting as short as 5 sec to 2 min. there was however no complete loss of vision reported by any of them. Visual obscuration appeared prominent when the headache intensity increases.

Symptoms	No of patients
Headache	40
Diplopia	32
Tinitus	15
Visual obscuration	23

Clinical findings

All Patients Underwent Neuro Opthalmological evaluation which includes examination of Fundus by Neuro ophthalmologist and visual field charting by perimetry . Fundus examination revealed Papilledema in all patients with bilateral Papilledema in 28 patients, while 8 patients had Papilledema only in right and 4 only in left. Even in Patients with unilateral papilledema, hyperemia of disc on opposite side was noted. Visual field examination revealed enlargement of blind spot corresponding to finding of Papilledema.

Among 40 patients,32 had evidence of lateral rectus palsy, of which 22 had right, 10 patients had left and 4 patients had bilateral lateral rectus palsy. However visual acuity measured using Snellen's chart was normal in 24 patients, while remaining had visual acnity ranging from 6/24-6/60. Worst vision recorded was 5/60 in a patient who subsequently underwent surgery for detoriation of vision.

Papilledema:	
Bilateral -	28
Right alone –	8
Left alone -	4

Lateral Rectus Palsy	
Bilateral –	4
Right alone –	22
Left alone -	6

Associated conditions :

Three of our patients were pregnant during our study, one in first trimester and 2 in second trimester. All the 3 had history of consumption of oral contraceptives prior and also had headache prior to conception. One patient was detected to have IIH prior to pregnancy and was on Acetazolamide, while other 2 patients were detected only after conception

Ten patients were on chronic drug use. Seven had oral contraceptive pills and 3 steroids. Among seven, 4 had taken them regularly during preceding 1 year. Among 3 patients who had taken steroids 2 had taken for nephritic syndrome 10 years prior to onset but discontinued and was not on drugs now. One patient had taken steroids for bronchial asthma through all routes, inhalation, oral and systemic.

Investigations

All patients were evaluated with routine biochemical investigations including sugar, renal and liver parameter. MRI brain was taken with special focus over orbital cuts MRI changes including perioptic nerve sheath widening, empty sella, kinking of optic nerve, flattening of posterior globe of sclera were noted. MRI was also screened for SOL and hydrocephalus.

MRI FINDINGS	Alone	1+2	1+2+3	Total
1) Empty Sella	10	4	10	24
2) Peri optic nerve sheath widening	10	-	10	20
3) Flattening of posterior globe	-	-	10	10
4) Transverse sinus Stenosis	-	4	4	4
5) Normal				10

CSF analysis

All 40 patients underwent lumbar puncture and CSF analysis including biochemical, microbiological, cytology and pressure studies were done after obtaining written consent and under strict aseptic precautions.

All patients had normal biochemical values (sugar, protein, sodium + chloride). All of them were acellular. Opening pressure and Rout as described earlier were done, CSF Opening pressure greater than 250 mm of CSF was taken as abnormal or high and CSF outflow pressure calculated greater than 9.6mm hg was considered raised .

Elevated opening pressure	26
Elevated CSF Rout	28
Normal opening pressure with elevated Rout	13
Both normal	1

All 40 patients were started T. Acetazolamide at 250- twice a day and escalated if necessary. Apart from Acetazolamide, Frusemide, loop diuretics was started for 16 patients in addition to Acetazolamide.

Patients symptoms were re-analyzed after 3 months after starting medications.

Symptomatic	-26
Asymptomatic	– 8
Not reported	-6

Among 26 symptomatic patients, repeat lumbar puncture and therapeutic CSF drainage for persistence of symptoms were done in 7 patients. Among 26 symptomatic patients, 7 were taken up for surgery, all had worsening of vision, enlargement of blind spot and persistence of headache. All underwent lumbo peritoneal stunt. Surgery was uneventfull

STATISTICAL ANALYSIS

All result were tabulated and statistically analyzed using SPSS 17.0 Software. All the Symptoms and BMI were analyzed with outcome after 3months and for surgery. None of them had statistically significant co-relation with the Outcome.

CSF Pressures, opening pressure and Rout had statistically significant Co-relation with outcome (Opening Pressure P = 0.009 and Rout P = 0.037). Elevated opening Pressure and CSF Rout Considerably affects outcome and patent with elevated values remain symptomatic at the end 3 months of treatment with adequate dose of acetazolamide.

Correlation between CSF - Opening Pressure and Symptoms After 3 Months

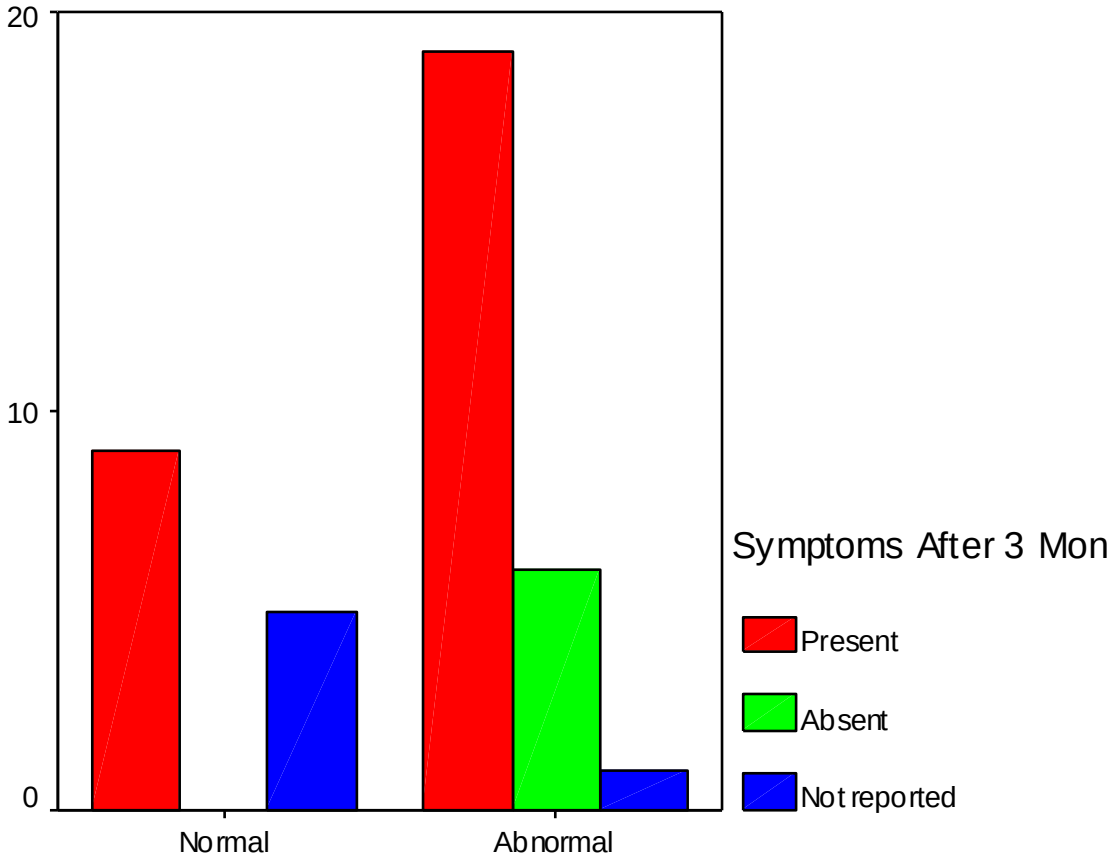
			Symptoms After 3 Months			
			Present	Absent	Not reported	Total
CSF - Opening Pressure	Normal	Count	9	0	5	14
		% within CSF - Opening Pressure	64.3%	.0%	35.7%	100.0%
		% within Symptoms After 3 Months	32.1%	.0%	83.3%	35.0%
	Abnormal	Count	19	6	1	26
		% within CSF - Opening	73.1%	23.1%	3.8%	100.0%

		Pressure				
		% within Symptoms After 3 Months	67.9%	100.0%	16.7%	65.0%
Total		Count	28	6	6	40
		% within CSF - Opening Pressure	70.0%	15.0%	15.0%	100.0%
		% within Symptoms After 3 Months	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	9.492(a)	2	.009
Likelihood Ratio	11.224	2	.004
Linear-by-Linear Association	2.679	1	.102
N of Valid Cases	40		

a 4 cells (66.7%) have expected count less than 5. The minimum expected count is 2.10.



CSF - Opening Pressure

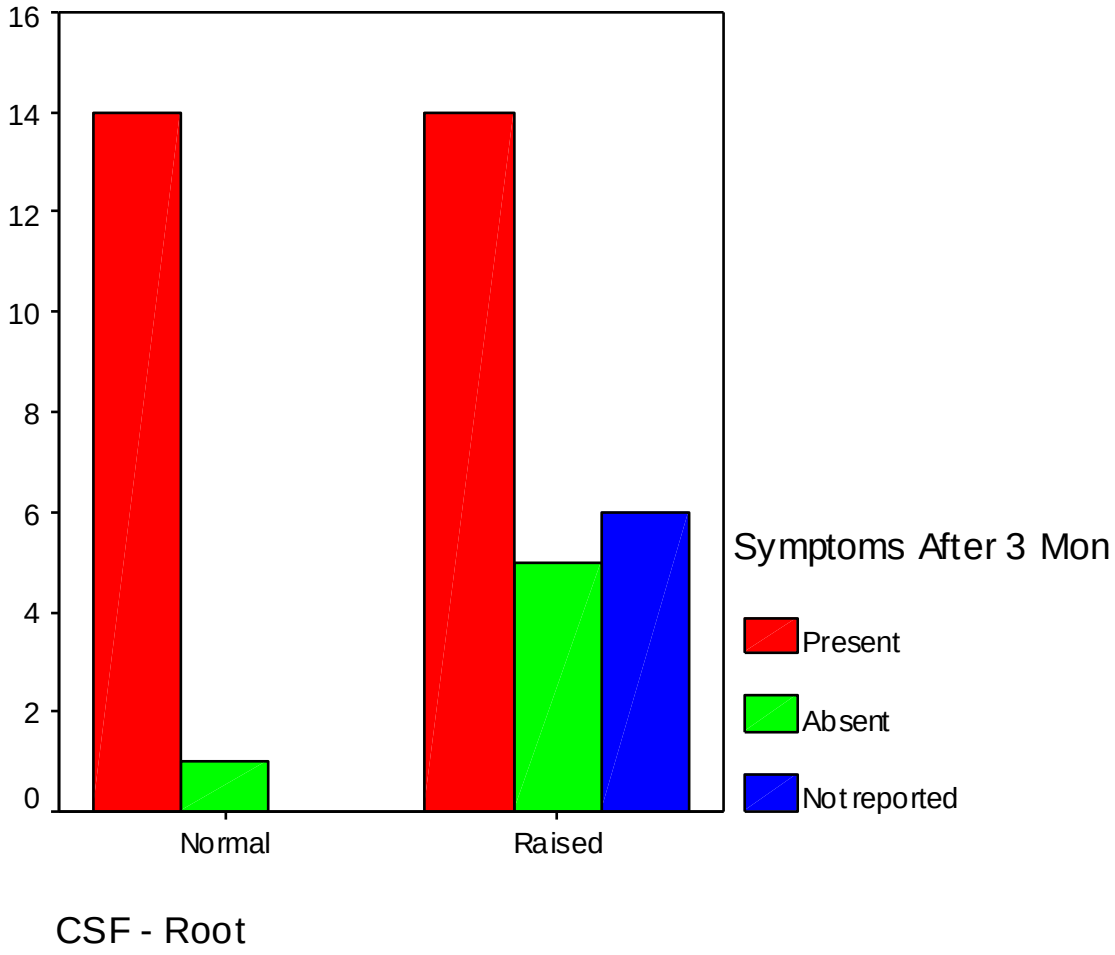
Correlation between CSF - Rout and Symptoms After 3 Months

			Symptoms After 3 Months			
			Present	Absent	Not reported	Total
CSF – Rout	Normal	Count	14	1	0	15
		% within CSF – Rout	93.3%	6.7%	.0%	100.0%
		% within Symptoms After 3 Months	50.0%	16.7%	.0%	37.5%
	Raised	Count	14	5	6	25
		% within CSF – Rout	56.0%	20.0%	24.0%	100.0%
		% within Symptoms After 3 Months	50.0%	83.3%	100.0%	62.5%
Total		Count	28	6	6	40
		% within CSF – Rout	70.0%	15.0%	15.0%	100.0%
		% within Symptoms After 3 Months	100.0%	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	6.578(a)	2	.037
Likelihood Ratio	8.702	2	.013
Linear-by-Linear Association	6.280	1	.012
N of Valid Cases	40		

a 4 cells (66.7%) have expected count less than 5. The minimum expected count is 2.25.



Elevated CSF opening pressure, shows a statistically significant correlation ($p<0.050$ Significant at 5 Level) on deciding the need for surgery in IIH patients. However CSF Rout does not show significant co- relation in predicting the need for surgery.

Correlation between CSF - Opening Pressure and Surgery

Crosstab

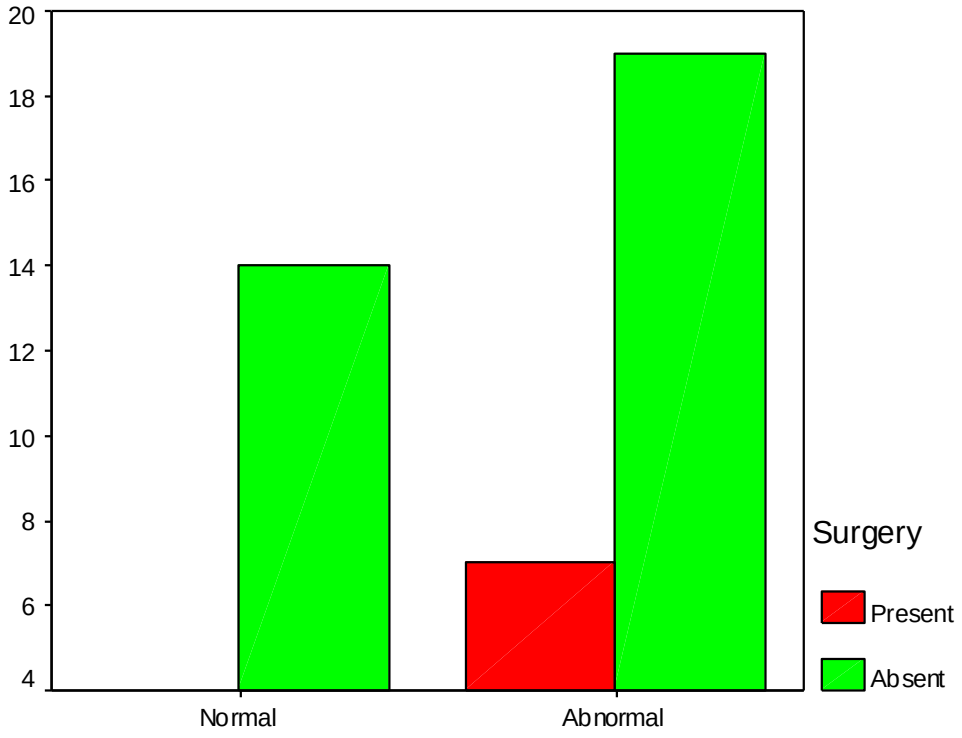
			Surgery		
			Present	Absent	Total
CSF - Opening Pressure	Normal	Count	0	14	14
		% within CSF - Opening Pressure	.0%	100.0%	100.0%
		% within Surgery	.0%	42.4%	35.0%
	Abnormal	Count	7	19	26
		% within CSF - Opening Pressure	26.9%	73.1%	100.0%
		% within Surgery	100.0%	57.6%	65.0%
Total		Count	7	33	40
		% within CSF - Opening Pressure	17.5%	82.5%	100.0%
		% within Surgery	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.569(b)	1	.033		
Continuity Correction(a)	2.894	1	.089		
Likelihood Ratio	6.809	1	.009		
Fisher's Exact Test				.075	.035
Linear-by-Linear Association	4.455	1	.035		
N of Valid Cases	40				

a Computed only for a 2x2 table

b 2 cells (50.0%) have expected count less than 5. The minimum expected count is 2.45.



CSF - Opening Pressure

Correlation between CSF - Rout and Surgery

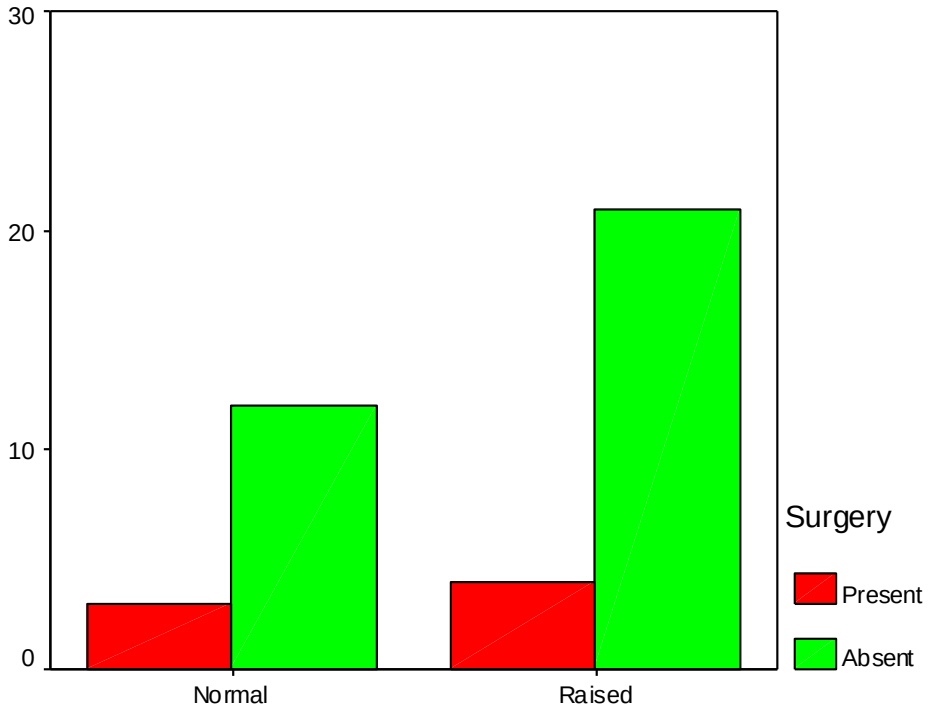
			Surgery		
			Present	Absent	Total
CSF – Rout	Normal	Count	3	12	15
		% within CSF – Rout	20.0%	80.0%	100.0%
		% within Surgery	42.9%	36.4%	37.5%
	Raised	Count	4	21	25
		% within CSF – Rout	16.0%	84.0%	100.0%
		% within Surgery	57.1%	63.6%	62.5%
Total		Count	7	33	40
		% within CSF – Rout	17.5%	82.5%	100.0%
		% within Surgery	100.0%	100.0%	100.0%

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.104(b)	1	.747		
Continuity Correction(a)	.000	1	1.000		
Likelihood Ratio	.103	1	.749		
Fisher's Exact Test				1.000	.533
Linear-by-Linear Association	.101	1	.750		
N of Valid Cases	40				

a Computed only for a 2x2 table

b 2 cells (50.0%) have expected count less than 5. The minimum expected count is 2.63.



CSF - Root

DISCUSSION

In our study, we examined the age, BMI, clinical Presentation, Investigations (Imaging & CSF Pressures) and outcome of 40 Patients with IIH.

Demographic features

Age - The Youngest age in our study is 18 and the oldest is 42 years old, with mean age of 31.7 years. This correlates well with study by Wall & George (1991) in which mean age was 31 years.

Male : Female ratio was 1:7, This is slightly lower than study by Wall and George (1991) which had a prevalence of 1:12 and Bruce et al (2009) which had 1:10.1. Keslare et al reported a similar prevalence 1:8 in 2000.

Analysis of BMI showed only I patient had normal weight, 11 (27%) patients were overweight (BMI 25-29.9) and 28 (70%) were obese (BMI >30). (NIH 2000). Studies done in western population has greater incidence of obesity (92% Ak ball 2009). Among IIH patient Wall and George reported 47 out of 50 patients as obese.

Symptoms

Headache is present in all 40 Patients in our study, Headache has a quality of raised ICP in all of them, and majority experience vomiting and nausea at least once during this course of illness. Various studies (Wall and

George 1991, Radhakrishnan et al 1993, kesler et al) all noted a similar prevalence of headache among IIH patients (80-98%)

Diplopia was noted in 32 (80%)of our patients and most of them described it as double vision for distant objects and on looking to either side. Visual obscurations was noted in 23 (57%) of patients in our study. Similar incidence was noted in various other studies between 57-72%. (Wall and George 1991, Radhakrishna 1993).

Intracranial noises, describing as ringing and whooshing sounds, were heard in 15 (37%) of patients. Studies by Wall and George noted higher prevalence (60%) of intra cranial noises.

Clinical Signs

Papilledema is almost a universal finding in IIH patients all patients had papilledema either unilateral or bilateral. Bilateral papilledema was noted in 28 patients whereas right alone in 8 patients and left eye alone in 4 patients. Another marked feature is presence of asymmetry in fundus findings even in patients with bilateral papilledema.

Visual Acuity - 24 patients had decreased visual acuity either unilaterally or bilaterally as measured by Snellen's chart. Best vision was 6/6 and worst visual acuity recorded was 5/60, in a patients who had subsequent detoriation of vision and had to be taken up for surgery. 13 of this patients were wearing

correction glasses prior to onset of headache, for refractory errors. None of them had earlier prescription to compare the previous visual acuity. Only 11 patients were detected newly to have decreased visual acuity. Literature suggests that there is reduced central visual acuity and that too in 1/4th of Patents (Craig et al 2001).

Associated Conditions

3 Patients in our study population were pregnant one in 1st trimester and other 2 in second trimester. One patient was diagnosed as IIH 1 year Period to conception and was on Acetazolamide. She had increased intensity of headache, Acetazolamide dose was titrated upwards, and intermittent analgesics were given. Her pregnancy and post partum period were uneventful many studies have failed to prove the association of pregnancy with IIH. Two Patient were diagnosed as Nephrotic syndrome 10 and 13 years before and was given a course of steroids. Both of them have stopped steroids long ago. Another patient was known asthmatic since childhood and was taking salbutamol and budesonide nebulisation along with oral and systemic corticosteroids intermittently. However there is no history of abrupt withdrawl of corticosteroids.

Investigations

Visual field defects - The most common field defect noted is enlargement of blind spot, and this finding correlated with those patients who had papilledema. It is the most common visual field defect noted by Rowe and sarkies in 1998, Hung et al in 2003 and gallium and van tavern 2004. Apart from blind spot enlargement one patient had arcuate scotoma. Field defects other than blind spot enlargement, like arcuate scotoma, and centrocecal scotoma, with temporal and altitudinal field defect were also noted by Wall et al in 1991.

CSF pressures.

CSF opening pressures were elevated in 26 patient (>250mm CSF) in our study. CSF Rout was elevated in 28 patient. 13 patient who had normal CSF opening pressure had elevated CSF Rout with clinical picture typical of IIH. Only one patient had both CSF opening and rout values normal, but his clinical features, MRI features strongly correlated with IIH and was started on Acetazolamide, following which there was improvement of clinical symptoms. Gjerris et al found raised Rout in the initial phase in most patients with IIH. Calabrese et al found Rout was increased in all Patients with IIH. Increased Rout was found in 8 of 70 patients by Sklas et al and many of them were receiving medical treatment. 10 out of 12 patients with IIH were found to have

higher than normal Rout values (Alback et al). In a study by Ramesh et al in our institute in 2011, showed 71% patient had raised Rout. Combined use of CSF opening and Rout values has detected IIH in 43 out of 47 Patient. 10 out of 14 patients having normal opening pressure, had raised Rout values.

Imaging

The most common imaging finding in MRI was empty sella, which was seen in 24 patient in our study (10 alone& others in combination), which was followed by perioptic nerve sheath widening seen in 20 patient. 4 patient had combination of both. Flattening of posterior globe was seen in 10 patient. Transverse sinus stenosis (left) was noted in 4 patients. There was no thrombosis. Study by Friedman and Jacobson (2002) noted similar MRI changes, however perioptic nerve stealth widening and kinking of optic nerve was noted in majority of patient.

Course

All 40 patient enrolled in study were started on acetazolamide and six were also started on diuretics. One month after starting treatment 32 patient reported improvement in symptoms. Headache and visual disturbances decreased. 6 Reported to have same degree of symptoms and 2 reported worsening of headache and visual disturbance. All 8 were started on diuretics also and repeat LP was done in 2 of them.

Patients were asked report at the end of 3 months. 34 Patient reported after 3 months. 28 Patients had recurring symptoms.

Among 32 Patient who were symptom free at 1 month, 28 Patient reported and 22 had recurrence of symptoms. Among 8 patients who were symptomatic at end of 1month, 6 reported and all of them had persistence of symptoms.

Recurrence rate was high in our study 54% (22 Patient) recurrence rates were low in studies by corbet et al 1982 (8%) and shall et al 2008 (15%). However study by kesler et al had a recurrence rate of 38%.

Among 22 Patients who had recurrence of symptoms around 17 patients had either discontinued drugs or had poor drug compliance.

Surgery

Surgery was done in 7 patient enrolled in our study, who had recurrence of symptoms at the end of three month treatment period. Worsening of vision was the indication for surgery in all the 7 patients. All of them underwent lumbo peritoneal shunt. Six patients had repeat LP done prior to surgery. Post operative period was uneventful for 6 patients. One Patient developed 2 episodes of generalized tonic - conic seizures on 5^{th} post operative day and new onset varying of type of headache that aggravated on assuming erect posture. There was no focal neurological deficits or meaningeal signs. Post

Operative MRI showed T2 and FLAIR hyper intensities in left posterior cortex with gyral enhancement and pachymeningeal enhancement in contrast, suggestive of intracranial hypotension. Patient improved with bed rest and steroids.

Clinical characteristics

All the seven were females. 3 were in 26-30yrs, 2 in 36-40 yrs age group, others were 22 and 41 yrs old. 1 patient was overweight and the rest were obese. All had presenting visual acuity of 6/60 or lower and visual acuity was reduced bilaterally. None of them had associated conditions like pregnancy and none of them had taken any other medications previously.

Symptom analysis

PT. NO	HEADACHE	DIPLOPIA	VISION DIST.	TINNITUS
1	+	+	+	-
2	+	-	-	-
3	+	+	+	+
4	+	+	+	-
5	+	+	+	-
6	+	+	+	-
7	+	-	-	+
TOTAL	7	5	5	2

All patients had complaints of headache at presentation, diplopia and vision disturbances were present in 5 patients and tinnitus was present in only 2 of them. Headache was the most prominent complaint, followed by diplopia and vision disturbances.

Signs

PT. NO	PAPILLEDEMA		VISUAL ACUITY DEFECT		LATERAL RECTUS PALSY		
	U/L	B/L	U/L	B/L	U/L	B/L	NIL
1	-	+	-	+	+	-	-
2	+	-	-	+	-	-	+
3	-	+	-	+	+	-	-
4	-	+	-	+	+	-	-
5	-	+	-	+	-	+	-
6	-	+	-	+	-	+	-
7	+	-	-	+	-	-	+
TOTAL	2	5		7	3	2	2

Papilledema was present in all patients, bilaterally in 5 and unilaterally in 2 and all patient had a decreased visual acuity of 6/60 or lesser bilaterally. Presence of decreased visual acuity at the early in the course of illness may predict early surgical intervention in those patients.

INVESTIGATIONS

All seven had abnormalities in MRI, which are consistent with diagnosis of IIH

PT.NO	Empty sella	Optic nerve sheath widening and kinking of optic nerve	Thining of post. Globe	Transverse sinus stenosis
1	-	+	-	-
2	+	+	+	+
3	-	+	+	-
4	+	+	+	-
5	+	+	-	+
6	-	+	-	-
7	+	+	-	-
Total	4	7	3	2

All the 7 patients had perioptic nerve sheath widening and kinking of optic nerve, while empty sella was present in 4 and transverse sinus stenosis in only 2 patients. Empty sella and Optic nerve sheath widening and kinking of optic nerve together was found in 4 patients and all the findings in only 2 patients.

CSF Pressure studies

PT.NO	OPENING PRESSURE(mm CSF)	CSF Rout (mmHg/ml/min)
1	35	6.6
2	36	10.9
3	65	17
4	80	36
5	45	8.1
6	37	5.8
7	37	16.1

All 7 patients had elevated opening pressure ranging from 35 to 80 mm CSF. However CSF Rout was elevated only in 4 patients and 3 had normal opening pressure.

Among the parameters assessed high BMI in the range of obesity, decreased visual acuity early in course of illness and presence of peri optic nerve sheath widening and kinking of optic nerve in MRI were more associated with need for surgical intervention.

CONCLUSION

In our study we analysed the demographic features, clinical presentation, investigations and outcome of IIH patients, following conclusions were observed

- Majority of our patients were in 2^{nd} – 3^{rd} decade.
- There was high female preponderance in our study
- Obesity was the most common risk factor.
- Headache is the universal presenting symptom in all patients, followed by diplopia and visual disturbances
- All of them had papilledema as a predominant finding, followed by lateral rectus palsy but no other focal neurologic deficits
- Typical imaging findings of IIH were present in majority of patients
- Three fourths of our patients had elevated CSF opening pressure
- Significant number of patients with normal opening pressure had raised CSF Rout and classical symptoms and imaging findings of IIH which helped in diagnosis
- One fourth of the patient improved with medications alone and remained symptom free by three months

- Three fourths of the patients had recurrence of symptoms, which was managed with increased dosage of drugs and half of them showed improvement in symptoms
- Eighteen percent of the patients underwent surgery for recurrence of symptoms while remaining were managed conservatively
- Among the parameters assessed, high BMI in the range of obesity, decreased visual acuity early in course of illness and presence of peri optic nerve sheath widening in MRI were more associated with need for surgical intervention .

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ABBREVATIONS

IIH	- Idiopathic Intracranial Hypertension
ICP	- Intracranial pressure
CSF	- Cerebro Spinal Fluid
BMI	- Body Mass Index
R out	- Resistance to outflow
NSAIDS	- Non steroidal anti inflammatory drugs
LP	-Lumbar puncture

PROFORMA

Patient Number:
Age: Sex
HISTORY
Symptoms: Headaches
Visual loss
Transient visual obscurations
Double vision
Nausea & vomiting
Others

Duration of symptoms :

Associations: Obesity or recent weight gain
Medical conditions
Pregnancy
Medication or drug use

Examination:

Visual acuity: Initial R L

Ocular motility: R L

Pupil reactions: R L

Fundus examination:
Optic discs R L

Rest of fundus R L

Neurological examination: HMF

CN

SMS
SENSORY
EPS
PN S

INVESTIGATIONS: Hemogram
RFT
LFT

VISUAL FIELD CHARTING R L

NEUROIMAGING: CT SCAN

MRI

LUMBAR PUNCTURE: Opening pressure

R out

CSF composition PROTEIN
SUGAR
CELLS

MANAGEMENT:

Weight loss:

Medical: acetazolamide Steroids

Surgical: ONSF Lumbar peritoneal shunt Multiple LP'S

PATIENT CONSENT FORM

Study Details: **"**ANALYSIS OF CLINICAL PROFILE, LABORATORY INVESTIGATIONS AND MANAGEMENT MODALITIES OF PATIENTS WITH **IDIOPATHIC INTRACRANIAL HYPERTENSION"**

Study Centre: Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai- 03

Patient may check (✓) these boxes:

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask questions and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that the sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authourities will not need my permission to look at my health records, both in respect of current study and any future research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms. ☐

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, microbiological, radiological tests and lumbar puncture procedures, if deemed necessary. ☐

I hereby consent to participate in the study.

Signature/Thumb impression: Place:
Patient name and address: Date:

Signature/Thumb impression: Place:
Study investigator's name: Date:

INFORMATION SHEET

- We are conducting a study of **"**ANALYSIS OF CLINICAL PROFILE, LABORATORY INVESTIGATIONS AND MANAGEMENT MODALITIES OF PATIENTS WITH **IDIOPATHIC INTRACRANIAL HYPERTENSION"** attending the Neurology services of Rajiv Gandhi Government General Hospital, Chennai.

- The purpose of the study is to study the clinical profile of patients with idiopathic intracranial hypertension

- The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

- Taking part in the study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time; your decision will not result in any loss of benefits to which you are otherwise entitled.

- The results of the study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment

Signature of the investigator **Signature of the participant**
Date:

MASTER CHART

S.NO.	Age in years	Age in years	Sex	BMI	Level of BMI	Headache	Diplopia	Vision Disturbance	Tinnitus	papilledema RT	papilledema LT	LR Palsy - Right	LR Palsy - Left	Visual Acuity - Right	Visual Acuity - Left	Pregnancy	Drugs	Others	Visual Field - Right	Visual Field - Left	CSF - Opening Pressure	CSF - Opening Pressure	CSF - Rout	CSF - Rout	MRI	Medication - Acetazolamide	Medication - Diuretics	Medication - Others	Symptoms After 3 Months	Surgery
1	27	26-30	Female	30.8	Obese	Positive	Positive	Positive	Positive	Positive	Negative	Positive	Positive	6/36	Normal	Negative	Oral Contracepitives	Nil	BE	Nil	42.5	Abnormal	21.4	Raised	Abnormal	Positive	Positive	Nil	Present	Absent
2	30	26-30	Female	26.4	Overweight	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	6/36	6/36	Negative	Nil	Nil	BE	BE	27	Abnormal	28.5	Raised	Abnormal	Positive	Positive	Nil	Present	Absent
3	25	21-25	Female	28.1	Overweight	Positive	Negative	Negative	Positive	Negative	Positive	Negative	Negative	Normal	Normal	Negative	Oral Contracepitives	Nil	Nil	BE	19.5	Normal	14.3	Raised	Abnormal	Positive	Negative	Nil	Not reported	Absent
4	18	16-20	Female	21	Normal	Positive	Positive	Negative	Negative	Positive	Positive	Negative	Positive	Normal	Normal	Negative	Steroids	Nephrotic	BE	BE	41	Abnormal	4.1	Normal	Abnormal	Positive	Positive	LP	Present	Absent
5	33	31-35	Male	30.2	Obese	Positive	Positive	Positive	Positive	Positive	Positive	Positive	Negative	6/36	6/36	Negative	Nil	Nil	BE	BE	47	Abnormal	61.2	Raised	Normal	Negative	Negative	Nil	Absent	Absent
6	42	41-45	Female	31.4	Obese	Positive	Positive	Negative	Negative	Positive	Positive	Positive	Positive	Normal	Normal	Negative	Nil	Nil	BE	BE	39	Abnormal	5.1	Normal	Abnormal	Positive	Negative	Nil	Present	Absent
7	24	21-25	Female	30.1	Obese	Positive	Positive	Positive	Positive	Positive	Positive	Positive	Negative	Normal	Normal	Negative	Nil	Nil	BE	BE	60	Abnormal	18.3	Raised	Abnormal	Positive	Negative	Nil	Present	Absent
8	40	36-40	Female	31.2	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	Normal	6/24	Negative	Nil	Nil	BE	BE	33	Abnormal	6.9	Normal	Abnormal	Positive	Negative	Nil	Present	Absent
9	28	26-30	Female	31	Obese	Positive	Positive	Positive	Positive	Positive	Positive	Positive	Negative	Normal	Normal	Positive	Oral Contracepitives	Nil	BE	BE	64	Abnormal	25	Raised	Abnormal	Positive	Positive	Nil	Present	Absent
10	23	21-25	male	32.1	Obese	Positive	Negative	Negative	Negative	Positive	Positive	Negative	Negative	Normal	Normal	Negative	Steroids	Asthma	BE	BE	55	Abnormal	10.1	Raised	Abnormal	Positive	Negative	Nil	Not reported	Absent
11	22	21-25	Female	33.5	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	5/60	5/60	Negative	nil	Nil	BE	BE	35	Abnormal	6.6	Normal	Abnormal	Positive	Positive	LP	Present	Present
12	40	36-40	Female	27.5	Overweight	Positive	Positive	Negative	Negative	Positive	Positive	Negative	Positive	Normal	Normal	Negative	Nil	Nil	BE	BE	25	Normal	7.3	Normal	Normal	Positive	Negative	Nil	Present	Absent
13	27	26-30	Female	26.8	Overweight	Positive	Positive	Positive	Negative	Positive	Negative	Positive	Negative	Normal	Normal	Positive	Nil	Nil	BE	Nil	24	Normal	13	Raised	normal	Positive	Negative	Nil	absent	Absent
14	37	36-40	Female	25.6	Overweight	Positive	Negative	Negative	Negative	Positive	Negative	Negative	Negative	5/60	5/60	Negative	Nil	Nil	BE	Nil	26	Abnormal	10.9	Raised	Abnormal	Positive	Negative	Nil	present	Present
15	40	36-40	Female	30.1	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	Normal	Normal	Negative	Nil	Nil	BE	BE	23	Normal	7.8	Normal	Abnormal	Positive	Positive	Nil	Present	Absent
16	32	31-35	Female	32.8	Obese	Positive	Negative	Positive	Negative	Positive	Positive	Negative	Negative	Normal	Normal	Negative	Oral Contracepitives	Nil	BE	BE	19	Normal	3.2	Normal	Abnormal	Positive	Negative	Nil	Present	Absent
17	30	26-30	Female	34.1	Obese	Positive	Positive	Negative	Negative	Positive	Positive	Negative	Positive	Normal	Normal	Negative	Nil	Nil	BE	BE	60	Abnormal	17.9	Raised	Normal	Positive	Negative	Nil	Absent	Absent
18	41	41-45	Female	34	Obese	Positive	Positive	Positive	Positive	Positive	Positive	Positive	Negative	5/60	5/60	Negative	Nil	Nil	BE	BE	65	Abnormal	17	Raised	Abnormal	Positive	Positive	LP	Present	Present
19	29	26-30	Male	31.8	Obese	Positive	Positive	Negative	Negative	Positive	Negative	Positive	Negative	Normal	Normal	Negative	Nil	Nil	BE	Nil	41	Abnormal	7.9	Normal	Abnormal	Positive	Negative	Nil	Present	Absent
20	26	26-30	Female	32	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	6/60	5/60	Negative	Nil	Nil	BE	BE+S	80	Abnormal	36	Raised	Abnormal	Positive	Positive	LP	Present	Present

S.NO.	Age in years	Age in years	Sex	BMI	Level of BMI	Headache	Diplopia	Vision Disturbance	Tinnitus	papilledema RT	papilledema LT	LR Palsy - Right	LR Palsy - Left	Visual Acuity - Right	Visual Acuity - Left	Pregnancy	Drugs	Others	Visual Field - Right	Visual Field - Left	CSF - Opening Pressure	CSF - Opening Pressure	CSF - Rout	CSF - Rout	MRI	Medication - Acetazolamide	Medication - Diuretics	Medication - Others	Symptoms After 3 Months	Surgery
21	38	36-40	Female	33.6	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Positive	6/60	5/60	Negative	Nil	Nil	BE	BE	45	Abnormal	8.1	Normal	Abnormal	Positive	Positive	LP	Present	Present
22	24	21-25	Female	28.1	Overweight	Positive	Positive	Negative	Positive	Positive	Negative	Positive	Negative	Normal	Normal	Negative	Oral Contracepitives	Nil	BE	Nil	18	Normal	8.9	Normal	Abnormal	Positive	Positive	Nil	Present	Absent
23	39	36-40	Male	31.6	Obese	Positive	Negative	Positive	Negative	Positive	Positive	Negative	Negative	6/36	6/24	Negative	Nil	Nil	BE	BE	21	Normal	10.9	Raised	Normal	Positive	Negative	Nil	Not reported	Absent
24	28	26-30	Female	26.4	Overweight	Positive	Positive	Negative	Positive	Positive	Positive	Positive	Negative	6/36	Normal	Negative	Nil	Nil	BE	BE	49	Abnormal	15.1	Raised	Abnormal	Positive	Negative	Nil	Present	Absent
25	30	26-30	Female	27.2	Overweight	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	6/36	6/36	Negative	Nil	Nil	BE	BE	25	Normal	27.8	Raised	Abnormal	Positive	Positive	LP	Present	Absent
26	38	36-40	Female	31.8	Obese	Positive	Positive	Negative	Negative	Positive	Positive	Negative	Positive	Normal	Normal	Negative	Nil	Nil	BE	BE	14	Normal	27.1	Raised	Abnormal	Positive	Negative	Nil	Not reported	Absent
27	36	36-40	Female	30.4	Obese	Positive	Positive	Positive	Negative	Negative	Positive	Positive	Negative	Normal	Normal	Negative	Oral Contracepitives	Nil	Nil	BE	31	Abnormal	11.4	Raised	Normal	Positive	Negative	Nil	Absent	Absent
28	26	26-30	Female	33.6	Obese	Positive	Positive	Negative	Positive	Positive	Positive	Positive	Negative	Normal	Normal	Positive	Nil	Nil	BE	BE	25	Normal	10.3	Raised	Abnormal	Positive	Positive	Nil	Present	Absent
29	28	26-30	Female	32.8	Obese	Positive	Negative	Positive	Negative	Positive	Positive	Negative	Negative	6/36	6/24	Negative	Nil	Nil	BE	BE	33	Abnormal	4.7	Normal	Normal	Positive	Negative	Nil	Absent	Absent
30	21	21-25	Female	27.5	Overweight	Positive	Positive	Negative	Positive	Positive	Negative	Negative	Positive	Normal	Normal	Negative	Steroids	Nephrotic	BE	Nil	16	Normal	43.7	Raised	Abnormal	Positive	Negative	Nil	Not reported	Absent
31	36	36-40	Female	32.1	Obese	Positive	Positive	Negative	Positive	Positive	Negative	Positive	Negative	Normal	Normal	Negative	Nil	Nil	BE	Nil	40	Abnormal	4.3	Normal	Abnormal	Positive	Positive	Nil	Present	Absent
32	40	36-40	Male	30.1	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	6/36	Normal	Negative	Nil	Nil	BE	BE	42	Abnormal	53.4	Raised	Normal	Positive	Negative	Nil	Absent	Absent
33	29	26-30	Female	32.8	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Positive	5/60	6/60	Negative	Nil	Nil	BE	BE	37	Abnormal	5.8	Normal	Abnormal	Positive	Positive	LP	Present	Present
34	37	36-40	Female	26.1	Overweight	Positive	Positive	Negative	Negative	Negative	Positive	Positive	Negative	Normal	Normal	Negative	Nil	Nil	Nil	BE	19	Normal	22.6	Raised	Abnormal	Positive	Positive	Nil	Not reported	Absent
35	32	31-35	Female	25.8	Overweight	Positive	Positive	Positive	Positive	Positive	Negative	Positive	Negative	6/36	Normal	Negative	Oral Contracepitives	Nil	BE	Nil	15	Normal	2.8	Normal	Normal	Positive	Negative	Nil	Present	Absent
36	27	26-30	Female	30.8	Obese	Positive	Negative	Negative	Positive	Negative	Positive	Negative	Negative	6/60	5/60	Negative	Nil	Nil	Nil	BE	37	Abnormal	16.1	Raised	Abnormal	Positive	Positive	LP	Present	Present
37	45	41-45	Female	31.2	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Negative	Positive	Normal	Normal	Negative	Nil	Nil	BE	BE	21	Normal	19.2	Raised	Abnormal	Positive	Negative	Nil	Present	Absent
38	40	36-40	Female	32.3	Obese	Positive	Negative	Positive	Positive	Positive	Positive	Negative	Negative	Normal	Normal	Negative	Nil	Nil	BE	BE	26	Abnormal	14.1	Raised	Normal	Positive	Negative	Nil	Present	Absent
39	28	26-30	Female	34.2	Obese	Positive	Positive	Positive	Negative	Positive	Positive	Positive	Negative	Normal	6/24	Negative	Nil	Nil	BE	BE	35	Abnormal	6.1	Normal	Abnormal	Positive	Negative	Nil	Present	Absent
40	33	31-35	Female	36	Obese	Positive	Positive	Negative	Positive	Positive	Positive	Positive	Negative	Normal	Normal	Negative	Nil	Nil	BE	BE	40	Abnormal	13.5	Raised	Abnormal	Positive	Negative	Nil	Present	Absent

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970
EC RegNo:ECR/270/Inst./TN/2013

CERTIFICATE OF APPROVAL

To
Dr.Senthil Nathan.J,
PG in Neurology,
MMC,Chennai-3.

DearJ.SENTHIL NATHAN

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled "Analysis of clinical profile, laboratory Investigations and management modalities of patients with Idiopathic intracranial hypertension" No.03062013.

The following members of Ethics Committee were present in the meeting held on 11.06.2013 conducted at Madras Medical College, Chennai -3.

1. Dr.SivaKumar, MS FICS FAIS --- Chairperson
2. Prof. R. Nandhini MD -- Member Secretary
   Director, Instt. of Pharmacology ,MMC, Ch-3
3. Prof. Shyamraj MD -- Member
   Director i/c , Instt. of Biochemistry , MMC, Ch-3
4. Prof. P. Karkuzhali. MD -- Member
   Prof., Instt. of Pathology, MMC, Ch-3
5. Prof. A. Radhakrishnan MD -- Member
   Prof of Internal Medicine, MMC, Ch-3
6. Prof. S. Deivanayagam MS -- Member
   Prof of Surgery, MMC, Ch-3
7. Thiru. S. Govindsamy. BABL -- Lawyer
8. Tmt. Arnold Saulina MA MSW -- Social Scientist

We approve the proposal to be conducted in its presented form. Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

R Nadhini
Member Secretary, Ethics Committee
12/7/13

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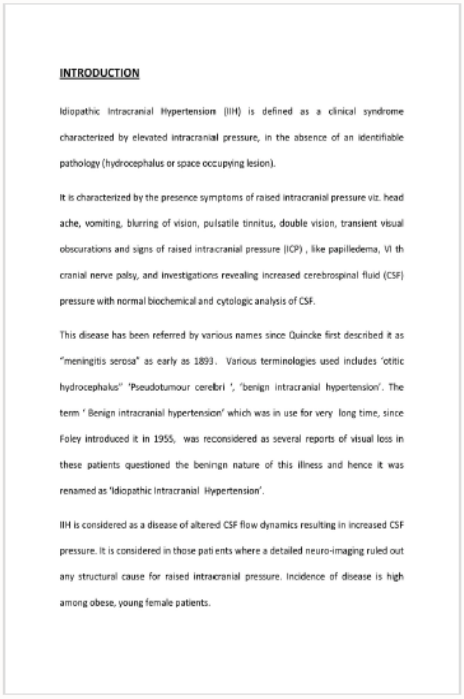

INTRODUCTION

Idiopathic Intracranial Hypertension (IIH) is defined as a clinical syndrome characterized by elevated intracranial pressure, in the absence of an identifiable pathology (hydrocephalus or space occupying lesion).

It is characterized by the presence symptoms of raised intracranial pressure viz. head ache, vomiting, blurring of vision, pulsatile tinnitus, double vision, transient visual obscurations and signs of raised intracranial pressure (ICP) , like papilledema, VI th cranial nerve palsy, and investigations revealing increased cerebrospinal fluid (CSF) pressure with normal biochemical and cytologic analysis of CSF.

This disease has been referred by various names since Quincke first described it as "meningitis serosa" as early as 1893. Various terminologies used includes 'otitic hydrocephalus" 'Pseudotumour cerebri ', 'benign intracranial hypertension'. The term ' Benign intracranial hypertension' which was in use for very long time, since Foley introduced it in 1955, was reconsidered as several reports of visual loss in these patients questioned the beninign nature of this illness and hence it was renamed as 'Idiopathic Intracranial Hypertension'.

IIH is considered as a disease of altered CSF flow dynamics resulting in increased CSF pressure. It is considered in those pati ents where a detailed neuro-imaging ruled out any structural cause for raised intracranial pressure. Incidence of disease is high among obese, young female patients.

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