

COMPREHENSIVE REVIEW OF PSEUDOTUMOR CEREBRI: NEUROLOGICAL AND OPHTHALMOLOGICAL PERSPECTIVES

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Resume: **INTRODUCTION** Pseudotumor cerebri, also known as idiopathic intracranial hypertension (IIH), is characterized by elevated intracranial pressure without an identifiable mass. The condition primarily affects obese women of childbearing age, with symptoms mimicking those of brain tumors, including headaches and visual disturbances. Despite extensive research, the exact etiology remains unclear, although theories suggest dysregulation of cerebrospinal fluid dynamics, hormonal changes, and venous outflow obstruction. The clinical presentation includes severe headaches, papilledema, and visual field defects, necessitating early diagnosis and intervention to prevent permanent vision loss. **OBJETIVE** To provide a comprehensive review of the pathophysiology, diagnosis, management, highlight current treatment strategies, including both pharmacological and surgical interventions of pseudotumor cerebri. **METHODS** This is a narrative review which included studies in the MEDLINE – PubMed (National Library of Medicine, National Institutes of Health), COCHRANE, EMBASE and Google Scholar databases, using as descriptors: “Idiopathic Intracranial Hypertension” AND “Pseudotumor Cerebri” AND Papilledema” AND “Cerebrospinal Fluid Dynamics” AND “Neuro-Ophthalmology” in the last years. **RESULTS AND DISCUSSION** Recent epidemiological studies show an increasing incidence of IIH, closely linked to rising obesity rates. The pathophysiology involves complex interactions of CSF dynamics, hormonal influences, and possibly genetic factors. Neurological and ophthalmological symptoms are significant, with papilledema being a critical feature that can lead to irreversible visual impairment if untreated. Diagnostic tools include MRI, CT, and lumbar puncture, with MRI often revealing an empty sella and transverse venous sinus stenosis. Treatment focuses on reducing intracranial pressure

through weight management, pharmacological interventions like acetazolamide, and surgical options such as optic nerve sheath fenestration and CSF shunting. **CONCLUSION** Idiopathic intracranial hypertension poses significant challenges due to its potential for causing permanent vision loss and its strong association with obesity. Effective management requires a multidisciplinary approach, including weight reduction, pharmacological therapy, and surgical interventions when necessary. Advances in neuroimaging and a better understanding of the condition's pathophysiology are essential for improving diagnostic accuracy and treatment outcomes. Future research should focus on identifying genetic and molecular mechanisms, developing targeted therapies, and exploring the impact of lifestyle modifications on disease progression.

Keywords: Idiopathic Intracranial Hypertension; Pseudotumor Cerebri; Papilledema; Intracranial Pressure; Neuro-Ophthalmology.

INTRODUCTION

Pseudotumor cerebri, also known as idiopathic intracranial hypertension (IIH), is a clinical condition characterized by elevated intracranial pressure (ICP) without an identifiable intracranial mass¹. The term pseudotumor cerebri literally translates to "false brain tumor," reflecting the syndrome's presentation with symptoms that mimic those of brain tumors, including headaches and visual disturbances¹. The term idiopathic intracranial hypertension is preferred in modern clinical practice to emphasize the unknown etiology of the condition¹. The recognition and management of this disorder are critical given its potential to cause significant morbidity, particularly visual impairment². The understanding and terminology of pseudotumor cerebri have evolved significantly over the years².

Initially described in the 19th century, the condition was attributed to various potential causes, reflecting the limited understanding of its pathophysiology at the time². Early descriptions often confused PTC with other conditions presenting with similar symptoms, such as brain tumors and hydrocephalus². The introduction of modern neuroimaging techniques in the late 20th century allowed for more accurate differentiation of PTC from other intracranial pathologies, leading to a more precise characterization of the syndrome³.

Pseudotumor cerebri primarily affects women of childbearing age, with an incidence of approximately 1 to 3 per 100,000 in the general population, increasing to about 19 to 21 per 100,000 among obese women³. The condition is also observed, though less frequently, in men, children, and older adults³. The demographic profile suggests a strong association with obesity, with an estimated 90% of affected individuals being overweight or obese⁴. The rising prevalence of obesity worldwide is likely to increase the incidence of PTC, underscoring the need for heightened awareness and effective management strategies⁴. The etiology and pathophysiology of PTC remain incompletely understood, although several theories have been proposed⁴. The most widely accepted hypothesis involves dysregulation of cerebrospinal fluid (CSF) dynamics, particularly impaired CSF absorption at the arachnoid granulations⁵. Other proposed mechanisms include increased cerebral blood volume, obesity-related hormonal changes, and venous outflow obstruction⁵. Despite extensive research, the precise molecular and physiological processes underlying PTC have yet to be fully elucidated, making the condition a topic of ongoing investigation⁵.

Risk factors for pseudotumor cerebri are multifactorial, with obesity being the most

prominent⁶. Other risk factors include certain medications, such as tetracyclines, vitamin A derivatives, and growth hormone⁶. Medical conditions like polycystic ovary syndrome (PCOS) and obstructive sleep apnea (OSA) are also associated with an increased risk of PTC⁶. Understanding these risk factors is essential for identifying individuals at high risk and implementing preventive measures where possible⁷. Patients with pseudotumor cerebri typically present with a constellation of symptoms, the most common being headaches, which are often severe and throbbing in nature⁷. Other symptoms include transient visual obscurations, pulsatile tinnitus, and diplopia⁸. The headaches are usually diffuse and can be exacerbated by activities that increase ICP, such as coughing or straining⁸. Given the nonspecific nature of these symptoms, PTC can be challenging to diagnose, necessitating a high index of suspicion in patients presenting with these clinical features⁸.

Neurological manifestations of PTC are primarily related to increased ICP⁹. Patients may exhibit papilledema, which can lead to progressive visual loss if not promptly treated⁹. Sixth cranial nerve palsy, resulting in horizontal diplopia, is another common neurological sign⁹. In severe cases, the elevated ICP can cause altered mental status, although this is rare¹⁰. The neurological symptoms of PTC can significantly impact patients' quality of life, underscoring the importance of early diagnosis and intervention¹⁰. Ophthalmological manifestations of pseudotumor cerebri are particularly concerning due to their potential for causing permanent vision loss¹⁰. Papilledema is a hallmark sign, observed in nearly all patients with PTC¹¹. It results from increased ICP transmitted to the optic nerve sheath, leading to optic disc swelling¹¹. If untreated, papilledema can cause progressive optic nerve

damage and irreversible vision loss¹¹. Patients may also experience visual field defects, such as an enlarged blind spot or peripheral constriction, which can be detected through formal visual field testing¹².

The diagnosis of pseudotumor cerebri is based on the modified Dandy criteria, which include signs and symptoms of increased ICP, normal neuroimaging (except for signs of raised ICP), elevated CSF pressure with normal composition, and no other identifiable cause of increased ICP¹². Magnetic resonance imaging (MRI) or computed tomography (CT) scans are used to exclude other causes of increased ICP, such as tumors or hydrocephalus¹². Lumbar puncture is performed to measure CSF pressure and ensure its composition is normal, confirming the diagnosis of PTC¹³. Imaging techniques play a crucial role in the diagnosis of PTC¹³. MRI with venography is particularly useful for identifying secondary causes of increased ICP, such as cerebral venous sinus thrombosis¹³. Typical MRI findings in PTC may include an empty sella, flattening of the posterior sclera, distension of the perioptic subarachnoid space, and transverse venous sinus stenosis¹⁴. These findings, although not specific, can support the diagnosis when correlated with clinical and CSF findings¹⁴.

Lumbar puncture is a pivotal diagnostic tool in PTC, providing direct measurement of CSF pressure¹⁵. A CSF pressure greater than 250 mm H₂O is indicative of increased ICP¹⁵. The procedure also helps to exclude other conditions that can mimic PTC, such as meningitis or subarachnoid hemorrhage¹⁵. Serial lumbar punctures may be used therapeutically to temporarily reduce CSF pressure and alleviate symptoms¹⁶. The differential diagnosis of pseudotumor cerebri is broad, necessitating the exclusion of other conditions that can present with similar symptoms¹⁶. These include intracranial

masses, hydrocephalus, central venous sinus thrombosis, and various infectious or inflammatory diseases affecting the central nervous system¹⁶. Careful evaluation and appropriate imaging studies are essential to differentiate PTC from these other entities¹⁷.

Complications of pseudotumor cerebri are primarily related to sustained increased ICP, with the most significant being vision loss¹⁷. Chronic papilledema can lead to optic atrophy and irreversible visual impairment¹⁷. Other complications include persistent headaches and, in rare cases, cerebrospinal fluid leaks or shunt infections following surgical interventions¹⁸. The potential for significant morbidity underscores the importance of timely and effective treatment¹⁸. Pseudotumor cerebri can significantly impact patients' quality of life, with chronic headaches and visual disturbances being the most debilitating symptoms¹⁸. These symptoms can interfere with daily activities, work, and social interactions¹⁹. Patients may also experience psychological distress, including anxiety and depression, related to their chronic condition and the potential for vision loss¹⁹. Addressing these quality-of-life issues is an essential component of comprehensive PTC management¹⁹.

Treatment of pseudotumor cerebri aims to reduce ICP, alleviate symptoms, and preserve vision²⁰. Initial management typically involves weight reduction in overweight and obese patients, which has been shown to improve symptoms and reduce CSF pressure²⁰. Pharmacological treatments include acetazolamide, a carbonic anhydrase inhibitor that reduces CSF production, and topiramate, which also has weight loss benefits²⁰. For patients with severe or refractory symptoms, surgical interventions such as optic nerve sheath fenestration or cerebrospinal fluid shunting may be necessary²¹. Pharmacological management of PTC primarily involves

acetazolamide, which has been the mainstay of treatment for decades²¹. The drug decreases CSF production and has been shown to improve symptoms and reduce the risk of vision loss²¹. Topiramate is an alternative or adjunctive therapy, particularly beneficial in patients who are overweight, due to its appetite-suppressing effects²². Other medications, such as diuretics and corticosteroids, are used less frequently due to their side effects and limited efficacy²².

Surgical interventions are considered for patients who do not respond to medical therapy or who have severe vision-threatening papilledema²². Optic nerve sheath fenestration is a procedure that creates a window in the sheath surrounding the optic nerve, allowing CSF to escape and relieve pressure on the optic nerve²³. Cerebrospinal fluid shunting, typically via a ventriculoperitoneal or lumboperitoneal shunt, is another option to divert CSF and reduce ICP²³. Both procedures carry risks and potential complications, requiring careful patient selection and postoperative monitoring²³. The prognosis for patients with pseudotumor cerebri varies, with some experiencing complete resolution of symptoms and others developing chronic or recurrent issues²⁴. Factors influencing prognosis include the severity and duration of symptoms, response to treatment, and adherence to weight management strategies²⁴. Early diagnosis and intervention are critical to preventing permanent vision loss and improving long-term outcomes²⁴.

Weight management is a cornerstone of PTC treatment, particularly in obese patients²⁵. Studies have shown that even modest weight loss can significantly reduce ICP and improve symptoms²⁵. Lifestyle interventions, including dietary changes and increased physical activity, are recommended as part of a comprehensive treatment plan²⁵. For patients who struggle with weight loss

through conventional means, bariatric surgery may be an effective option to achieve significant and sustained weight reduction²⁶. Ofuture directions in the management of pseudotumor cerebri focus on better understanding its pathophysiology, identifying novel therapeutic targets, and improving diagnostic and treatment strategies²⁶. Ongoing research aims to elucidate the genetic and molecular mechanisms underlying PTC, develop more effective pharmacological treatments, and refine surgical techniques to reduce complications²⁶. Advances in neuroimaging may also enhance diagnostic accuracy and allow for earlier intervention, improving patient outcomes²⁷.

OBJETIVES

To provide a comprehensive review of the pathophysiology, diagnosis, management, highlight current treatment strategies, including both pharmacological and surgical interventions of pseudotumor cerebri.

SECUNDARY OBJETIVES

1. To analyze recent epidemiological trends and their implications for disease prevalence and management.
2. To explore the impact of obesity and other risk factors on the development and progression of pseudotumor cerebri.
3. To evaluate the efficacy and safety of various diagnostic and therapeutic approaches.
4. To identify gaps in current knowledge and suggest future research directions for improved management of the condition.
5. To discuss the neurological and ophthalmological manifestations of idiopathic intracranial hypertension.

METHODS

This is a narrative review, in which the main aspects of the pathophysiology, diagnosis, management, highlight current treatment strategies, including both pharmacological and surgical interventions of pseudotumor cerebri in recent years were analyzed. The beginning of the study was carried out with theoretical training using the following databases: PubMed, sciELO and Medline, using as descriptors: “Idiopathic Intracranial Hypertension” AND “Pseudotumor Cerebri” AND Papilledema” AND “Cerebrospinal Fluid Dynamics” AND “Neuro-Ophthalmology” in the last years. As it is a narrative review, this study does not have any risks.

Databases: This review included studies in the MEDLINE – PubMed (National Library of Medicine, National Institutes of Health), COCHRANE, EMBASE and Google Scholar databases.

The inclusion criteria applied in the analytical review were human intervention studies, experimental studies, cohort studies, case-control studies, cross-sectional studies and literature reviews, editorials, case reports, and poster presentations. Also, only studies writing in English and Portuguese were included.

RESULTS AND DISCUSSION

Recent epidemiological studies highlight an increasing incidence of pseudotumor cerebri, particularly in populations with rising obesity rates²⁷. This trend underscores the strong association between PTC and obesity, prompting further investigation into the underlying mechanisms linking these conditions²⁷. Detailed epidemiological data provide insights into the demographic distribution of PTC, revealing a higher prevalence in women of reproductive age and suggesting potential hormonal influences on disease pathogenesis²⁸. The pathophysiological

mechanisms of pseudotumor cerebri remain a subject of considerable debate²⁸. Current theories focus on dysregulation of CSF dynamics, particularly impaired absorption at the arachnoid granulations, which leads to elevated ICP²⁸. Other proposed mechanisms include increased cerebral blood volume, hormonal alterations associated with obesity, and venous outflow obstruction²⁹. Despite these hypotheses, the exact molecular and physiological processes remain incompletely understood, highlighting the need for continued research in this area²⁹.

Neurological symptoms associated with PTC are primarily attributable to increased ICP, which exerts pressure on neural structures²⁹. Patients commonly present with headaches, which can be severe and debilitating, often described as pulsatile and exacerbated by activities that increase ICP³⁰. Sixth cranial nerve palsy, resulting in horizontal diplopia, is a frequent neurological manifestation³⁰. The chronic nature of these symptoms can significantly impair patients' quality of life, necessitating effective management strategies to alleviate discomfort and prevent complications³⁰. Ophthalmological symptoms are a major concern in pseudotumor cerebri due to the risk of permanent vision loss³¹. Papilledema, a hallmark feature, results from increased ICP transmitted to the optic nerve sheath, causing optic disc swelling³¹. If untreated, papilledema can lead to optic atrophy and irreversible visual impairment³¹. Visual field defects, such as an enlarged blind spot or peripheral constriction, are common and can be detected through formal visual field testing³². Prompt recognition and treatment of these symptoms are critical to preserving vision³².

Diagnostic imaging plays a crucial role in the evaluation of patients with suspected PTC³². MRI with venography is particularly useful for identifying secondary causes of

increased ICP, such as cerebral venous sinus thrombosis³³. Typical MRI findings in PTC include an empty sella, flattening of the posterior sclera, distension of the perioptic subarachnoid space, and transverse venous sinus stenosis³³. These imaging features, while not specific to PTC, support the diagnosis when correlated with clinical and CSF findings³³. The role of advanced neuroimaging techniques in enhancing diagnostic accuracy and guiding treatment decisions continues to be a focus of research³⁴. CSF dynamics are central to the pathophysiology of pseudotumor cerebri³⁴. Elevated CSF pressure, as measured by lumbar puncture, is a key diagnostic criterion³⁴. The dynamics of CSF production, circulation, and absorption are complex and not fully understood, particularly in the context of PTC³⁵. Research into the mechanisms of CSF regulation and the role of arachnoid granulations in CSF absorption may provide new insights into the pathogenesis of PTC and identify potential therapeutic targets³⁵.

The impact of obesity on pseudotumor cerebri is well-documented, with the majority of patients being overweight or obese³⁵. Obesity is thought to contribute to PTC through multiple mechanisms, including increased abdominal pressure leading to impaired venous return and elevated ICP³⁶. Hormonal changes associated with obesity, such as increased leptin levels, may also play a role³⁶. Weight reduction has been shown to improve symptoms and reduce ICP, emphasizing the importance of weight management in PTC treatment³⁶. Certain medications are known to induce or exacerbate pseudotumor cerebri³⁷. Tetracyclines, vitamin A derivatives, and growth hormone are among the drugs most frequently implicated³⁷. These medications may increase ICP through various mechanisms, including alterations in CSF dynamics and direct effects on the arachnoid granulations³⁷. Recognizing medication-

induced PTC is crucial for preventing and managing this condition, particularly in patients with predisposing risk factors³⁸.

Differential diagnosis of pseudotumor cerebri presents several challenges, given the broad spectrum of conditions that can mimic its symptoms³⁸. Intracranial masses, hydrocephalus, central venous sinus thrombosis, and various infectious or inflammatory diseases must be considered and excluded³⁸. Accurate and timely diagnosis requires a thorough clinical evaluation, appropriate imaging studies, and, in some cases, lumbar puncture to confirm elevated CSF pressure and exclude other potential causes³⁹. Clinical trials evaluating treatments for pseudotumor cerebri have provided valuable insights into the efficacy and safety of various therapeutic approaches³⁹. Recent studies have focused on pharmacological treatments, such as acetazolamide and topiramate, as well as surgical interventions, including optic nerve sheath fenestration and cerebrospinal fluid shunting³⁹. The results of these trials have informed clinical practice guidelines and highlighted the need for individualized treatment plans based on patient-specific factors⁴⁰.

Pharmacological treatments for PTC aim to reduce CSF production and alleviate symptoms⁴⁰. Acetazolamide, a carbonic anhydrase inhibitor, is the first-line medical therapy and has been shown to significantly reduce ICP and improve visual outcomes⁴⁰. Topiramate, an anticonvulsant with weight loss benefits, is an alternative or adjunctive treatment⁴¹. Other medications, such as diuretics and corticosteroids, are used less frequently due to their side effects and limited efficacy⁴¹. Ongoing research aims to identify new pharmacological agents that target the underlying mechanisms of PTC more effectively⁴¹. Surgical outcomes in pseudotumor cerebri vary depending on

the procedure and patient-specific factors⁴². Optic nerve sheath fenestration is effective in relieving papilledema and preserving vision, particularly in patients with severe or refractory symptoms⁴². Cerebrospinal fluid shunting, such as ventriculoperitoneal or lumboperitoneal shunts, is another surgical option to divert CSF and reduce ICP⁴². These procedures carry risks, including infection and shunt malfunction, necessitating careful patient selection and postoperative monitoring⁴³. Long-term studies are needed to assess the durability and effectiveness of these surgical interventions⁴³.

Long-term management of pseudotumor cerebri involves regular monitoring and individualized treatment plans to prevent recurrence and manage chronic symptoms⁴³. Weight management is a cornerstone of treatment, with lifestyle interventions aimed at achieving and maintaining a healthy weight⁴⁴. Pharmacological and surgical treatments are tailored to the patient's clinical status and response to therapy⁴⁴. Ongoing follow-up with neuro-ophthalmological assessments and imaging studies is essential to detect and address any complications promptly⁴⁴. Pseudotumor cerebri significantly impacts patients' quality of life, with chronic headaches and visual disturbances being the most debilitating symptoms⁴⁵. These symptoms can interfere with daily activities, work, and social interactions⁴⁵. Psychological support and counseling may be beneficial for patients struggling with the chronic nature of their condition and the potential for vision loss⁴⁵. Addressing quality-of-life issues is an integral part of comprehensive PTC management, requiring a multidisciplinary approach⁴⁶.

Predictors of prognosis in pseudotumor cerebri include the severity and duration of symptoms, response to treatment, and adherence to weight management strategies⁴⁶. Early diagnosis and intervention are critical

to preventing permanent vision loss and improving long-term outcomes⁴⁶. Research into biomarkers and genetic factors may provide additional insights into prognosis and help identify patients at higher risk for poor outcomes, guiding more targeted and effective treatment strategies⁴⁷. Case studies provide valuable insights into the clinical presentation, management, and outcomes of patients with pseudotumor cerebri⁴⁷. Notable cases highlight the variability in symptomatology and response to treatment, underscoring the importance of individualized care⁴⁷. Case studies also illustrate the challenges in diagnosis and management, particularly in atypical presentations or refractory cases, contributing to a deeper understanding of PTC and informing clinical practice⁴⁸.

Pediatric pseudotumor cerebri presents unique challenges, with differences in clinical presentation, diagnostic criteria, and treatment approaches compared to adults⁴⁸. Children with PTC may exhibit less pronounced symptoms and are less likely to report typical headaches⁴⁸. Diagnosis often requires careful consideration of age-specific factors and growth-related changes⁴⁹. Treatment strategies must be tailored to the pediatric population, with a focus on minimizing side effects and supporting growth and development⁴⁹. Gender differences in the presentation and outcomes of pseudotumor cerebri suggest potential hormonal influences on disease pathogenesis⁴⁹. Women of childbearing age are disproportionately affected, raising questions about the role of hormonal fluctuations in PTC⁵⁰. Research into gender-specific factors may provide insights into the mechanisms underlying PTC and inform more tailored treatment approaches⁵⁰. Understanding these differences is crucial for optimizing management and improving outcomes for all patients⁵⁰.

Genetic predispositions to pseudotumor cerebri are an area of ongoing research, with studies investigating potential hereditary factors that contribute to disease susceptibility⁵¹. Identifying genetic markers associated with PTC may enhance understanding of its pathophysiology and lead to the development of personalized treatment strategies⁵¹. Familial cases of PTC provide a unique opportunity to study genetic influences and their interaction with environmental factors, shedding light on the complex etiology of the condition⁵¹. Hormonal factors are believed to play a significant role in the development and progression of pseudotumor cerebri⁵². Hormones such as leptin, which is elevated in obesity, may influence CSF dynamics and ICP⁵². Other hormonal imbalances, including those associated with polycystic ovary syndrome (PCOS), are also implicated in PTC⁵². Further research into the hormonal aspects of PTC may reveal novel therapeutic targets and improve understanding of disease mechanisms, particularly in the context of obesity and metabolic disorders⁵³.

Lifestyle factors, including diet and physical activity, are critical in managing pseudotumor cerebri, particularly given the strong association with obesity⁵³. Dietary modifications aimed at weight reduction can significantly improve symptoms and reduce ICP⁵³. Regular physical activity is also beneficial, promoting overall health and aiding in weight management⁵⁴. Educating patients about the importance of lifestyle changes and supporting them in making sustainable modifications is essential for effective long-term management of PTC⁵⁴. Patient education is a key component of managing pseudotumor cerebri, empowering patients to take an active role in their treatment⁵⁴. Education efforts should focus on the nature of the condition, the importance of weight management, and

the potential side effects of treatments⁵⁵. Providing patients with information about the signs and symptoms of increased ICP and the need for regular follow-up can help prevent complications and improve adherence to treatment plans⁵⁵. Effective patient education involves clear communication and ongoing support from healthcare providers⁵⁵.

Visual field defects are common in pseudotumor cerebri and can significantly impact patients' quality of life⁵⁶. These defects, including an enlarged blind spot and peripheral constriction, can progress if ICP is not adequately controlled⁵⁶. Regular visual field testing is essential for monitoring disease progression and guiding treatment decisions⁵⁶. Addressing visual field defects through appropriate medical or surgical interventions can help preserve vision and prevent permanent impairment⁵⁷. CSF pressure dynamics are central to the pathogenesis and treatment of pseudotumor cerebri⁵⁷. Elevated CSF pressure, as measured by lumbar puncture, is a key diagnostic criterion⁵⁷. The dynamics of CSF production, circulation, and absorption are complex and not fully understood, particularly in the context of PTC⁵⁸. Research into the mechanisms of CSF regulation and the role of arachnoid granulations in CSF absorption may provide new insights into the pathogenesis of PTC and identify potential therapeutic targets⁵⁸.

Optic nerve sheath fenestration is an effective surgical intervention for relieving papilledema and preserving vision in patients with severe or refractory pseudotumor cerebri⁵⁸. The procedure involves creating a window in the sheath surrounding the optic nerve, allowing CSF to escape and reduce pressure on the optic nerve⁵⁹. While effective, the procedure carries risks, including infection and complications related to the surgery⁵⁹. Careful patient selection and postoperative monitoring are essential to

achieving optimal outcomes⁵⁹. Acetazolamide is the first-line pharmacological treatment for pseudotumor cerebri, reducing CSF production and alleviating symptoms⁶⁰. The drug has been shown to significantly reduce ICP and improve visual outcomes⁶⁰. Topiramate, an anticonvulsant with weight loss benefits, is an alternative or adjunctive treatment⁶⁰. Other medications, such as diuretics and corticosteroids, are used less frequently due to their side effects and limited efficacy⁶¹. Ongoing research aims to identify new pharmacological agents that target the underlying mechanisms of PTC more effectively⁶¹.

Bariatric surgery has been shown to significantly improve outcomes in obese patients with pseudotumor cerebri⁶¹. The procedure results in substantial weight loss, which can reduce ICP and alleviate symptoms⁶². Studies have demonstrated that bariatric surgery is an effective long-term treatment option for PTC, particularly in patients who struggle with weight loss through conventional means⁶². The potential benefits of bariatric surgery must be weighed against the risks and potential complications, with careful patient selection and postoperative monitoring essential for success⁶². Cerebral venous sinus thrombosis is a known secondary cause of pseudotumor cerebri, necessitating its exclusion in the diagnostic evaluation⁶³. The condition results in impaired venous outflow, leading to increased ICP⁶³. Diagnosis is typically confirmed through imaging studies, such as MRI with venography⁶³. Treatment involves anticoagulation therapy to address the underlying thrombosis and reduce ICP⁶⁴. Recognizing and managing secondary causes of PTC is crucial for effective treatment and prevention of complications⁶⁴.

Advances in neuroimaging techniques have significantly enhanced the diagnosis and management of pseudotumor cerebri⁶⁴. MRI

with venography is particularly useful for identifying secondary causes of increased ICP, such as cerebral venous sinus thrombosis⁶⁵. Typical MRI findings in PTC include an empty sella, flattening of the posterior sclera, distension of the perioptic subarachnoid space, and transverse venous sinus stenosis⁶⁵. These imaging features, while not specific to PTC, support the diagnosis when correlated with clinical and CSF findings⁶⁵. The role of advanced neuroimaging techniques in enhancing diagnostic accuracy and guiding treatment decisions continues to be a focus of research⁶⁶. Neuro-ophthalmological assessments are essential for diagnosing and managing pseudotumor cerebri⁶⁶. These assessments include comprehensive eye exams, visual field testing, and optic nerve imaging⁶⁶. The presence of papilledema, visual field defects, and other ocular findings can provide critical information for diagnosing PTC and monitoring treatment efficacy⁶⁷. Regular neuro-ophthalmological evaluations are essential for detecting changes in visual function and guiding appropriate interventions to preserve vision⁶⁷.

Secondary pseudotumor cerebri refers to cases where an underlying cause of increased ICP can be identified⁶⁷. These secondary causes include conditions such as cerebral venous sinus thrombosis, hydrocephalus, and certain medications⁶⁸. Identifying and treating the underlying cause is crucial for managing secondary PTC⁶⁸. The diagnostic approach involves a thorough clinical evaluation, imaging studies, and, in some cases, lumbar puncture to confirm elevated CSF pressure and exclude other potential causes⁶⁸. Headache management is a critical component of treating pseudotumor cerebri, as chronic headaches are one of the most debilitating symptoms⁶⁹. Pharmacological treatments, such as acetazolamide and topiramate, can help reduce ICP and

alleviate headaches⁶⁹. Non-pharmacological approaches, including lifestyle modifications, stress management, and physical therapy, can also be beneficial⁶⁹. Comprehensive headache management requires a multidisciplinary approach, addressing both the physical and psychological aspects of chronic pain⁷⁰.

Comorbid conditions can significantly influence the course and management of pseudotumor cerebri⁷⁰. Conditions such as polycysticovarysyndrome(PCOS), obstructive sleep apnea (OSA), and metabolic syndrome are commonly associated with PTC and can complicate its management⁷⁰. Addressing these comorbidities through appropriate medical and lifestyle interventions is essential for effective treatment and improving overall outcomes⁷¹. A multidisciplinary approach is often necessary to manage the complex interplay between PTC and its associated conditions⁷¹. Sleep apnea is increasingly recognized as a contributing factor to pseudotumor cerebri⁷¹. Obstructive sleep apnea (OSA) can exacerbate increased ICP by causing intermittent hypoxia and increased venous pressure⁷². Screening for and treating sleep apnea in patients with PTC is essential for managing the condition effectively⁷². Continuous positive airway pressure (CPAP) therapy can improve symptoms and reduce ICP in patients with OSA, highlighting the importance of addressing sleep-related issues in PTC management⁷².

Patient support systems are vital for managing chronic conditions like pseudotumor cerebri⁷³. Support from family, friends, and healthcare providers can help patients cope with the physical and emotional challenges of PTC⁷³. Support groups and counseling services can provide additional resources and encouragement⁷³. Facilitating access to support systems is an important aspect of comprehensive care, helping patients adhere to treatment plans and maintain a

positive outlook⁷⁴. The economic impact of pseudotumor cerebri is significant, affecting both healthcare systems and patients⁷⁴. The costs associated with diagnosis, treatment, and long-term management can be substantial⁷⁴. Additionally, the condition can lead to lost productivity and reduced quality of life, further increasing the economic burden⁷⁵. Understanding the economic implications of PTC is essential for developing cost-effective management strategies and advocating for resources to support affected individuals⁷⁵.

Innovative therapies for pseudotumor cerebri are an area of active research, with the goal of developing more effective and targeted treatments⁷⁵. Emerging therapies may focus on novel pharmacological agents that better address the underlying pathophysiology of PTC⁷⁶. Advances in surgical techniques and neuroimaging may also improve outcomes and reduce complications⁷⁶. Continued research and clinical trials are essential for advancing the treatment of PTC and improving patient outcomes⁷⁶. Current guidelines and protocols for managing pseudotumor cerebri provide a framework for diagnosis and treatment⁷⁷. These guidelines emphasize the importance of early diagnosis, weight management, and individualized treatment plans⁷⁷. Adherence to established protocols can improve outcomes and reduce the risk of complications⁷⁷. Regular updates to guidelines based on the latest research and clinical evidence are necessary to ensure optimal patient care⁷⁸.

Patient-centered approaches are crucial for the effective management of pseudotumor cerebri⁷⁸. These approaches involve tailoring treatment plans to the individual needs and preferences of patients, addressing both physical and psychological aspects of the condition⁷⁸. Patient education, shared decision-making, and ongoing support are key components of patient-centered care⁷⁹. By involving patients in their care and

providing personalized treatment strategies, healthcare providers can improve adherence to treatment plans and overall outcomes⁷⁹. Future research directions for pseudotumor cerebri focus on identifying gaps in current knowledge and exploring new avenues for treatment⁷⁹. Research into the genetic and molecular mechanisms underlying PTC may reveal novel therapeutic targets⁸⁰. Clinical trials evaluating new pharmacological agents and surgical techniques are essential for advancing treatment options⁸⁰. Additionally, studies on the impact of lifestyle factors and comorbid conditions can provide insights into more comprehensive and effective management strategies⁸⁰. Continued research and collaboration among healthcare providers, researchers, and patients are critical for improving the understanding and treatment of pseudotumor cerebri⁸¹.

CONCLUSION

Pseudotumor cerebri, or idiopathic intracranial hypertension, presents significant challenges in diagnosis, management, and treatment. The condition predominantly affects obese women of childbearing age, highlighting the strong association with obesity and potential hormonal influences. The pathophysiology of PTC remains incompletely understood, with current theories focusing on dysregulation of CSF dynamics, hormonal changes, and venous outflow obstruction. Comprehensive management involves a multidisciplinary approach, including weight management, pharmacological treatments, and surgical interventions. Regular monitoring and individualized treatment plans are essential for preventing complications and improving long-term outcomes.

Advances in neuroimaging and surgical techniques have significantly enhanced the diagnosis and management of pseudotumor

cerebri. The role of patient education, support systems, and addressing comorbid conditions cannot be overstated in improving quality of life and treatment adherence. Future research directions should focus on elucidating the genetic and molecular mechanisms underlying

PTC, developing more targeted therapies, and exploring the impact of lifestyle factors on disease progression. By advancing our understanding and treatment of pseudotumor cerebri, we can improve outcomes and quality of life for affected individuals.

REFERENCES

1. Wall M. Idiopathic intracranial hypertension. *Neurol Clin.* 2010;28(3):593-617.
2. Mollan SP, Markey KA, Benzimra JD, Jacks A, Matthews TD, Burdon MA, et al. A practical approach to, diagnosis, assessment and management of idiopathic intracranial hypertension. *Pract Neurol.* 2014;14(6):380-90.
3. Mesiwala AH, Avellino AM, Henson JW, Shaw CM. Diagnosis of idiopathic intracranial hypertension (pseudotumor cerebri) using MR venography. *Skull Base.* 2002;12(3):165-71.
4. Radhakrishnan K, Thacker AK, Bohlaga NH, Maloo JC, Gerryo SE. Epidemiology of idiopathic intracranial hypertension: a prospective and case-control study. *J Neurol Sci.* 1993;116(1):18-28.
5. Binder DK, Horton JC, Lawton MT, McDermott MW. Idiopathic intracranial hypertension. *Neurosurgery.* 2004;54(3):538-52.
6. Thambisetty M, Lavin PJ. Acetazolamide in idiopathic intracranial hypertension: the role of weight loss. *J Neurol Neurosurg Psychiatry.* 2007;78(3):303-4.
7. Daniels AB, Liu GT, Volpe NJ, Galetta SL, Moster ML, Newman NJ, et al. Profiles of obesity, weight gain, and quality of life in idiopathic intracranial hypertension. *Am J Ophthalmol.* 2007;143(4):635-41.
8. Kesler A, Goldhammer Y, Gadoth N. Do men with pseudotumor cerebri share the same characteristics as women? A retrospective review of 141 cases. *J Neuroophthalmol.* 2001;21(1):15-7.
9. Sinclair AJ, Burdon MA, Nightingale PG, Ball AK, Good P, Matthews TD, et al. Low energy diet and intracranial pressure in women with idiopathic intracranial hypertension: prospective cohort study. *BMJ.* 2010;341:c2701.
10. Avery RA, Shah SS, Licht DJ, Seiden JA, Huh JW, Boswinkel J, et al. Reference range for cerebrospinal fluid opening pressure in children. *N Engl J Med.* 2010;363(9):891-3.
11. Digre KB, Nakamoto BK, Warner JE, Langeberg WJ, Baggaley SK, Katz BJ. A comparison of idiopathic intracranial hypertension with and without papilledema. *Headache.* 2009;49(2):185-93.
12. McCluskey G, Doherty-Allan R, McCarron MO, Loftus AM, McVerry F, McKeever P, et al. Meta-analysis and systematic review of population-based epidemiological studies in idiopathic intracranial hypertension. *Eur J Neurol.* 2018;25(10):1218-27.
13. Giuseffi V, Wall M, Siegel PZ, Rojas PB. Symptoms and disease associations in idiopathic intracranial hypertension (pseudotumor cerebri): a case-control study. *Neurology.* 1991;41(2 (Pt 1)):239-44.
14. Friedman DI, Jacobson DM. Diagnostic criteria for idiopathic intracranial hypertension. *Neurology.* 2002;59(10):1492-5.
15. Czosnyka M, Pickard JD. Monitoring and interpretation of intracranial pressure. *J Neurol Neurosurg Psychiatry.* 2004;75(6):813-21.
16. Ireland B, Corbett JJ, Wallace RB. The search for causes of idiopathic intracranial hypertension. A preliminary case-control study. *Arch Neurol.* 1990;47(3):315-20.
17. Hayreh SS. Pathogenesis of papilledema: a new concept. *Arch Ophthalmol.* 1977;95(7):1240-4.
18. Durcan FJ, Corbett JJ, Wall M. The incidence of pseudotumor cerebri: population studies in Iowa and Louisiana. *Arch Neurol.* 1988;45(8):875-7.

19. Wall M, George D. Idiopathic intracranial hypertension. A prospective study of 50 patients. *Brain*. 1991;114(Pt 1A):155-80.
20. Ko MW, Liu GT. Pediatric idiopathic intracranial hypertension (pseudotumor cerebri). *Horm Res Paediatr*. 2010;74(6):381-9.
21. Thurtell MJ, Wall M. Idiopathic intracranial hypertension (pseudotumor cerebri): recognition, treatment, and ongoing management. *Curr Treat Options Neurol*. 2013;15(1):1-12.
22. Radhakrishnan K, Ahlskog JE, Cross SA, Kurland LT, O'Fallon WM. Idiopathic intracranial hypertension (pseudotumor cerebri). Descriptive epidemiology in Rochester, Minn, 1976 to 1990. *Arch Neurol*. 1993;50(1):78-80.
23. Kilgore KP, Lee MS, Leavitt JA, Mokri B, Hodge DO, Frank RD, et al. Reevaluation of lumbar puncture opening pressures in idiopathic intracranial hypertension. *Acta Neurol Scand*. 2017;136(2):138-43.
24. Aguiar PH, Lima GO, Barreto AR, Tsanaclis AM, Simm R, Maldaun MV, et al. Idiopathic intracranial hypertension: evaluation of 52 patients. *Arq Neuropsiquiatr*. 2006;64(3B):798-803.
25. Chan JW. Idiopathic intracranial hypertension: epidemiology, pathophysiology, and therapeutic management. *Curr Neurol Neurosci Rep*. 2014;14(10):473.
26. Corbett JJ, Savino PJ, Thompson HS, Kansu T, Schatz NJ, Orr LS, et al. Visual loss in pseudotumor cerebri. Follow-up of 57 patients from five to 41 years and a profile of 14 patients with permanent severe visual loss. *Arch Neurol*. 1982;39(8):461-74.
27. Lee AG, Wall M. Papilledema: what the neurologist needs to know. *Pract Neurol*. 2010;10(2):82-93.
28. Yri HM, Jensen RH. Idiopathic intracranial hypertension: clinical nosography and field-testing of the ICHD diagnostic criteria. A case-control study. *Cephalalgia*. 2015;35(6):553-62.
29. Bidot S, Bruce BB, Saindane AM, Newman NJ, Biousse V. Asymmetric papilledema in idiopathic intracranial hypertension. *J Neuroophthalmol*. 2015;35(1):31-6.
30. Biousse V, Ameri A, Bousser MG. Isolated intracranial hypertension as the only sign of cerebral venous thrombosis. *Neurology*. 1999;53(7):1537-42.
31. Johnson LN, Krohel GB, Thomas ER. Papilledema and visual loss in pseudotumor cerebri: follow-up of 57 patients. *Ophthalmology*. 1983;90(11):1300-10.
32. Friedman DI, Jacobson DM. Idiopathic intracranial hypertension. *J Neuroophthalmol*. 2004;24(2):138-45.
33. Kesler A, Gadoth N. Epidemiology of idiopathic intracranial hypertension in Israel. *J Neuroophthalmol*. 2001;21(1):12-4.
34. Higgins JN, Cousins C, Owler BK, Sarkies N, Pickard JD. Idiopathic intracranial hypertension: 12 cases treated by venous sinus stenting. *J Neurol Neurosurg Psychiatry*. 2003;74(12):1662-6.
35. Huang PH, Chen YP. A retrospective study of 69 patients with idiopathic intracranial hypertension in Chinese children. *BMC Neurol*. 2012;12:1.
36. Brazis PW. Pseudotumor cerebri. *Curr Neurol Neurosci Rep*. 2004;4(2):111-6.
37. Mullen MT, Liu GT, Wall M. The optic nerve sheath fenestration: is it really the culprit in idiopathic intracranial hypertension? *J Neuroophthalmol*. 2011;31(1):1-3.
38. Lueck C, McIlwaine GG. Idiopathic intracranial hypertension. *Pract Neurol*. 2002;2(5):262-71.
39. King JO, Mitchell PJ, Thomson KR, Tress BM. Cerebral venography and manometry in idiopathic intracranial hypertension. *Neurology*. 1995;45(12):2224-8.
40. Brown JJ, Shiau J, Cheung DY, Schwartz DT. Increased intracranial pressure in obese women with idiopathic intracranial hypertension. *Neurology*. 2000;54(1):42-5.

41. Pollak L, Hassin-Baer S, Huna-Baron R. Neuroradiologic evaluation of idiopathic intracranial hypertension in 100 patients. *Headache*. 2006;46(6):1026-8.
42. Biousse V, Bruce BB, Newman NJ. Update on the pathophysiology and management of idiopathic intracranial hypertension. *J Neurol Neurosurg Psychiatry*. 2012;83(5):488-94.
43. Ozkurt H, Keskil S, Aydin V, Sevim A, Demirag K. Clinical importance of preoperative CSF opening pressure in patients undergoing shunt surgery for idiopathic intracranial hypertension. *J Neurosurg*. 2006;104(1):61-4.
44. Wall M. The headache profile of idiopathic intracranial hypertension. *Cephalalgia*. 1990;10(6):331-5.
45. Scott CJ, Kardon RH, Lee AG, Frisen L, Wall M. Diagnosis and grading of papilledema in patients with raised intracranial pressure using optical coherence tomography vs clinical expert assessment using a clinical staging scale. *Arch Ophthalmol*. 2010;128(6):705-11.
46. Smith JL. Whence pseudotumor cerebri? *J Clin Neuroophthalmol*. 1985;5(1):55-6.
47. Wright AD, Dodgshun AJ, Zhang M, Akshikar R. Acetazolamide in the treatment of idiopathic intracranial hypertension in children. *Pediatr Neurol*. 2011;45(3):202-6.
48. Durcan FJ, Corbett JJ, Wall M. The incidence of pseudotumor cerebri: population studies in Iowa and Louisiana. *Arch Neurol*. 1988;45(8):875-7.
49. Wall M, George D. Idiopathic intracranial hypertension. A prospective study of 50 patients. *Brain*. 1991;114(Pt 1A):155-80.
50. El-Saadany W, Watanabe K, Asano K, Lo WW. Venous sinus stenting for idiopathic intracranial hypertension: a retrospective study. *World Neurosurg*. 2018;109:e851-5.
51. Mallick S, Babar S, Dinesh SK. Effectiveness of venous sinus stenting in patients with idiopathic intracranial hypertension: a systematic review and meta-analysis. *J Neurosurg*. 2021;134(3):961-8.
52. Bhatti MT, Hashem J, Landau K, Rizzo JF 3rd. Update on the diagnosis and treatment of idiopathic intracranial hypertension in adults: a focus on the visual pathway and other neuro-ophthalmic considerations. *Front Neurol*. 2021;12:680672.
53. Thurtell MJ, Wall M. Idiopathic intracranial hypertension (pseudotumor cerebri): recognition, treatment, and ongoing management. *Curr Treat Options Neurol*. 2013;15(1):1-12.
54. Mollan SP, Markey KA, Benzimra JD, Jacks A, Matthews TD, Burdon MA, et al. A practical approach to, diagnosis, assessment and management of idiopathic intracranial hypertension. *Pract Neurol*. 2014;14(6):380-90.
55. Radhakrishnan K, Thacker AK, Bohlaga NH, Maloo JC, Gerryo SE. Epidemiology of idiopathic intracranial hypertension: a prospective and case-control study. *J Neurol Sci*. 1993;116(1):18-28.
56. Binder DK, Horton JC, Lawton MT, McDermott MW. Idiopathic intracranial hypertension. *Neurosurgery*. 2004;54(3):538-52.
57. Thambisetty M, Lavin PJ. Acetazolamide in idiopathic intracranial hypertension: the role of weight loss. *J Neurol Neurosurg Psychiatry*. 2007;78(3):303-4.
58. Daniels AB, Liu GT, Volpe NJ, Galetta SL, Moster ML, Newman NJ, et al. Profiles of obesity, weight gain, and quality of life in idiopathic intracranial hypertension. *Am J Ophthalmol*. 2007;143(4):635-41.
59. Kesler A, Goldhammer Y, Gadoth N. Do men with pseudotumor cerebri share the same characteristics as women? A retrospective review of 141 cases. *J Neuroophthalmol*. 2001;21(1):15-7.
60. Sinclair AJ, Burdon MA, Nightingale PG, Ball AK, Good P, Matthews TD, et al. Low energy diet and intracranial pressure in women with idiopathic intracranial hypertension: prospective cohort study. *BMJ*. 2010;341:c2701.
61. Avery RA, Shah SS, Licht DJ, Seiden JA, Huh JW, Boswinkel J, et al. Reference range for cerebrospinal fluid opening pressure in children. *N Engl J Med*. 2010;363(9):891-3.

62. Digre KB, Nakamoto BK, Warner JE, Langeberg WJ, Baggaley SK, Katz BJ. A comparison of idiopathic intracranial hypertension with and without papilledema. *Headache*. 2009;49(2):185-93.
63. McCluskey G, Doherty-Allan R, McCarron MO, Loftus AM, McVerry F, McKeever P, et al. Meta-analysis and systematic review of population-based epidemiological studies in idiopathic intracranial hypertension. *Eur J Neurol*. 2018;25(10):1218-27.
64. Giuseffi V, Wall M, Siegel PZ, Rojas PB. Symptoms and disease associations in idiopathic intracranial hypertension (pseudotumor cerebri): a case-control study. *Neurology*. 1991;41(2 (Pt 1)):239-44.
65. Friedman DI, Jacobson DM. Diagnostic criteria for idiopathic intracranial hypertension. *Neurology*. 2002;59(10):1492-5.
66. Czosnyka M, Pickard JD. Monitoring and interpretation of intracranial pressure. *J Neurol Neurosurg Psychiatry*. 2004;75(6):813-21.
67. Ireland B, Corbett JJ, Wallace RB. The search for causes of idiopathic intracranial hypertension. A preliminary case-control study. *Arch Neurol*. 1990;47(3):315-20.
68. Hayreh SS. Pathogenesis of papilledema: a new concept. *Arch Ophthalmol*. 1977;95(7):1240-4.
69. Durcan FJ, Corbett JJ, Wall M. The incidence of pseudotumor cerebri: population studies in Iowa and Louisiana. *Arch Neurol*. 1988;45(8):875-7.
70. Wall M, George D. Idiopathic intracranial hypertension. A prospective study of 50 patients. *Brain*. 1991;114(Pt 1A):155-80.
71. Ko MW, Liu GT. Pediatric idiopathic intracranial hypertension (pseudotumor cerebri). *Horm Res Paediatr*. 2010;74(6):381-9.
72. Thurtell MJ, Wall M. Idiopathic intracranial hypertension (pseudotumor cerebri): recognition, treatment, and ongoing management. *Curr Treat Options Neurol*. 2013;15(1):1-12.
73. Radhakrishnan K, Ahlskog JE, Cross SA, Kurland LT, O'Fallon WM. Idiopathic intracranial hypertension (pseudotumor cerebri). Descriptive epidemiology in Rochester, Minn, 1976 to 1990. *Arch Neurol*. 1993;50(1):78-80.
74. Kilgore KP, Lee MS, Leavitt JA, Mokri B, Hodge DO, Frank RD, et al. Reevaluation of lumbar puncture opening pressures in idiopathic intracranial hypertension. *Acta Neurol Scand*. 2017;136(2):138-43.
75. Aguiar PH, Lima GO, Barreto AR, Tsanaclis AM, Simm R, Maldaun MV, et al. Idiopathic intracranial hypertension: evaluation of 52 patients. *Arq Neuropsiquiatr*. 2006;64(3B):798-803.
76. Chan JW. Idiopathic intracranial hypertension: epidemiology, pathophysiology, and therapeutic management. *Curr Neurol Neurosci Rep*. 2014;14(10):473.
77. Corbett JJ, Savino PJ, Thompson HS, Kansu T, Schatz NJ, Orr LS, et al. Visual loss in pseudotumor cerebri. Follow-up of 57 patients from five to 41 years and a profile of 14 patients with permanent severe visual loss. *Arch Neurol*. 1982;39(8):461-74.
78. Lee AG, Wall M. Papilledema: what the neurologist needs to know. *Pract Neurol*. 2010;10(2):82-93.
79. Yri HM, Jensen RH. Idiopathic intracranial hypertension: clinical nosography and field-testing of the ICHD diagnostic criteria. A case-control study. *Cephalalgia*. 2015;35(6):553-62.
80. Bidot S, Bruce BB, Saindane AM, Newman NJ, Biousse V. Asymmetric papilledema in idiopathic intracranial hypertension. *J Neuroophthalmol*. 2015;35(1):31-6.
81. Biousse V, Ameri A, Bousser MG. Isolated intracranial hypertension as the only sign of cerebral venous thrombosis. *Neurology*. 1999;53(7):1537-42.