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REVIEW

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Idiopathic intracranial hypertension: from concise history to current management

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Abstract

Background Idiopathic intracranial hypertension (IIH), known as benign intracranial hypertension (BIH) since the report of Quincke in 1893, was described as a rare disorder of elevated intracranial hypertension with normal cerebrospinal fluid (CSF) and without any pathology. This review describes the history, pathophysiology, management, and prognosis of IIH; hence, the clinician can provide treatment based on the known possible mechanisms.

Results Headache and visual obscuration are the most typical reported manifestation of IIH. The pathophysiology remains unknown; however, some theories relate to its mechanism, including obesity and metabolic dysregulation. It was diagnosed based on Friedman's criteria diagnosis. The management of IIH, consisting of conservative (control body weight), medical treatment, and surgical treatment, aims to reduce the symptoms and maintain visual function.

Conclusion The pathophysiology of IIH underlies the current approaches and management. The recurrences in 1–5 years are likely developed after treatment; thus, long-term follow-up needs to consider depending on the severity of visual loss, papilledema, and symptoms.

Keywords Idiopathic intracranial hypertension, Papilledema, Headache, Visual obscuration

Introduction

Idiopathic intracranial hypertension (IIH) is an uncommon disorder of elevated intracranial pressure with standard cerebrospinal fluid (CSF) composition and no underlying intracranial pathology. The incidence of IIH is increasing with obese cases, specifically in women of childbearing age [1]. The terminology of IIH has changed over the decades since Quincke was known as benign intracranial hypertension (BIH) in 1893 [2]. Pseudotumor cerebri was used for intracranial hypertension after no intracranial mass was identified in brain magnetic resonance imaging (MRI) [3]. The prevalence of IIH is 0.5–2 per 100,000 population, predominating females 18–45 years old [4, 5]. Children can also experience IIH, although its prevalence is rare; however, it also has increased intracranial pressure (ICP) condition without a structural cause, hydrocephalus, or meningeal inflammation. The incidence of pediatric IIH is 0.9 per 100,000 in the United States, while in Canada, it is 4 per 100,000 [6, 7]. The underlying mechanism of IIH has been unknown. Classically, ICP is constant; however, it can be impaired by disturbances in CSF flow and dynamic or molecular mechanisms [6]. Several proposed hypotheses of IIH include overproduction of CSF, outflow obstruction, increased central venous pressure, metabolism, and hormonal changes commonly found in obese patients [4, 8]. Weight gain was significantly identified to induce the signs and symptoms of IIH due to elevated intraabdominal pressure [1]. A high body mass index has been founded in 90% of patients with IIH; even a weight gain of 5–15% may increase the risk of IIH [9].

Headache is the most typical symptom of IIH; however, transient visual loss was reported in 68% of cases [10]. The type of headache caused by IIH may be varied, such as migraine or tension-type headache. Despite transient visual obscurations, it may be challenging to differentiate

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from primary cephalgia; however, it has other complaints, such as tinnitus, blurred vision, diplopia, or neck stiffness. Adults may get irritable, but children are usually conscious [6].

IIH is an exclusion diagnosis based on Friedman's criteria diagnosis. It was revised by Friedman and colleagues in 2021. Definite IIH was diagnosed in a patient with papilledema, precise neurological examination, neuroimaging, CSF composition, and opening CSF opening pressure ≥ 25 cmH_2O. The MRI may show characteristic findings in IIH: empty sella, posterior globe flattening, distention of preoptic subarachnoid space, and transverse venous sinus stenosis [10].

Symptoms of IIH are often debilitating and may cause progressive and irreversible visual loss in improper treatment. The purpose of the management of IIH is to reduce symptoms caused by increased intracranial pressure and maintain visual function using current approaches and recommendations based on the known pathophysiology of IIH [3].

The published papers for this review were collected and selected from a search of the PubMed, Mendeley, and Cochrane Libraries for all peer-reviewed articles from 2002 to date with keywords, including "idiopathic intracranial hypertension," "pseudotumor cerebri" and "benign intracranial hypertension". Other articles with essential data for clinical studies of IIH treatment were also selected and reviewed for additional information or points of view.

This review article provides the latest knowledge and points of view concerning the concise history of IIH, pathophysiology, management, and prognosis of IIH, which is essential for clinical application. We present details about each manifestation's pathophysiology and pathophysiology diagram, a few clinical points in children with IIH, and current treatment, especially combination treatment (weight loss, drug, and surgical intervention) [3].

Main text

History of IIH

IIH was previously known as BIH, pseudotumor cerebral, meningeal hydrops, or serous meningitis. BIH is the first terminology cited by Quincke in 1893 as an elevated intracranial pressure without space-occupying lesions or hydrocephalus. Sixty cases have been identified from previous literature showing an episode of extended intracranial hypertension and typical ventricle system rather than a history of infection. Other earlier terms of BIH are serous meningitis by Quincke in 1897 and Warrington in 1914, intracranial pressure without brain tumor or 'pseudotumor cerebri' in 1904 by Nonne, and in 1937 by Walter Dandy, or unknown condition by Sahs and Hyndman in 1939, Pseudotumor dan Tumor Equivalent by Warrington 1914, Bailey in 1920, pseudo abscess by Adson in 1924, and papilledema of indeterminate etiology by Yaskin in 1949. These terminologies refer to clinical signs without meningeal inflammation, clear toxin, or meningeal hydrops [2]. Corbett and Thompson, 1989 started this condition as an IIH found in 14 of 57 patients with permanent visual loss [11]. Diagnostic criteria of IIH were prepared by Dandy in 1937, modified by Smith in 1985, and refined as a pseudotumor cerebral by Friedman in 2013. Headache due to IIH is classified in the International Classification of Headache Disorders, 3rd edition, as a non-vascular intracranial disorder [12].

Pathophysiology of IIH

The underlying mechanism of IIH has been unknown; however, the significant risk factor identified is weight gain-induced signs and symptoms of IIH [1, 13]. A high body mass index has been founded in 90% of patients with IIH; even a weight gain of 5–15% may increase the risk of IIH [9]. Some proposed hypotheses of IIH are increased central venous pressure, metabolism, and hormonal changes found in obese patients (9). Both IIH and secondary intracranial hypertension may be affected by obese female patients with a typical clinical presentation [14].

Central obesity may elevate intraabdominal, pleura, cardiac filling, and central venous pressure impairing CSF flows [1, 15]. Insulin resistance and hyperleptinemia in centripetal obesity correlate with increased opening pressure in lumbar punction, nonetheless still unknown [16, 17]. Adipose tissues function in the neuroendocrine by releasing cytokines and leptin [15]. Leptin helps to maintain food cravings and weight gain. Elevated serum leptin has been known to settle in IIH. Leptin enters the choroid plexus, previously obstructing the CSF flows and increasing leptin resistance. Leptin has a prothrombotic effect that works in thrombocyte leptin receptors, consequently inducing micro thrombosis of the venous sinus [18]. This theory is still controversial, excluding found clots in the cerebral venous sinus or earlier or a family history of thrombophilia [8].

Leptin and insulin involvement reinforce the theory of metabolic dysregulation in IIH [16]. Dysregulation of an intracellular enzyme, 11beta-hydroxysteroid dehydrogenase (11β-HSD1), has been discovered in obese, although it has low levels after weight loss. The 11β-HSD1 affects CSF production and homeostasis by its expression in the choroid plexus epithelium and granulation tissues of arachnoid [18]. Even if the mechanism may not explain IIH in non-obese, however, non-obese patients (body mass index < 30 kg/m^2) have an increased risk of IIH if they have a weight gain of 5–15% [8].

Stenosis of the dural venous sinus or distal transverse cerebral sinus impairs the venous flow, induces venous hypertension, and increases intracranial hypertension. Stenosis may appear due to fibrosis caused by remodeling of the transverse sinus [1]. The mechanism remains unknown whether it arose as an incidental pathology, a secondary condition of elevated intracranial pressure related to arachnoid granulation. It has been completely relieved after a lumbar puncture or CSF shunt procedure [8]. Mechanisms of IIH are described in Fig. 1.

Other risk factors and conditions related to IIH manifestation

Based on the previous theories, obesity is the most expected IIH risk factor; however, other risk factors have been identified, such as drugs, endocrine disease, or infection. Some commonly used medications have been identified as the most strongly associated with IIH, such as vitamin A derivates, tetracycline, recombinant growth hormone, and lithium. Moreover, Rakez and colleagues 2022 reported a case of a 9-year-old boy with cytosine arabinoside treatment for acute lymphoblastic leukemia. Even though this drug was not the only cause of IIH, it may increase CSF concentrations in high doses. Postpubertal children also have the same adult risk factors of IIH, whereas, for prepubertal children, obesity and gender were not related to the disease [19]. COVID-19 infection with IIH manifestation has been reported as a mimicking condition due to cerebral venous sinus thrombosis, papillophlebitis, and meningoencephalitis [20, 21].

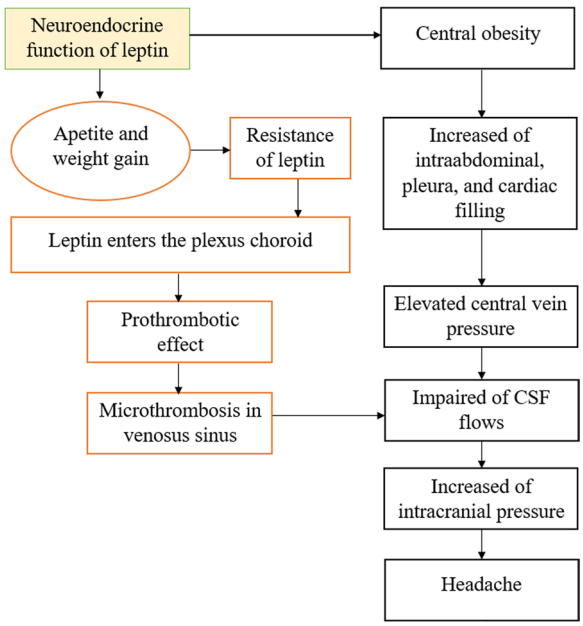

Fig. 1 Pathophysiology of idiopathic intracranial hypertension (IIH)

Moreover, endocrine diseases (thyroid dysfunction, Addison's disease, or hypoparathyroidism), levothyroxine, recombinant growth hormone, and anabolic steroids were associated with IIH. A few cases are being reported, mainly in the children age group. A systematic review of 21 patients with levothyroxine-related IIH by Datta in 2023 showed it was manifest after two months of levothyroxine replacement. It is more frequent in pediatric age and may be associated with prolonged symptom duration and higher levothyroxine doses. The mechanism is not well known; however, it was possibly related to a maladaptive response to rapid changes in thyroid-releasing hormone and vasopressin and hypercortisolism in the immediate correction by the treatment. Temporary discontinuation and watchful observation were essential management keys [22].

Moreover, IIH can be suffered in a woman's first trimester of pregnancy [23]. IIH-related post-spinal surgery cases have been reported. Even if it is relatively rare, it should be communicated as a surgery complication [24]. Anemia, especially iron deficiency anemia, causes increased platelet levels and a hypercoagulable state because of decreased iron as a regulator of thrombopoiesis; moreover, iron deficiency causes red cell deformities [25].

Clinical manifestation

Meninges and cerebral venous are pain-sensitive intracranial structures that would be irritated by vascular wall changes in IIH. Mediators of inflammation are released in the meninges (such as calcitonin gene-related peptide [CGRP], pituitary adenylate cyclase-activating polypeptide [PACAP], P substance, glutamate, and nitrite oxide. These mediators activate inflammatory cascades in the trigeminovascular system and central projections like migraine. Ophthalmic nerves project sensory impulses to the trigeminal ganglion and the second neuron in the brainstem and cervical spinal cord, then involve periaqueductal gray, locus coeruleus, and raphe magnus nucleus. Frontal and periorbital pain are commonly reported symptoms by patient IIH and enhanced by eye movement caused by distension of the optic nerve sheath due to intracranial hypertension [26].

Headache is the most typical symptom of IIH, especially in obese women. It felts progressively in the morning or during supination. Headache in IIH may be found in various types, such as pulsating headache, trigeminal neuralgia, referred pain, or migraine. This variation depends on the primary cephalgia phenotype [10]. The IIH treatment trial result showed that 67% of IIH participants have migraines without aura or probable migraines, 25% have tension-type headaches/potential tension-type headaches, and 8% have unclassified headaches.

Headache may persist after normal CSF pressure, although papilledema has been recovered and is in remission [10, 26]. The symptoms of intracranial hypertension, otherwise from adults, are subtle in pediatrics. Headache is the most common sign of pediatric IIH, followed by nausea and vomiting. Ophthalmological complaints can be present, while olfactory or cognitive dysfunction is less reported [19].

Transient visual obscuration is reported by 68% of IIH patients, caused by impairment of axoplasmic flow that promotes edema of retinal nerve fibers and short-term ischemic of optic nerves [12]. Males are more likely to experience visual loss than females; nevertheless, no significant correlation has been found [15]. Other visual symptoms are dyschromatopsia, visual field defect, and diplopia [9].

Pulsatile tinnitus can appear bilateral or unilateral, caused by turbulence of blood flows in obstructed transverse venous, high standard vascular pulse transmission, or increased CSF flows in the cochlea. 60% of IIH patients described this symptom. Other symptoms are diplopia due to abducens nerve palsy [12].

Procedures of diagnostic

Vital signs are essential to recognize malignant hypertension that provokes similar symptoms [15]. The neurological examination involves visual functions such as visual acuity, visual field, color vision, and ophthalmoscope. Acute loss of visual acuity does not frequently occur due to papilledema; however, it can be mildly diminished due to globe flattening, chorioretinal folds, or subretinal fluid in intracranial hypertension [9]. Papilledema is a merit of this disease; however, it may be challenging to identify papilledema using conventional undilated direct ophthalmoscopy because it needs the ability and considerable practice to visualize the optic disc. This condition increased referrals to neuro-ophthalmology clinics due to the unconfidence of papilledema using conventional ophthalmoscopy. Rissan and colleagues have had an intermethod and interrater validity study of fundus imaging and perimetry visual field assessment system for IIH. This system is reasonably sensitive to identifying papilledema in patients suspected of IIH [28].

Dyschromatopsia is an early sign of optic nerve dysfunction and could be detected by the Ishihara test. Visual field changes are usually not complained about by the patient and not found with confrontation visual field testing. Because it is a meaningful clinical sign, it should be investigated with automated static threshold perimetry to measure mean deviation (MD; typical < -2 decibel or dB). IIH defines mild visual loss as an MD -2 to -7 dB [9].

Moreover, extraocular movement should be investigated to find abducens nerve palsy referring to elevated intracranial pressure. The results of the ophthalmoscope in IIH are papilledema or optic atrophy in chronic IIH. Optical coherence tomography is a non-invasive 3D imaging that helps distinguish the pseudopapilledema condition. These examinations should be regular in IIH, except for abducens nerve palsy [15].

Furthermore, Friedman and colleagues' revised diagnostic criteria of pseudotumor cerebral syndrome (PTCS) in 2021 must be fulfilled to diagnose a definite IIH (Table 1). Brain MRI can demonstrate characteristic radiological findings that may allow crucial clinical information, such as elevated CSF peri-optic nerve, empty sella

Table 1 Recommended Friedman diagnosis criteria of PTCS [10]

1. Essential criteria diagnosis
a. Papilledema
b. Routine neurologic examination findings-except cranial nerve abnormalities
c. Normal neuroimaging findings: absence of hydrocephalus, structural lesion, an abnormal meninges involvement in brain MRI with/without contrast. Routine venography MRI with/without contrast if the patient is not obese patient and female
d. Normal CSF composition
e. Increased CSF opening pressure (≥ 25 cmH_2O) with correctly performed lumbar puncture
2. Diagnosis of PTCS without papilledema:
Unilateral or bilateral abducens nerve palsy with criteria b-e must be fulfilled
If there is no papilledema or abducens nerve palsy, PTCS can be suggested, but not a definite diagnosis
Recommendation of a PTCS can be made with at least three MRI findings together with criteria b-e:
• Empty sella
• Posterior globe flattening
• Distension of preoptic subarachnoid space (with/without tortuous optic nerve)
• Transverse venous sinus stenosis

MRI magnetic resonance image, *CSF* cerebrospinal fluid, *PTCS* cerebral pseudotumor syndrome

turcica, and stenosis of transverse sinuses. Those findings have high sensitivity and specificity, namely empty sella (sensitivity 80%, specificity 64%) and posterior globes flattening (specificity 97% and sensitivity 57%). These MRI findings have 64% sensitivity and 97–100% specificity [10, 29].

Management

There is no consensus on the management of IIH; however, identifying any risk factor is a vital management strategy of IIH such as obesity, drug usage, and endocrine disease. The management options are conservative treatment to control body weight and medical and surgical treatment depending on severity, visual function, comorbidity, response, and tolerability of therapy [8]. Levothyroxine-related IIH can be managed by a triple regimen, such as levothyroxine dose reduction or temporary discontinuation, CSF drainage, and carbonic anhydrase [22].

Conservative treatment

The steady reduction of 6–15% body weight over 6 months can effectively reduce or resolve papilledema [9]. Patients with a body mass index ≥ 35 kg/m^2 should perform the weight loss program and other treatments. This program would be challenging, so patients who fail to lose body weight with low calories diet, exercise, and lifestyle modification are indicated to perform bariatric surgery [10].

Medical treatment

Acetazolamide, a carbonic anhydrase enzyme inhibitor, is recommended as a therapeutic option in IIH, but no clinical trial data are available on its effectiveness. This drug plays a role in reducing CSF secretion in the choroid plexus. The IIH treatment trial conducted on 165 IIH patients in 2014 showed acetazolamide and weight loss of 6% in 6 months improved papilledema and vision function compared to IIH patients with weight control without medical treatment; moreover, this drug followed by low-sodium weight-reduction diet resulted in modest improvement in visual field function [8, 10, 30].

The optimal dose of acetazolamide has not been determined; however, the initial dose is 250–500 mg divided by two doses per day, followed by a maintenance dose of 1–2 g per day and a maximum dose of 4 g [10]. Acetazolamide is considered for IIH patients without visual decreased, while moderate vision function diminished or papilledema is recommended with acetazolamide 1000 mg every 12 h besides 1% weight loss [9]. Its side effects are paresthesia, nausea, vomiting, and diarrhea. Acetazolamide is a category C drug with teratogenic side effects obtained from animal studies. In pregnant women, the risk of spontaneous abortion is found either before or during pregnancy. No significant complications have been reported [10].

Other diuretic agents are topiramate (voltage-gated sodium, calcium inhibitor, and weak carbonic anhydrase enzyme inhibitor) show no significant difference between acetazolamide and furosemide, which was not recommended as a monotherapy. Moreover, corticosteroids are not routinely used for IIH due to unexpected side effects, namely weight gain; however, it is still suggested for progressive visual loss and fulminant IIH waiting for surgical treatment [8, 10].

A new therapy that reduces intracranial pressure faster than the previous method is still observed. Glucagon-like peptide-1 (GLP-1) agonist, exendin-4, is evident to reduce intracranial pressure. Moreover, the 11β-HSD1 (AZD4017) inhibitor can change from inactive to active cortisol, thus regulating serum cortisol that influences CSF production [10].

Surgical treatment

Some options for surgical treatment therapy for IIH include venous sinus stenting, optic nerve sheath fenestration (ONSF), ventriculoperitoneal shunting, lumbar-peritoneal shunt, and bariatric surgery. These options are conducted for a patient with fulminant IIH clinical courses, such as progressive visual diminished or severe visual loss, unresponsive to medical treatment, or extended symptoms (refractory headache). There are no guidelines on its effectiveness; however, the option of surgical treatment is determined by source availability and experiences [8, 10].

Venous sinus stenting

Venous sinus stenting is the first-line surgical intervention for refractory IIH. This intervention has high technical success, approximately 99.5%, and only 10% of repeated procedures with low significant complication rates (about 1.5%). A meta-analysis by Kalyvas and colleagues in 2021 showed that papilledema and visual function have improved by this intervention, subsequently in 87.1% and 72.7% of the IIH patients; however, it had a 2.3% severe complication rate and 11.3% failure rate. This intervention presented the best clinical outcomes (headache and visual function) with low failure and favorable complications [31, 32].

CSF diversion methods

In visual saving, CSF diversion might be performed by reducing CSF volume from a lumbar-peritoneal and ventriculoperitoneal shunt. It should be suggested if surgical intervention was likely delayed in 24–48 h [3]. The CSF diversion method improved papilledema, visual field, and headaches in 78.9%, 66.8%, and 69.8% of

cases, with a minimal complication rate of about 9.4% and a 43.4% failure rate [31].

Optic nerve sheath fenestration

By forming a fistula between the optic nerve sheath, ONSF reduces the optic nerve damage of high intracranial pressure. It is indicated in some conditions, such as minor headaches, high risk, or contraindication for CSF diversion methods [3]. Papilledema, visual field defects, and headaches could ameliorate in 90.5, 65.2% and 49.3% of cases, with a minimal severe complication rate of.2% and a 9.4% failure rate [31].

Bariatric surgery

Considering obesity is the most typical risk factor, weight loss should be recommended for IIH patients, incredibly severely obese patients. Community weight management interventions have been related to adequate weight loss by about 5%; however, it was difficult to sustain long-term weight loss, so bariatric surgery must be superior to community weight management intervention. Bariatric surgery was also excellent for decreasing intracranial pressure than weight management in a randomized clinical trial conducted by Mollan and colleagues in 2021. It also improves other symptoms, such as headaches and visual [33, 34].

Prognosis

The clinical course of IIH is not always monophasic, although it can relapse. The risk factors are related to poor visual function in IIH patients, namely race (black), male sex, obesity, anemia, obstructive sleep apnea, and acute onset of increased intracranial pressure (fulminant IIH) [8]. Recurrences were reported in 23% of IIH patients without any correlation to demographic data and clinical characteristics. 23% of pediatric patients reported relapse one year after therapy was discontinued, while 50% of adolescent and 28% of adult patients had the condition in 3 years. Therefore, follow-up is considered over a long period or even a lifetime to monitor the risk of recurrence. Patients with severe papilledema and stable vision should be controlled weekly; if papilledema is mild, the patient should be maintained every 6 months [10].

Headaches that still arise after IIH remission can be a valuable sign of recurrence, especially in patients with a headache before IIH occurred; therefore, significant clinical findings of IIH relapse are an increased CSF pressure on a lumbar puncture or papilledema [10].

Conclusions

IIH induces symptoms of increased intracranial pressure, such as headache, transient visual obscuration, diplopia, changes in the visual field, dyschromatopsia, and pulsatile tinnitus. Diminished visual functions may be temporary or permanent. The pathophysiology of IIH remains unknown; however, some theories relate to its mechanism, including obesity and metabolic dysregulation.

The management of IIH, consisting of conservative (control body weight), medical treatment, and surgical treatment, aims to reduce the symptoms and maintain visual function. The recurrences in 1–5 years are likely developed after treatment; thus, long-term follow-up needs to consider depending on the severity of visual loss, papilledema, and symptoms.

A few numbers of published articles can be missing or unreachable by authors, and missing details in several articles are a few limitations of this review article. Nevertheless, this review article includes knowledge of IIH, from history to current management. The study also includes a pathophysiology diagram of IIH.

Abbreviations

BIH	Benign intracranial hypertension
CGRP	Calcitonin gene-related peptide
CSF	Cerebrospinal fluid
IIH	Idiopathic intracranial hypertension
GLP-1	Glucagon-like peptide-1
MRI	Magnetic resonance image
PACAP	Pituitary adenylate cyclase-activating polypeptide
PTCS	Pseudotumor cerebral syndrome
11β-HSD1	11Beta-hydroxysteroid dehydrogenase
MD	Mean deviation
dB	Decibel
ONSF	Optic nerve sheath fenestration

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Ethics approval and consent to participate

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Competing interests

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