World Journal of Pharmaceutical Science and Research
www.wjpsronline.com
Review Article
ISSN: 2583-6579
SJIF Impact Factor: 5.111
Year - 2025
Volume: 4; Issue: 5
Page: 45-60

IDIOPATHIC INTRACRANIAL HYPERTENSION

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Article Received: 7 August 2025 | | Article Revised: 28 August 2025 | | Article Accepted: 18 September 2025

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DOI: https://doi.org/10.5281/zenodo.17233895

How to cite this Article: Dr. Shaik Salma, Sk. Sheema, M. Kavya, G. Yagna Sree Aadi Reddy, A. Divya, P. Ramya and Dr. S. Nivedhitha. (2025) IDIOPATHIC INTRACRANIAL HYPERTENSION. World Journal of Pharmaceutical Science and Research, 4(5), 45-60. https://doi.org/10.5281/zenodo.17233895

ABSTRACT

Idiopathic intracranial hypertension (IIH) is a neurological disorder characterized by elevated intracranial pressure (ICP) due to accumulation of Cerebro spinal fluid (CSF) within the skull without an identifiable cause such as tumor or hydrocephalus. IIH is also known as pseudotumor cerebri. This condition mainly affected in women and obesity population. The exact etiology remains unclear but obesity, hormonal changes and medications like antibiotics are recognized as risk factors. IIH predominantly affects the brain and optic nerves, leading to symptoms such as headache, papilledema, visual disturbances which can result in permanent vision loss if untreated. Management strategies mainly focus on reducing ICP, maintaining eye vision and weight reduction. The first line therapy includes weight loss and medications like acetazolamide and Topiramate and in severe cases, surgical interventions including optic nerve sheath fenestration or CSF shunting. Lifestyle modifications, including weight loss through regular exercise, can be beneficial in managing patients with IIH. Early diagnosis and timely treatment are crucial to prevent irreversible visual impairment and improve patient outcomes.

KEY WORDS: Idiopathic intracranial hypertension (IIH), obesity, vision loss, headache, Intracranial pressure (ICP), weight loss.

INTRODUCTION

Idiopathic intracranial hypertension (IIH) primarily affects optic nerve and cerebral venous sinuses within the brain and skull, rather than the specific region of brain parenchyma. This condition is characterized by elevated intracranial pressure (ICP) due to a buildup of cerebrospinal fluid (CSF) within the skull. CSF acts as a protective layer around brain and spinal cord. It primarily consists of 99 % water and 1% is composed of proteins, glucose, electrolytes and other substances like immunoglobulins, mononuclear cells and neurotransmitters. The normal CSF pressure ranges from 70-180 mm H2O (millimeters of water) which is equivalent to 8-15 mm Hg (millimeters of mercury). In

idiopathic intracranial hypertension the CSF pressure is greater than 250 mm H2O in adults and 200 mm H2O in children indicates increased intracranial pressure.[1] Idiopathic intracranial hypertension (IIH) is also known as Pseudotumor cerebri, characterized by increased pressure inside the skull without any tumour, abnormal brain structure, or issues with blood vessels and all other body systems (like metabolism, hormones and blood) are normal.[2] IIH is a rare condition where the pressure inside the skull is abnormally high. The increased pressure can put strain on optic nerve, leading to vision problems. The effects of IIH on the visual system may cause sudden great damage which results in visual impairment.[3]

EPIDEMIOLOGY

The global incidence of idiopathic intracranial hypertension (IIH) varies significantly and is closely linked to obesity rates in different populations. In regions where obesity prevalence is below 10%, the estimated incidence is 0.14 per 100,000 people annually, but this rises sharply to 1.48 per 100,000 in populations with obesity rates exceeding 20% .[4] Recent UK-based studies reinforce the connection between obesity, socioeconomic deprivation, and IIH. For example, in Wales, where obesity rates climbed from 29% in 2003 to 40% in 2017, IIH prevalence surged six-fold (to 76 per 100,000), while incidence tripled (7.8 per 100,000 annually). The highest rates were observed in obese women, reaching 180 per 100,000 in prevalence and 23.5 per 100,000 in incidence.[5] In towns and cities throughout England, people have observed very similar patterns emerging.[6,7] Notably, childhood cases account for 30% of male IIH diagnoses but only 10% of female cases. Interestingly, while obesity affects both genders, the link to deprivation is seen only in women.[5,6]

Subgroup analyses reveal that IIH, while uncommon, occurs at a significantly higher rate in women. The incidence is between 0.65 and 10.3 cases per 100,000 people, with women facing a risk that is 2.2 to 44 times greater than that of men. In Western countries, the annual incidence is 0.9/100,000 overall but rises to 3.5/100,000 among women aged 15-44.[8,9] The incidence of IIH varies significantly across the world,with rates far lower in Japan than in Libya.[10,11] The increase in IIH diagnoses is closely linked to the global obesity epidemic, a key risk factor for developing the condition.[9] A U.S. study (Durcan et al.) found an incidence of 19/100,000 in obese women (20-44 years, ≥20% above ideal weight), compared to 0.9/100,000 in the general population.[9] Multiple studies confirm that most IIH patients are obese women of childbearing age.[11,14] with an average diagnosis age of 30. Although less common, IIH can also occur in children, men, and older adults. After puberty, obese females remain the highest-risk group, mirroring adult-onset patterns.

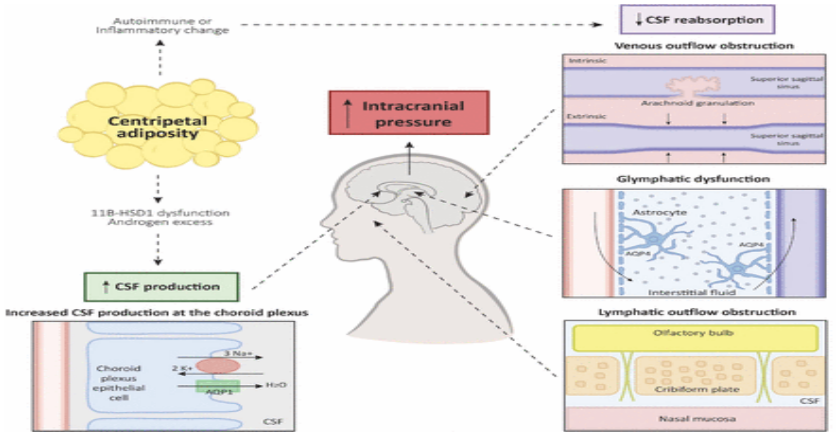


Figure 1: Represents the Pathophysiological Mechanisms of Idiopathic Intracranial Hypertension (IIH).

PATHOPHYSIOLOGY

1. CSF PRODUCTION & HYPERSECRETION

Dysfunctional epithelial cells & water permeable channels within the choroid plexus, indicates the rise in volume directly.

⇩

This results in overall cerebrospinal fluid production.[15]

2. ALTERED CSF REABSORPTION

Factors like impaired drainage through arachnoid granulations, venous sinus stenosis & potential involvement of the glymphatic system & conditions like obesity, hormonal imbalance &certain medications contribute to reduced reabsorption of CSF which results in excessive production of CSF.

3. ARACHNOID GRANULATION

The barrier between the subarachnoid spaces and venous sinuses results in hydrostatic gradients which increases the pressure within the subarachnoid spaces this causes CSF reabsorption through arachnoid granulation.[16,17] Intracranial Pressure (ICP) remains normal despite of variation of arachnoid granulations in their size, number & location.[18] Arachnoid Granulations may play a bigger role in regulating CSF pressure than in actual fluid reabsorption. This suggests their importance in CSF physiology has likely been overstated.

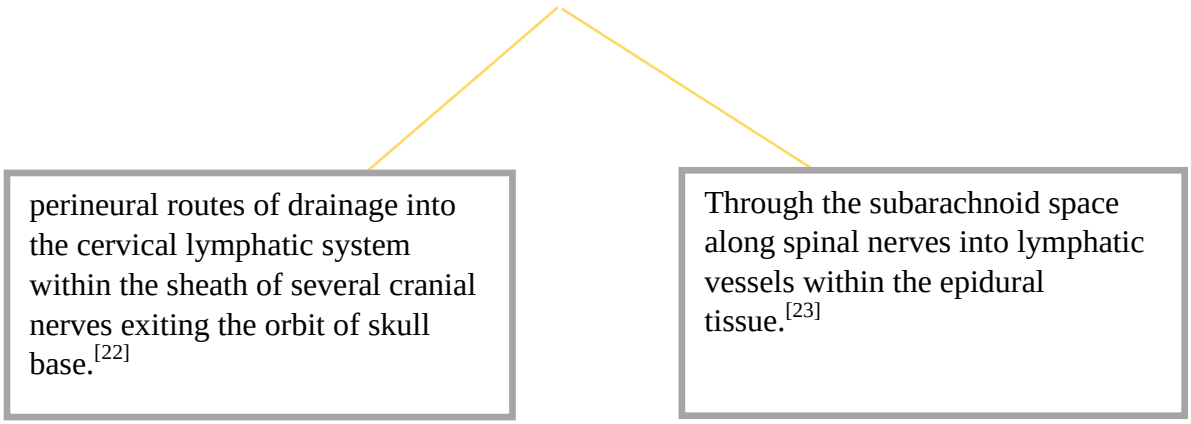
4. VENOUS SYSTEM ABNORMALITIES

The enlarged arachnoid granulations cause stenoses which results in anatomical irregularities in the venous sinuses (especially the transverse sinuses). This narrowed sinus reduces blood flow out of the brain, raising central venous pressure.[19,20] This disrupts the balance between CSF and venous pressure, potentially causing CSF build up in brain`s fluid space. There is a clear link between high venous pressure and high CSF pressure but it`s unclear if venous stenosis causes IIH or is a result of high ICP.

5. LYMPHATIC OUTFLOW PATHWAY

The lymphatic outflow pathway offers an alternative mechanism for reabsorption and opposes the historical belief that the CNS lacked a functioning lymphatic drainage system.[21]

CSF drainage into the extracranial lymphatic system through 2 mechanisms



6. GLYMPHATIC SYSTEM

The glymphatic system is a recently identified central nervous system (CNS) fluid transport pathway that connects existing routes, offering an additional mechanism for cerebrospinal fluid (CSF) drainage and waste clearance.[24] Functionally, it involves a continuous exchange between CSF and interstitial fluid within the brain tissue. CSF primarily enters via the subarachnoid space, flowing into perivascular spaces surrounding cerebral arteries and veins. The critical exchange occurs across a specialized glial barrier formed by astrocytic end feet enveloping the blood vessels, enabled by a high density of AQP4 (Aquaporin 4) water channels.[24,25] Following this glymphatic exchange, solutes are supposed to drain into the perivenous space This fluid eventually leaves the brain through one of three primary routes: it is absorbed back into the bloodstream, channelled into the body's lymphatic system, or reabsorbed into the brain's ventricles.. Given its vital role in clearing metabolic waste, dysfunction of the glymphatic system is implicated in the pathogenesis of various neurological diseases.[26]

7. OBESITY AND NEUROENDOCRINE DYSFUNCTION

The risk of IIH is significantly higher in individuals like overweight or obese, with increased BMI (Body Mass Index) over 40kg/m², which increases disease severity and higher recurrence rates.[27,28,29] Weight loss has been shown to improve symptoms, reducing ICP, alleviating papilledema, enhancing visual function and decreasing headaches.[30] Despite the association between obesity and IIH the exact mechanism remains unclear, suggesting multifactorial causes, given that many obese individuals do not develop the condition. The mechanism which represents increased obesity and which causes intra-abdominal pressure, leading to elevated central venous pressure and impaired cranial venous drainage.[31] The fat distribution has been considered as a potential factor, where in IIH patients had more lower-body fat compared to obese individuals.[32] Obesity is associated with chronic inflammation marked by increased levels of pro inflammatory cytokines, chemokines, adipokines and hormones. The elevated CCL2 levels (C-C motif chemokine ligand 2) in CSF of IIH patients after adjusting for BMI, but larger studies found no significant difference in inflammatory markers between IIH patients and obese patients.[33]

SIGNS AND SYMPTOMS

- Headache associated with nausea and vomiting.
- Persistent pain in neck and shoulders.[34]
- Pulsatile tinnitus.[34,35]
- Numbness of extremities.
- Loss of smell & coordination can be seen rarely.[34]
- The compression of ABDUCENS NERVE (6^{th} nerve) palsy leads to increased pressure which results in horizontal double vision in the affected site.
- Eye movements are disorganized due to damage of OCCULOMOTOR & TROCHLEAR NERVE (3^{rd} and 4^{th} nerve).[36,37]
- The damage of FACIAL NERVE (7^{th} cranial nerve) can lead to complete or partial weakness in the facial muscles, affecting one or both sides of the face and impairing facial expressions.[34]
- Papilledema.

CAUSES OF IIH

The cause of Idiopathic Intracranial Hypertension is unknown, which is what the word 'idiopathic' signifies. The precise trigger for IIH is unknown, but physicians have identified clear patterns that point to certain risk factors. For many,

especially women in their childbearing years, carrying extra weight is the single biggest risk factor. Certain medications can also play a role, like high-dose vitamin A treatments used for severe acne or some long-term antibiotic regimens. Other factors include recent weight gain and certain endocrine conditions.[38]

RISK FACTORS OF IIH

Highly likely risk factors

1. Female Gender

- A key risk factor is gender, as more than 90% of IIH cases occur in women. In older children, the condition typically emerges after puberty and primarily affects females.[39] This pattern implies that hormonal influences contribute to the disease's development. Additionally, sex hormones can be influenced by adipose tissue and obesity, which may further alter hormonal balance.

2. Obesity

- Obesity is a significant risk factor for IIH, though the exact physiological connection between the two remains unclear. Higher BMI levels correlate with an increased likelihood of developing IIH and related vision impairment. Weight reduction has proven to be an effective management strategy for IIH. While lifestyle changes offer some support in sustaining weight loss over time, certain commercial weight loss programs may also contribute. Emerging anti-obesity medications could expand future treatment options for IIH.

3. Endocrine disorders

- Several endocrine disorders have been convincingly linked to the development of intracranial hypertension. These include adrenal insufficiency (Addison’s disease), hypoparathyroidism, and the administration of growth hormone therapy in children.

4. Nutritional disorders

Certain vitamin A derivatives, including isotretinoin (used in acne treatment) and all-trans retinoic acid (employed in acute promyelocytic leukaemia therapy), have also been linked to intracranial hypertension.[40, 41] Given the well-established connection between hypervitaminosis A and elevated intracranial pressure, researchers have proposed that dysregulated vitamin A metabolism may play a role in the pathogenesis of idiopathic intracranial hypertension (IIH).[42] Recent studies provide a compelling clue that individuals with IIH frequently have an excess of vitamin A in their spinal fluid. This occurs naturally, not just as a result of taking supplements, pointing to a potential problem with how their bodies regulate the vitamin.[43] One proposed mechanism suggests that excessive vitamin A may overstimulate retinoic acid receptor alpha (RARα) in the central nervous system, leading to impaired CSF reabsorption and subsequent increases in intracranial pressure.[44,45] Although issues with vitamin A processing may contribute to IIH, the fact that women are disproportionately affected points to additional causes. Since no gender difference exists in how vitamin A is metabolized, other biological or environmental factors likely explain why IIH is more prevalent in women.

❖ **Probable risk factors**

Chlordecone

- Chlordecone, an organochlorine pesticide, has been associated with Idiopathic Intracranial Hypertension (IIH). Occupational exposure to the pesticide chlordecone led to severe poisoning. The chemical accumulated in workers'

blood and fat tissue, causing a dangerous increase in brain pressure and impairing the flow of spinal fluid. This evidence points to a possible cause-and-effect link between chlordecone exposure and the onset of IIH.

- Ketoprofen or indomethacin in Bartter's syndrome and thyroid replacement therapy in hypothyroid children, tetracycline and its derivatives and uraemia are other probable risk factors for development of IIH.[46]

Possible risk factors

Systemic lupus erythematous

- **Immune-mediated damage to arachnoid villi:** This may impair cerebrospinal fluid (CSF) reabsorption, contributing to elevated intracranial pressure.
- **Hypercoagulable state:** In some SLE patients, an increased tendency for blood clot formation could result in cerebral venous thrombosis, potentially leading to intracranial hypertension.
- **Steroid withdrawal:** The onset or exacerbation of IIH in SLE patients has sometimes been linked to steroid withdrawal, indicating it may act as a trigger independently of SLE-related inflammation.[47]

Hypovitaminosis

- Vitamin A deficiency may cause fibrosis in the arachnoid villi, structures that regulate CSF absorption, leading to reduced fluid drainage and elevated intracranial pressure.

- Sleep apnea, amiodarone, iron deficiency anaemia, Lithium carbonate, Nalidixic acid, sarcoidosis, sulfa antibiotics, hypophosphatasia are other possible risk factors of IIH.

Unlikely or Unproven risk factors of IIH

a) Corticosteroid intake
b) Hyperthyroidism
c) Menarche
d) Arterial hypertension
e) Menstrual irregularities
f) Multivitamin intake
g) Oral contraceptive use
h) Pregnancy

COMPLICATIONS OF IIH

❖ **Papilledema**

- Papilledema occurs when elevated pressure on the optic nerve (linking the eye and brain) causes swelling. Choroidal folds, retinal haemorrhages & subretinal neovascularization are other ocular complications.

❖ **Headaches**

- People with IIH often experience persistent and severe headaches that can feel like a throbbing pain, typically worsening in the mornings or when straining, such as during coughing, sneezing, or bowel movements. Headaches often occur on a daily basis and are the leading cause of disability in nearly 90% of patients.[48]

❖ **Pulsatile tinnitus**

➢ Some people with IIH may hear a rhythmic or whooshing sound in their ears, often in sync with their heartbeat. This can be quite bothersome and is known as pulsatile tinnitus.

❖ **Diplopia**

➢ IIH can cause double vision when pressure on certain nerves disrupts normal eye movement, making it hard to focus and see clearly.

DIAGNOSIS

1. CLINICAL EXAMINATION

Patients suspected of having Idiopathic Intracranial Hypertension (IIH) should undergo a thorough neurological assessment. This includes evaluating the cranial nerves (especially the 2nd, 3rd, 4th, and 6th) and examining the upper and lower limbs to detect the other causes of increased intracranial pressure.

- The assessment includes

1. Papilledema - graded using the Modified Frisen Scale.
2. Blind spot enlargement.
3. Peripheral visual field loss - which may progress to central defects in advanced cases or with coexisting macular pathology.
4. Horizontal diplopia- due to non-localizing sixth nerve palsy.

2. DIFFERENTIAL DIAGNOSIS

When evaluating a patient with elevated intracranial pressure, it's essential to consider secondary causes, including:

1. Intracranial masses (e.g., tumours, abscesses, or other space-occupying lesions).
2. Excessive CSF production (e.g., due to choroid plexus papilloma).
3. Impaired CSF absorption (e.g., from arachnoid granulation scarring after meningitis or subarachnoid haemorrhage).
4. Venous drainage obstruction (e.g., venous sinus thrombosis, jugular vein compression, or post-neck surgery complications).
5. Severe hypertension (malignant hypertension).
6. Obstructive sleep apnea.

3. INVESTIGATIONS

❖ **MRI, MRV & CT: {MAGNETIC RESONANCE IMAGING, MAGNETIC RESONANCE VENOGRAPHY & COMPUTED TOMOGRAPHY}**

➢ MRI and MRV are valuable diagnostic tools for Idiopathic Intracranial Hypertension (IIH). They help exclude secondary causes and identify specific imaging markers associated with high intracranial pressure (ICP), such as:

a) Empty sella turcica
b) Optic nerve protrusion
c) Vertical tortuosity & Posterior globe flattening
d) Optic nerve sheath distension & transverse sinus stenosis

❖ **LUMBAR PUNCTURE**

➢ A lumbar puncture (LP) plays a key role in diagnosing Idiopathic Intracranial Hypertension (IIH) by assessing cerebrospinal fluid (CSF) pressure and its composition. This procedure helps verify elevated intracranial pressure (ICP) while excluding other possible causes, such as infections or tumours. In some patients, an LP may also offer temporary relief from headaches and reduce papilledema.

❖ **BLOOD PRESSURE**

➢ Malignant hypertension involves dangerously high blood pressure (diastolic ≥120 mmHg or systolic ≥180 mmHg) and may cause papilledema, a symptom shared with IIH. Blood pressure measurement is essential to differentiate between these two critical conditions.

❖ **OPENING PRESSURE**

➢ In the diagnosis of Idiopathic Intracranial Hypertension (IIH), measuring cerebrospinal fluid (CSF) opening pressure via lumbar puncture plays a pivotal role. A key diagnostic criterion is an elevated opening pressure, generally exceeding 25 cm H2O, particularly when supported by compatible clinical and imaging findings. This assessment is essential for distinguishing IIH from other disorders with similar presentations.

❖ **PERIMETRY**

➢ Automated static perimetry plays a key role in evaluating IIH (Idiopathic Intracranial Hypertension) by detecting visual field impairments resulting from papilledema. This method assesses peripheral vision, frequently compromised in IIH, and identifies the severity and pattern of vision loss. Regular visual field testing is vital for tracking disease development and determining the efficacy of treatment.

❖ **OPTICAL COHERENCE TOMOGRAPHY**

1. Detecting papilledema

➢ Optical coherence tomography (OCT) helps identify and measure optic disc swelling (papilledema), a hallmark of IIH, by assessing retinal nerve fibre layer (RNFL) thickness and other optic nerve head characteristics. Increased intracranial pressure (ICP) in IIH leads to optic nerve head swelling, which OCT can clearly visualize and quantify.

2. Differentiating papilledema from pseudo papilledema

➢ OCT aids in distinguishing true papilledema (optic disc swelling caused by increased intracranial pressure) from pseudo papilledema (a benign mimic with a similar clinical presentation). By analysing structural differences, OCT provides objective measurements to confirm or rule out elevated ICP-related swelling.

MANAGEMENT

- **IIH management includes following techniques**

1. CONSERVATIVE MANAGEMENT

The conservative treatment of idiopathic intracranial hypertension (IIH) emphasizes non-surgical methods to lower intracranial pressure (ICP), alleviate symptoms such as headaches and visual disturbances, and prevent long-term vision damage. This approach usually involves personalized strategies based on the patient's specific condition and risk factors.

- **Weight Management**

A key aspect of non-surgical IIH treatment is weight loss, especially for overweight or obese patients. Research shows that even modest weight reduction can lead to a significant decrease in intracranial pressure and symptom relief.[49] Approaches may include dietary modifications (such as balanced nutrition and controlled portions), regular exercise, and lifestyle changes. Emerging evidence also suggests that medications like GLP-1 agonists which may influence cerebrospinal fluid production could play a role in supporting weight loss for IIH patients.

2. MEDICAL MANAGEMENT

The pharmacological treatment for Idiopathic Intracranial Hypertension (IIH) mainly aims to lower intracranial pressure (ICP) and protect vision. Key medications include.

Carbonic anhydrase inhibitors

Acetazolamide

- Acetazolamide, a carbonic anhydrase inhibitor, is the primary drug for managing idiopathic intracranial hypertension (IIH). By inhibiting carbonic anhydrase, it reduces cerebrospinal fluid (CSF) production in the choroid plexus, thereby lowering intracranial pressure (ICP). This mechanism helps relieve IIH symptoms and protects against potential vision deterioration. It is the preferred initial medication, and it can enhance visual field performance and quality of life in patients with mild vision impairment. The treatment typically begins with a dose of 250–500 mg twice daily, which may be adjusted up to a maximum of 4 g per day. Regular checks of serum electrolytes and venous blood gas levels are recommended during therapy.

Topiramate

- Topiramate is prescribed for Idiopathic Intracranial Hypertension (IIH) mainly because it helps lower intracranial pressure and alleviates related headaches. It helps manage IIH by lowering intracranial pressure (through carbonic anhydrase inhibition) and supporting weight loss, an important aspect of IIH treatment. An open-label study found that topiramate (50–200 mg daily) was as effective as acetazolamide.[50,51] Since obesity and migraines are common in IIH, topiramate could be a highly useful treatment option.

Furosemide [Diuretic]

- Furosemide, a loop diuretic, can help manage Idiopathic Intracranial Hypertension (IIH) by decreasing intracranial pressure (ICP).[52] It works by promoting diuresis and reducing sodium movement into the brain, which lowers cerebrospinal fluid (CSF) production. Although acetazolamide is typically the preferred treatment, furosemide serves as an alternative for patients who cannot tolerate or have contraindications to acetazolamide. The medication furosemide might be prescribed, with a maximum dose of 40mg taken twice daily.

3. SURGICAL MANAGEMENT

Surgical treatment offers a viable option to protect vision when rapid deterioration is imminent. In the short term, techniques like CSF diversion and optic nerve sheath fenestration (ONSF) have been successfully used.

A) CSF diversion surgery

- Neurosurgical CSF diversion is the primary treatment option. Both ventriculoperitoneal (VPS) and lumboperitoneal shunts (LPS) have shown effectiveness in managing progressive vision loss.[53] Surgical CSF

(cerebrospinal fluid) diversion involves redirecting CSF flow away from the brain and spinal cord to other body areas. This approach is mainly used to address hydrocephalus (abnormal fluid buildup in the brain) and control elevated intracranial pressure, frequently after traumatic brain injuries. Standard methods include shunt placement (inserting a drainage tube) and endoscopic techniques.

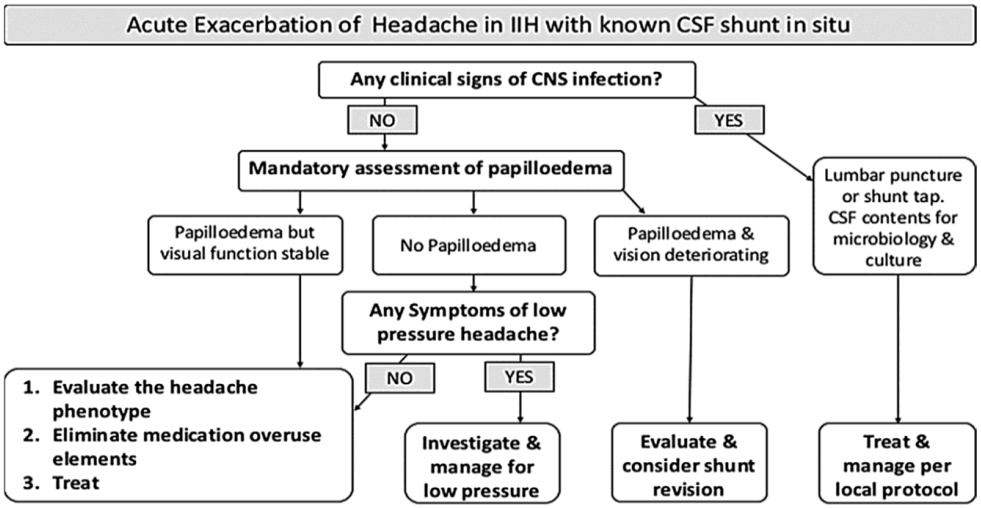


Figure 2: Presenting acute exacerbation of headache in IIH with known CSF shunt in situ.

B) Optic nerve sheath fenestration {ONSF}

- Optic nerve sheath fenestration (ONSF) is a safer surgical option with minimal complications and no documented fatalities. ONSF is an operation performed to relieve vision-threatening papilledema in patients with idiopathic intracranial hypertension (IIH). Therefore, ONSF is preferred by many experts when considering the re-approaches after the shunt placement.[54]

C) Venous sinus stenting {VSS}

- Venous sinus stenting (VSS) is a less invasive surgical technique for managing idiopathic intracranial hypertension (IIH).
- It has emerged as a viable alternative, especially for patients who fail medical therapy or develop severe vision-related complications.
- VSS is a procedure where a tiny, flexible stent is placed into a narrowed or obstructed venous sinus -the vein responsible for draining blood from the brain. By expanding the vein, the stent enhances blood flow and decreases pressure, which can help relieve IIH-related symptoms, including headaches, vision problems, and optic nerve swelling (papilledema).

D) Bariatric surgery

- Bariatric surgery is an effective intervention for Idiopathic Intracranial Hypertension (IIH) in patients with severe obesity. By facilitating substantial and long-term weight loss, the procedure lowers intracranial pressure (ICP), alleviating IIH symptoms. Research indicates benefits such as reduced papilledema and decreased headache intensity.

- **CONSIDERATIONS**
- Although bariatric surgery shows promise in treating IIH, patient selection is crucial—candidates must meet the BMI requirements for the procedure. Additionally, as a major surgical intervention, it carries inherent risks and potential complications that should be carefully weighed.

NON-PHARMACOLOGICAL TREATMENT

While lifestyle changes like a healthier diet, regular exercise, and stress management are foundational to treatment, it's important to have realistic expectations. For many people, this dedicated approach leads to a modest weight loss of 5-10%. While this is a significant and healthy achievement, it may not, on its own, be enough to achieve full remission for everyone.

NUTRITION

Effective management of Idiopathic Intracranial Hypertension (IIH) heavily relies on weight loss.[29] Research indicates that individuals with a BMI exceeding 40 face a greater risk of severe papilledema, and the potential for significant vision loss increases proportionally with higher BMI.[28] While earlier studies indicated that a 6% reduction in body weight could lead to papilledema remission, newer evidence suggests a more substantial loss of 15-25% may be necessary.

For successful weight management, the following nutritional strategies are recommended:

- A short-term calorie deficit of 500-1000 kcal per day can be effective for initial, modest weight loss.
- Adopting a diet focused on low-energy-dense foods (such as fruits, vegetables, whole grains, lean meats, and low-fat dairy) and practicing portion control can promote a feeling of fullness while reducing overall calorie intake.
- It may also be beneficial to limit foods rich in vitamin A (e.g., carrots, sweet potatoes, leafy greens) and tyramine (e.g., aged cheeses, cured meats, and certain alcoholic beverages).[37]

EXERCISE

Incorporating physical activity is a valuable component of a weight loss strategy for IIH. Engaging in resistance training or light-to-moderate aerobic exercise can yield positive results. However, since no studies have examined exercise in isolation for IIH, it should always be combined with dietary modifications for maximum effect.

A critical consideration is that intense physical exertion can temporarily raise intracranial pressure, potentially worsening symptoms like headaches or causing shortness of breath. Patients experiencing these issues after moderate-to-high-intensity workouts should proceed with caution and consider switching to lower-impact forms of exercise.

Additional lifestyle modifications for symptom management

Headache management in IIH can be supported by several key lifestyle changes.[53]

- Reduce Stimulants: Limit caffeine intake.
- Promote Hydration: Ensure adequate fluid consumption.
- Improve Sleep: Practice good sleep hygiene.
- Manage Stress: Utilize techniques like mindfulness, yoga, or cognitive behavioural therapy (CBT).
- Screen for Comorbidities: Evaluate for obstructive sleep apnea, considering a referral for a sleep study if indicated.

CONCLUSION

Idiopathic intracranial hypertension (IIH) is a complex neurological disorder characterized by elevated intracranial pressure that poses a significant threat to vision. While its precise etiology remains elusive, the pathophysiological mechanisms are multifactorial, involving a complex interplay of altered cerebrospinal fluid dynamics, venous abnormalities, and the recently elucidated glymphatic system. The most potent and well-established risk factor is obesity, particularly in women of reproductive age, with the global rise in obesity rates directly generating an increase in IIH incidence. The clinical presentation, dominated by headaches, papilledema, and visual disturbances. Diagnosis is one of exclusion, relying on a combination of neurological examination, neuroimaging (MRI/MRV), and confirmation of elevated CSF opening pressure via lumbar puncture. The primary management goals are to alleviate symptoms, reduce intracranial pressure, and, most critically, prevent permanent vision loss. First-line treatment depends on weight loss and pharmacotherapy with acetazolamide. In refractory or severe cases, surgical interventions become necessary to protect the optic nerve. Ultimately, early diagnosis and timely intervention are paramount to preserving visual function and improving patient quality of life. Given the strong link to obesity, public health initiatives aimed at weight management and increased awareness among healthcare providers serving obese populations are crucial for mitigating the impact of this potentially blinding condition. Future research must continue to unravel the underlying molecular and hormonal pathways to develop more targeted and effective therapies.

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