

# Review on Diagnostic Modalities in Idiopathic Intracranial Hypertension

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## Abstract

IIH is a well-known but under investigated condition with an unclear etio-pathogenesis. An increase in the incidence of IIH has also been noted in the recent years, which necessitates the need for diagnostic tools to diagnose and monitor the progression of the disease. Various tests are there for IIH. The aim of this article is to discuss important aspects about each of these tests.

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## Introduction

Idiopathic Intracranial Hypertension (IIH), also known as pseudotumor cerebri is a clinical syndrome of unknown etiology, characterised by increased intracranial pressure which typically affects young, obese women of childbearing age.<sup>1-5</sup> It is a diagnosis of exclusion. IIH occurs with a frequency of about 1 case per 100000 population per year or 19.3 per 100000 in obese women aged 20-44 years.<sup>6</sup>

Papilledema with subsequent visual field loss is the most dreaded clinical consequence, and it mainly determines the therapy and outcome of the syndrome.<sup>7-10</sup> Visual impairment is usually reversible if treated timely, however, 40% cases may show permanent visual loss, and 10% may finally suffer from bilateral blindness.<sup>11,12</sup>

IIH is a well-known but under investigated clinical entity with an unsolved patho-physiologic background and with the increase in global prevalence of obesity, an increase in the incidence of IIH has also been noted<sup>13</sup>, which necessitates the need for evidence based diagnostic tools and treatment strategies to monitor the progression of the disease.

## Etio-pathogenesis

The exact etio-pathogenesis of the disease still remains obscure.

### Role of CSF:

The optic nerve is formed by around 1.2 million axons, which must maintain their orthograde (optic nerve to brain) and retrograde flow (brain to optic nerve). An obstruction of the axonal transport mechanism can result in disc edema. Also, as optic nerve sheath is continuous with the subarachnoid space containing CSF, an increase in the CSF pressure or decrease in CSF drainage can cause compression of optic nerve.

Isolated increase in CSF pressure not related to any mass can be due either due to increased production of CSF or due to impaired absorption of CSF by arachnoid villi. Transverse sinus stenosis, which can be predisposed by risk factors like weight gain, endocrine changes, hypercoagulable states and obstructive sleep apnea are also risk factors for IIH.

### Role of vitamin A

Role of vitamin A in IIH remains controversial and can be

suggested due to impaired CSF absorption by excessive retinol and retinol binding protein.<sup>14-15</sup>

### Role of obesity:

Obesity considered to be associated with IIH can be due to release of adipose tissue-derived retinol binding protein from adipose tissue.<sup>14</sup> Increased intra-abdominal, visceral fat, as in polycystic ovarian diseases is also associated with elevated levels of adipose tissue-derived retinol binding protein.<sup>16</sup> Another theory which was put forward, explaining the relation of obesity with IIH, was associated with increased intra-abdominal pressure, causing increased cardiac filling pressure, thus impeding venous return from brain and subsequently leading to an increased intracranial venous pressure and IIH, but this theory fails to explain IIH in non-obese patients.<sup>21</sup>

Though, there is still uncertainty as to whether the degree of obesity affects the severity of symptoms and disease outcomes, but number of authors have reported that for every 10 unit increase in BMI, the odds of severe vision loss increases by 1.4 times.<sup>17</sup> Lampl et al reported significantly higher serum leptin levels in IIH patients compared to obese controls.<sup>18</sup> Daniels et al demonstrated a correlation between body mass index (BMI) and the risk of IIH<sup>19</sup> and increased weight was found to be associated with recurrence of the disease.<sup>20</sup>

Chronic inflammation associated with obesity can lead to a prothrombotic state with increased expression of various cytokines (leptin, interleukins, macrophage, chemotactic protein-1, plasminogen activator inhibitor-1) and is postulated as one of the possible etiological factor in the development of IIH.<sup>22</sup>

## Clinical Features

IIH is characterised by raised intracranial pressure leading to-

- Headache (Headache attributed to IIH should be progressive with atleast one of the following characteristics – daily occurrence, diffuse and/or constant (non pulsating) and aggravated by coughing or straining, increased on lying down or bending forward, may be associated with a whooshing tinnitus)

- Papilledema
- Visual symptoms and signs (transient visual obscuration, diplopia, diminution of vision)
- Absence of any lateralizing findings in the neurological examination
- Normal CSF findings.
- Polycystic ovarian disease, hypothyroidism, obesity are co-morbidities often noted in IIH.

**Diagnosis of IIH**

**A. Diagnostic criteria:**

*Modified Dandy criteria*

IIH is diagnosed based on **Modified Dandy criteria** (proposed by Friedman).<sup>23</sup>

**Modified Dandy Criteria**

|   |
|---|
| Signs and symptoms of raised intracranial pressure  |
| No localising signs on neurological examination with the exception of sixth nerve palsy     |
| Increased CSF opening pressure measured in left lateral decubitus position (ICP >25 cm H2O) |
| Normal CSF composition  |
| Normal MRI and MR venography and normal neurological examination except for papilledema     |

**B. Clinical grading of papilledema:**

*Modified Frisen Scale*

Clinical grading of papilledema is performed using modified Frisen scale grading (Figure 1):

- A. **Grade 0:** Normal optic disc
- B. **Grade 1:** Minimal degree of edema. C-shaped halo that is subtle and grayish with a temporal gap obscuring underlying retinal details.
- C. **Grade 2:** Low degree of edema. Circumferential halo. Elevation of the nasal border. No major vessel obscuration.

- D. **Grade 3:** Moderate degree of edema. Obscuration of 1 segment of major blood vessels leaving disc. Circumferential halo with an irregular outer fringe and finger-like extensions. Elevation of all borders.
- E. **Grade 4:** Marked degree of edema. Total obscuration on the disc of a segment of a major blood vessel on the disc. Elevation of whole nerve head, including the cup. Complete border obscuration.
- F. **Grade 5:** Severe degree of edema. Obscuration of all vessels on the disc and leaving the disc.

**C. Investigations**

**1. Radioimaging**

Neuroimaging of the brain and orbit including MRV (to rule out cerebral venous thrombosis) is an important tool in the diagnosis of IIH to rule out the presence of any obstructive or compressive lesion in the brain. The usual findings in IIH noted in MRI brain and orbit are :

- Distension of perioptic subarachnoid space
- Partial empty sella
- Flattening of posterior sclera
- Vertical tortuosity and elongation of orbital ON

MRV (magnetic resonance venography) does not reveal any abnormalities in IIH.

Some patients with IIH have bilateral transverse sinus stenosis.<sup>24</sup> The transverse sinuses are well known to be asymmetric in most individuals and an unilateral hypoplastic transverse sinus is considered a “normal variant” without any reported change in ICP. There is a debate on whether such stenosis is the cause or effect of IIH.

Suggested theories are either narrowing is secondary to compression of the sinuses by the raised ICP<sup>25</sup> or presence of trabeculae, septae or hypertrophied granulations in the transverse sinuses lead to stenosis.<sup>26</sup>

**2. Visual field analysis**

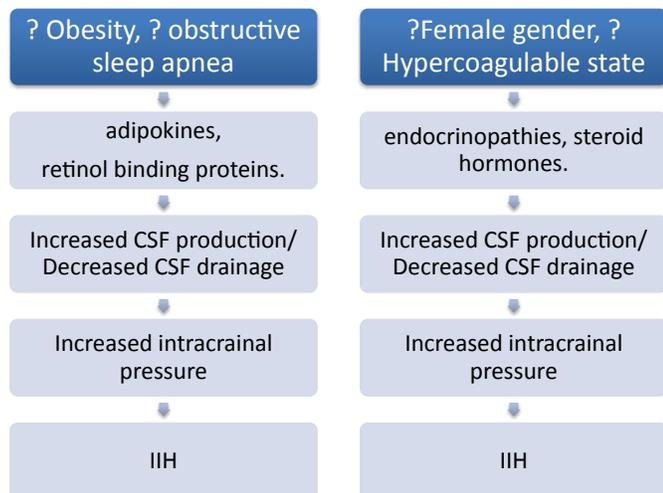
A static/kinetic perimetry is performed to document visual field defects.

Most common abnormality noted: Blind spot enlargement.

IIHTT (Idiopathic Intracranial Hypertension Treatment Trial) demonstrated that patients with IIH and mild visual loss have optic nerve related visual field deficits in localized nerve fibre bundle like patterns with the inferior hemifield exhibiting more abnormalities than the superior hemifield. Most common pattern revealed in the study was a partial inferior arcuate defect with an enlarged blind spot.

**3. Role of OCT in IIH**

OCT is a relatively new non-invasive, noncontact trans-pupillary imaging technology which provides high resolution, cross sectional images of ocular and biological structures to visualize and measure anatomic



**Figure 1: Pathophysiology of IIH.**

layers of the retina with an axial resolution of less than or equal to 10  $\mu\text{m}$  and a transverse resolution of 20  $\mu\text{m}$  in the eye.

OCT can play an important role in diagnosis and monitoring of the disease process in IIH.<sup>27</sup>

#### Diagnostic role:

The configuration of Bruch's membrane, as detected with OCT, has been shown to correlate with intracranial pressure and therefore is another feature that can be followed in IIH patients.<sup>36,37</sup> Patients with raised intracranial pressure have an upward defection of Bruch's membrane toward the vitreous, which can be helpful in differentiating papilledema from pseudopapilledema (Figure 2).

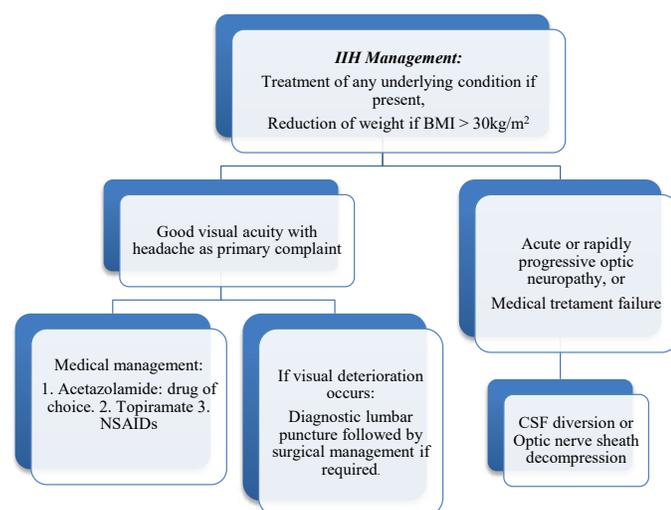


Figure 2: Management of IIH.

Johnson et al<sup>38</sup> study evaluated the role of optical coherence tomography in differentiating optic disc edema (ODE) due to papilledema and other optic neuropathies from optic nerve head drusen (ONHD) and observed an elevated optic nerve head with smooth internal contour and subretinal hyporeflective space (SHYPS) with recumbent "lazy V" pattern. Optic nerve head drusen displayed a "lumpy bumpy" internal optic nerve contour and a rapid decline in SHYPS.

#### Prognostic role:

Following the peripapillary retinal nerve fiber layer (RNFL) thickness over different visits provides a quantitative and sensitive measurement of changes in the papilledema. There is significant variability even among experts in grading papilledema based on the Frisén scale, and therefore the peripapillary RNFL is easier to follow objectively for change, especially when the patient is seen by different care providers.<sup>28-30</sup> Figure 3 reveals RNFL thickness measurement using optic disc cube (200 × 200 volume scans) centred on the optic disc in SD-OCT.

A reduction in peripapillary RNFL thickness in the eyes of IIH patients can be either a result of improving papilledema or worsening axonal loss from disease progression. Combining the macular ganglion cell layer (GCL) thickness with the

peripapillary RNFL thickness allows one to evaluate for optic neuropathy in the presence of papilledema.<sup>31-34</sup> Successful treatment with protection of neuroaxonal structure will cause a reduction in the peripapillary RNFL thickness with a preserved macular GCL thickness. However, a concordant reduction in the RNFL thickness and macular GCL thickness indicates worsening optic neuropathy and could be an indication of treatment failure or fulminant IIH.<sup>32</sup> Figure 4 reveals GCL-IPL thickness measurement using Macular cube (512 × 128 volume scans) centred on the fovea.

OCT can detect retinal causes of vision loss from IIH, such as subretinal fluid or choroidal folds.<sup>27,30,35</sup> This application has an important clinical role, because patients with decreased vision from optic neuropathy secondary to severe papilledema require aggressive treatment, while decreased vision due to subretinal fluid or choroidal folds is typically more benign.

#### Management

Goal of treatment in IIH is:

- Treatment of underlying disease.
- Preservation of vision.
- Treatment of symptoms (usually headache).

Hence, the management must be tailored according to the severity of symptoms and urgency of treatment.

First step in management of IIH is treatment of conditions such as obesity, sleep apnea, causative medications and venous sinus thrombosis.<sup>39</sup> Poor visual prognosis is associated with high-grade papilledema, severe and rapidly progressive vision loss, macular oedema, venous sinus thrombosis, and systemic hypertension require a more aggressive treatment.<sup>40</sup>

#### Conservative management

Observation is indicated in an asymptomatic patient presenting with papilledema.

#### Weight loss:

All patients of IIH with BMI > 30kg/m<sup>2</sup> should be counselled for weight reduction. Studies have shown that reduction of 6% of body weight is helpful in reduction of papilledema and discontinuation of systemic treatment.<sup>41,42</sup> Diet control and exercise is one of the most effective way to assist a patient in weight loss. Bariatric surgery has been shown to be beneficial in IIH, but it can cause anastomotic leaks, small bowel obstruction, malabsorption, and gastrointestinal bleeding.<sup>43-45</sup>

#### Medical management

In a patient with good visual acuity and with primary complaints of headache, medical management is indicated.

#### 1. Carbonic anhydrase inhibitors:

Carbonic anhydrase inhibitors (CAI), like acetazolamide are the treatment of choice. (46) The IIH treatment trial is a multicenter, double-blind, placebo-controlled North American clinical trial that reported the use of acetazolamide with a low-sodium weight-reduction diet compared with

diet alone resulted in modest improvement in visual field function in patients with mild visual loss. The improvement was more in patients with moderate to high grade papilledema. The greatest effect was in the first month. The IIHTT also reported improved quality of life outcomes at 6 months with acetazolamide.<sup>47</sup>

Carbonic anhydrase inhibitors like acetazolamide and methazolamide present in the choroid plexus, decrease CSF production and also act as mild diuretics. Acetazolamide in adult patients is usually started at 1 g daily (250 mg QID or 500 mg BID), with a maximum recommended daily dose of 4 g. Adverse effects of acetazolamide include paraesthesias, lethargy and altered taste. Hypokalemia is an important adverse effect and hence electrolytes must be monitored.

### 2. Topiramate:

Topiramate is an antiepileptic drug, it inhibits carbonic anhydrase and can suppress appetite. Topiramate has proven to have similar effects like acetazolamide in terms of papilledema and headache with greater reduction in weight.<sup>48</sup>

The dose of topiramate in IIH is from 25 mg to 50 mg bd. But the drug has side effects like depression, cognitive slowing and potential teratogenic risks.

### 3. NSAIDs:

Non-steroidal anti-inflammatory drugs like paracetamol may be used for headache. Indomethacin may have some added advantage due to its effect of reducing ICP.<sup>49</sup>

## Surgical management

Indications of surgical management:

- Acute or rapidly progressive optic neuropathy.
- Medical treatment failure.

The commonly performed surgical procedures include:

- Cerebrospinal fluid diversion using ventriculo-peritoneal, ventriculoatrial or lumboperitoneal shunt
- Optic nerve sheath decompression.

Cerebrospinal fluid shunting is the most widely performed surgical treatment for IIH.<sup>50</sup> Shunting results in rapid normalization of the ICP, resolution of papilledema, and improvement of vision and headache. Ventriculo-peritoneal diversion procedure has lower reported revision rate, so it is the preferred CSF diversion procedure.<sup>51</sup> Complications of the shunt are abdominal pain, shunt obstruction, low pressure headaches and subdural hematoma.<sup>52,53</sup> All shunt procedures have a high long-term failure rate and often need revision because of obstruction or failure.

Optic nerve sheath decompression (ONSD) is an effective treatment in patients with papilledema and severe visual loss but not an effective treatment for headache. ONSD rapidly decreases papilledema and bilateral improvement in visual function is seen in many cases.<sup>54</sup> A recent meta-analysis was done in 712 patients who underwent optic nerve sheath fenestration, 59% had improvement in vision, 44% had resolution of headache and 80% had reduction in papilledema.<sup>55</sup> This procedure causes local reduction in

pressure on the nerve by lowering the intrasheath pressure and in long-term results in fibrous scar formation between the dura and the optic nerve, thus protecting the anterior optic nerve from ICP. Patients should be kept under close follow-up after ONSD, as long-term visual deterioration may still occur.<sup>56</sup>

Temporary complications of optic nerve sheath fenestration are anisocoria, double vision, optic nerve head hemorrhages. Very rarely, it can cause traumatic optic neuropathy, branch and central retinal artery occlusion.<sup>55</sup>

Venous sinus stenting can also be tried in patients with IIH, as stenosis of distal transverse sinus is related to IIH.

## Conclusion

IIH is a syndrome characterised by increased intracranial pressure of unknown cause, leading to severe headache, papilledema and visual disturbances. There has been no consensus regarding the exact pathogenesis of IIH and investigative tool to monitor the progression and efficacy of treatment. Hence, a systematic approach with documentation of the change in the structural parameters of optic nerve with the help of OCT can help us to better monitor and prognosticate a case of IIH.

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