Pediatric Intracranial Hypertension

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Education Gap

Intracranial hypertension can lead to significant morbidity; because it is a rare disorder, the general practitioner can easily miss cases.

Objectives

After completing this article, readers should be able to:

1. Recognize the clinical features of intracranial hypertension.
2. Understand the basics of the ophthalmologic examination.
3. Recognize potential causative factors contributing to intracranial hypertension.
4. Appropriately initiate the evaluation in suspected cases and start management in confirmed cases.

Abstract

Headaches are common in the clinical setting. Fortunately, intracranial hypertension (IH) is rare, but when present it can lead to significant morbidity. Early diagnosis and proper management are important to lessen the potential morbidity. Careful headache history, ophthalmologic examination, head imaging, and lumbar puncture (LP) are crucial tools in the diagnosis of this condition. Management should be coordinated with a neurologist, ophthalmologist, or neuro-ophthalmologist.

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ABBREVIATIONS

BID	twice daily
CSF	cerebrospinal fluid
ICP	intracranial pressure
IH	intracranial hypertension
LP	lumbar puncture
MRI	magnetic resonance imaging
ONSF	optic nerve sheath fenestration
PIH	primary intracranial hypertension
SIH	secondary intracranial hypertension

CASE PRESENTATION

Two patients present to your general pediatric clinic with separate complaints but related pathology. The first is a 16-year-old girl with a chief complaint of headache. She reports a daily headache for the past 3 weeks that is worse in the morning and improves over the course of the day but never resolves. On examination she is obese (BMI, 29) but is otherwise well-appearing. Funduscopic examination reveals blurred disc margins bilaterally. Visual field testing by direct confrontation notes constricted peripheral vision on the right side. The next patient is a well-appearing 6-year-old boy who presents after failing his vision screening at school. He denies any symptoms, but his mother interjects that he has been complaining

of "seeing two of things" intermittently for the past few months. On examination he is a well-appearing boy with normal growth indices. He has a slight left esotropia and complains of diplopia when looking to the far left. Funduscopic examination reveals blurred disc margins bilaterally.

DEFINITION

The first description of IH in the literature was by Quincke in 1897, and he labeled it *meningitis serosa*. (1) Since this initial publication, advances in medicine have resulted in a multitude of names, including *serous meningitis, toxic hydrocephalus, otitic hydrocephalus, hypertensive meningeal hydrops, pseudotumor cerebri, benign intracranial hypertension, idiopathic intracranial hypertension,* and *pseudotumor cerebri syndrome*. Today, *pseudotumor cerebri* and *idiopathic intracranial hypertension* are the 2 terms most often used by physicians and the public. Unfortunately, the term *pseudotumor cerebri* can become confusing when a cause for the IH is found; thus, we prefer the terms *primary IH (PIH)* and *secondary IH (SIH)*. Primary refers to cases where no clear causative agent for the increased intracranial pressure (ICP) is found, replacing the terms *idiopathic intracranial hypertension* and *pseudotumor cerebri*. Individuals can have risk factors such as female sex, postpubertal status, obesity, or polycystic ovarian syndrome; however, these factors are not believed to directly result in increased pressure.

The term *SIH* is reserved for patients whose IH is the direct result of another condition (Table). Cerebral venous sinus thrombosis is a common cause of SIH, with the most frequent locations being the superior sagittal and transverse sinuses. Standridge and O'Brien (2) found that 11% of patients with IH had evidence of thrombosis on imaging without clear risk factors, highlighting the importance of obtaining a magnetic resonance venogram on all patients. It used to be common for patients with otitis or mastoiditis to present with IH due to sigmoid and/or jugular thromboses, previously referred to as otitic hydrocephalus. Fortunately, the modern use of antibiotics for these infections has greatly reduced the frequency of cases. Hypercoagulability in the acute postpartum period, oral contraceptive use, cancer, and various inherited coagulopathies can result in thrombosis and thus SIH. Because cerebrospinal fluid (CSF) is reabsorbed into the venous circulation, the prevailing theory is that the increased venous pressure results in reduced reabsorption and thus increased ICP.

Infection involving the intracranial components, such as meningitis or meningoencephalitis, can also result in SIH. Intracranial hypertension is an early presentation in bacterial meningitis, probably due to cerebral hyperemia. Later presentation of SIH is due to cerebral edema, obstructive hydrocephalus, infarction, and venous thrombosis resulting from the infection. The literature is filled with case reports of disseminated Lyme infection and IH. Presentation is often early in the course and is believed to be the result of decreased CSF absorption resulting from meningovascular inflammation.

Craniosynostosis due to premature closure of multiple sutures can lead to IH due to reduced intracranial volume and distortion of the intracranial venous sinuses.

The Table lists medications that have been strongly linked with SIH. Minocycline, tetracycline, and doxycycline have long been known to cause SIH. This is notable because many pediatricians use minocycline as first-line treatment of acne and are not aware of this potential adverse effect. Secondary IH due to minocycline has 3 separate presentation periods in relation to medication initiation: early (2 months), delayed (>2 months), and indeterminate (asymptomatic). Cessation of the medication results in return to normal pressure within 1 month, although rarely some patients continue to have symptoms.

Patients receiving growth hormone replacement for growth hormone deficiency can develop symptoms of IH. The most common scenario is complaints of headache and subsequent discovery of optic nerve edema. Patients typically present during the first 12 weeks of treatment, although it can occur at any time. Treatment consists of acetazolamide and cessation of the growth hormone until symptom resolution. Once all symptoms have resolved, patients can be started on a lower dose with gradual titration often without return of symptoms.

Long-term corticosteroid use can result in SIH; it is felt to be due to the weight gain associated with long-term corticosteroid use. A rapid wean or withdrawal is linked to a rebound increase in ICP and symptoms consistent with SIH. Gradual weaning with reduction by no more than 50% over the course of a few weeks reduces the chance of occurrence.

The adult criteria for PIH are referred to as the modified Dandy criteria. These criteria stipulate that a patient has 1) signs and symptoms of raised ICP (headache, nausea, vomiting, transient visual obscurations, or papilledema), 2) absence of localizing neurologic signs except for unilateral or bilateral abducens nerve palsy, 3) CSF opening pressure greater than 25 cm H_2O with normal composition, and 4) normal to small ventricles as demonstrated by computed tomography (today, magnetic resonance imaging [MRI] is the modality of choice). (3)

TABLE. **Common Causes of Secondary Intracranial Hypertension**

MEDICAL CONDITIONS	MEDICATIONS
Cerebral venous thrombosis	Tetracycline class (minocycline/tetracycline/doxycycline)
Meningitis/meningoencephalitis	Growth hormone
Intracranial hemorrhage	Corticosteroids (especially withdrawal)
Brain tumor	Lithium carbonate
Lyme disease	Cyclosporine A
Demyelinating disease/multiple sclerosis	Cytarabine
Leukemia/lymphoma	Nalidixic acid
Pregnancy/eclampsia	Retinoic acid
Crohn disease	Vitamin A excess
Hydrocephalus	Vitamin D (deficiency)
Craniofacial syndrome	Oral contraceptives (likely secondary to venous thrombosis)
Chiari malformation	
Traumatic brain injury	
Refeeding syndrome	
Adrenal insufficiency (often on corticosteroids)	
Hypoparathyroidism (early in correction)	
Vitamin A deficiency	

Further revisions to the criteria for PIH by Friedman and colleagues (4) have attempted to update the criteria and include requirements for pediatric cases and those presenting without optic nerve edema. Concerns have been raised that these stringent criteria will result in missed cases and increased potential morbidity. The pediatric normative values for CSF opening pressure have recently received scrutiny. Older studies determined normal opening pressures of 18 cm H_2O or less for children younger than 8 years and of 25 cm H_2O or less for children 8 years or older (mirrors adult normal values). Recent articles have questioned these values. Using 44 pediatric patients who had a sedated LP, Lee and Vedanarayanan (5) found a mean opening pressure of 20.3 cm H_2O. Avery et al (6) observed a mean opening pressure of 19.8 cm H_2O, and the 90th percentile for their cohort was 28 cm H_2O. They also found increases with moderate sedation and increased BMI. Both studies included patients with demyelinating and white matter disorders as well as healthy patients. Other published studies have shown increased opening pressures in patients with demyelinating conditions. Lee et al conducted a separate analysis of patients diagnosed as having demyelinating disorders and found that they had a mean opening pressure of 21.5 cm H_2O, higher than the total population mean of 20.3 cm H_2O. Morgan-Followell and Aylward (7) evaluated age- and sex-matched cohorts of patients with demyelinating conditions and PIH for comparison and found no statistically significant difference in the opening pressure between the 2 groups. This suggests that demyelinating conditions have elevated opening pressures and that including them in a normative population may falsely elevate the average. Fortunately, the debate surrounding the normal pressure is rarely needed because patients often have opening pressures well above either cutoff point. However, to avoid additional morbidities by missing cases, the authors prefer to use the older criteria.

EPIDEMIOLOGY

Traditionally, PIH is thought of as a rare disease. Outside the United States, the annual pediatric incidence is estimated to be 0.47 per 100,000 in Germany and 0.6 to 0.9 per 100,000 in the provinces of Nova Scotia and Prince

Edward Island. A recent study found an annual US PIH incidence of 0.63 per 100,000 children in a typical Midwestern city. (8)

Pediatric PIH is typically divided into prepubertal and pubertal groups. Those who have entered or completed puberty have the same risk factors as adults. Studies have shown a near 1:1 female to male distribution in prepubertal patients compared with the 4:1 ratio seen in adults. (9) In 2 separate studies, obesity was not shown to increase the risk of PIH in prepubertal patients. Examination of pediatric patients in a large IH registry found a significantly higher BMI (30.7 versus 21.6) in postpubertal patients with PIH compared with their prepubertal counterparts. (10)

The literature contains few reports of familial links with PIH, mostly parent and offspring relationships. Affected siblings have also been reported.

CLINICAL ASPECTS AND DIAGNOSIS

There is variability in clinical presentation depending on age (see the case presentations at the beginning of the article), with symptoms tending to be less evident in younger individuals. In prepubertal children, optic disc edema incidentally discovered on routine eye examination is reported in up to 33% of patients. Asymptomatic children are typically boys, younger, and less likely to be obese compared with their symptomatic counterparts.

The most common symptom reported is headache, in up to 96.5% of patients. The headaches are usually constant, with some variability in severity throughout the day. They are most often severe in the morning and can be exacerbated by maneuvers such as Valsalva, lying supine, bending over, or coughing.

The ophthalmic examination is crucial for the diagnosis and subsequent management of IH. The fundamental parts include pupillary and ocular motility assessments, color vision testing, visual acuity, and funduscopic examination (with attention to the optic nerve). Humphrey or Goldmann visual field testing, optical coherence tomography of the retinal nerve fiber layer, and fundus autofluorescence are useful tools for initial evaluation and serial monitoring.

Optic disc edema is frequently seen in IH and is often bilateral. Any patient presenting with unilateral edema should raise concerns for other diagnoses, such as optic neuritis. Up to 17.8% lack optic edema at presentation but have other symptoms consistent with elevated ICP (headache, nausea, vomiting, transient visual obscurations) and documented elevated opening pressure. The exact mechanism by which optic disc edema develops is poorly understood, and those lacking edema are believed to have unique anatomical variants at the level of the optic canal.

At presentation, up to 85% of patients able to participate in visual field testing demonstrate a visual field abnormality. Enlarged blind spots are the most common visual field defect, with peripheral constriction, nasal field loss, and inferior arcuate defects also seen. Visual acuity is often not affected unless severe edema is present and thus should not be used as a method to exclude the diagnosis of IH or to monitor progress. Transient visual obscurations are often reported as brief episodes (<30 seconds) of monocular or binocular blurring or "graying out" of vision. Events occur multiple times per day and can be provoked by position changes.

Cranial nerve VI palsy is seen in 12% to 60% of patients. Patients often note diplopia when looking in the direction of the nerve palsy. Often unreported until asked, pulsatile tinnitus is experienced by up to 44.3% of patients. Distinct from common tinnitus, it is often described as a "whooshing" sound coinciding with the heartbeat.

Direct funduscopic examination is used to visualize the optic nerve for the presence of edema and, if present, is graded using the Frisén scale. (11) The Figure contains clear examples of the different grades of optic edema. (9) Spontaneous venous pulsations are seen in 87% to 90% of the healthy population. They have also been seen in those with documented elevated ICP, making decisions regarding further evaluation and treatment difficult. The help of an ophthalmologist should be solicited for detailed evaluation of these findings.

If the child is able to cooperate, Humphrey or Goldmann visual field testing is performed at the initial and subsequent visits. These tests evaluate the peripheral vision using a dome-shaped screen on which visual targets are projected in static (Humphrey) or dynamic (Goldmann) fashion. The ability to maintain gaze fixation for 15 to 30 minutes is essential for visual field test reliability; thus, usually these tests are performed on older, more cooperative children. Optical coherence tomography is a noninvasive test that evaluates the thickness of the retinal nerve fiber layer surrounding the optic nerve and is used to monitor the resolution of papilledema during treatment.

It is important to note that optic disc drusen, globular aggregates of hyaline and calcium salts on the optic nerve, can mimic optic nerve edema. When this occurs, the patient is said to have pseudopapilledema. B-scan ultrasonography and fundus autofluorescence photography can be used by the ophthalmologist to differentiate between true papilledema and pseudopapilledema. Optical coherence tomography has been explored as a method of differentiating between the two as well, but as yet this method is

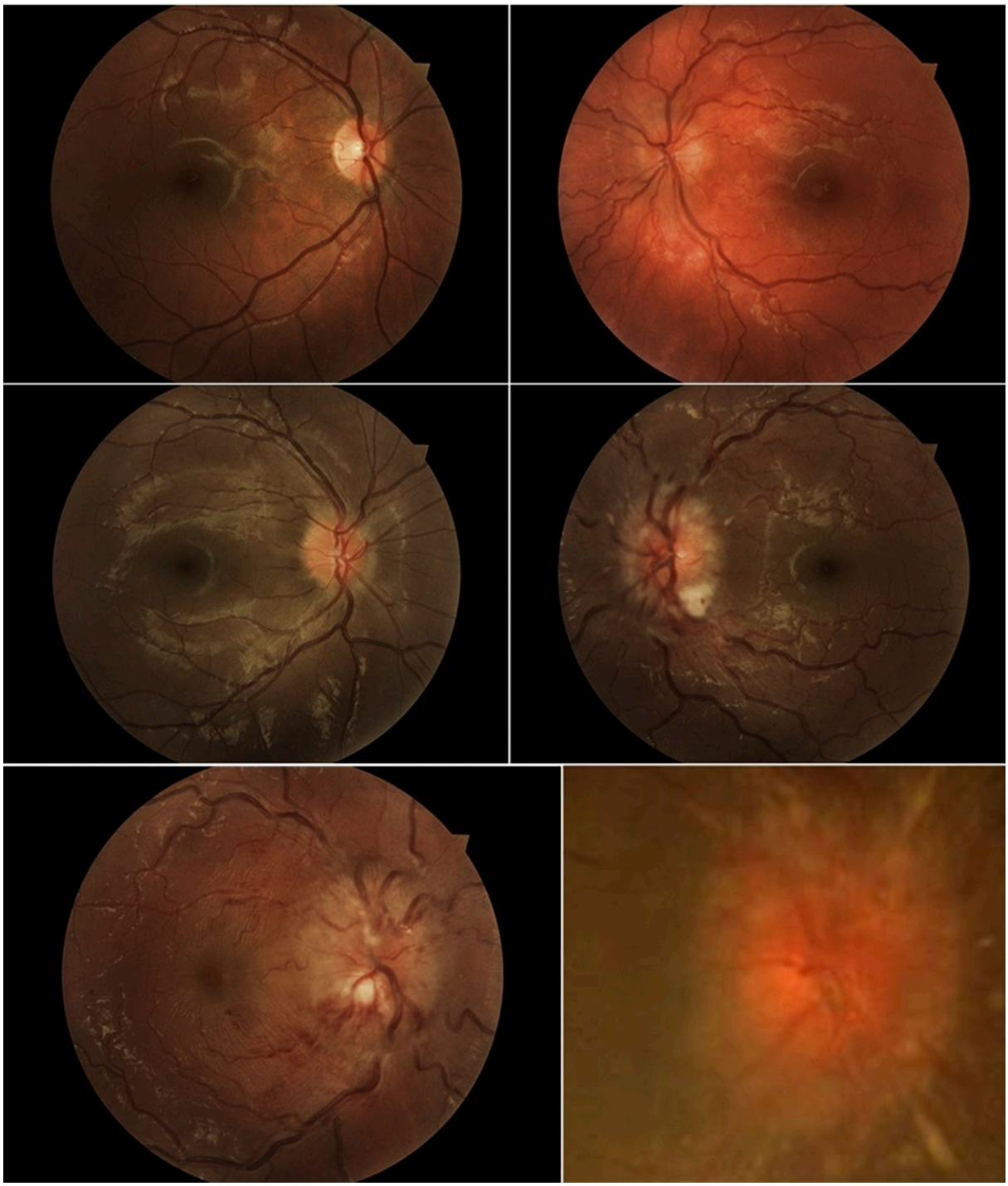

Figure. Optic nerve edema stages. Left to right. Top row: normal (0), stage 1. Middle row: stage 2, stage 3 (note cotton wool spot with hemorrhage at 5 o'clock). Bottom row: stage 4 (note hemorrhages at 1-2 and 6-8 o'clock, retinal striae due to severity of edema), stage 5. (Reprinted with permission from Elsevier. [9])

somewhat less reliable. The most reliable method is currently fluorescein angiography. (12)

Neuroimaging (computed tomography or MRI) should be performed as an initial step in the evaluation to rule out mass lesion, hydrocephalus, or other conditions that would be a contraindication to performing the LP. All pediatric patients diagnosed as having IH should eventually undergo MRI and magnetic resonance venography to rule out other

secondary causes, such as sinus venous thrombosis. Although the MRI should be devoid of structural abnormalities, subtle findings suggestive of IH can sometimes occur, including empty or partially empty sella turcica, dilation of the optic nerve sheath, flattening of the posterior globe, anterior protrusion of the optic nerve head, tortuosity of the optic nerve, and distal transverse sinus stenosis. Although the presence of 1 or more of these subtle radiographic findings increases the probability of IH, lack of these findings does not decrease its likelihood. Bilateral transverse sinus stenosis is thought to be a consequence and not a cause of the IH as studies have shown normalization of the stenosis after high-volume LP. (13)

One of the most important diagnostic tools, and also the most fraught with error, is the LP. The most common error is improper positioning. Opening pressure measurements should not be performed with the patient in the seated position. Accepted positioning is in lateral decubitus with the legs and head extended at the time of pressure measurement. For sedated patients, spontaneous respirations with normal arterial carbon dioxide is preferred. One pediatric study found that an increase of 1 kPa in end-tidal carbon dioxide increases CSF pressure by 3.5 to 12 cm H_2O.

Popular convention is for withdrawal of large CSF volumes and normalization of the pressure in an effort to protect the vision. An adult study found that the average time to return to initial pressure after LP is 82 minutes. The authors' experience found that normalizing higher pressures results in increased rates of post-LP headache. Reduction of less than 10 cm H_2O of pressure still results in transient headache relief with fewer post-LP headaches.

MANAGEMENT

Management of a patient with IH often requires a multidisciplinary approach with neurology and ophthalmology. If clinically indicated, neurosurgery, psychology, psychiatry, dietetics, physical therapy, hematology, and/or endocrinology may be involved.

Often, pubertal and adult patients report weight gain in the 12 months before diagnosis. Numerous adult studies have shown that aside from removing an offending medication, weight loss is the only other modifiable risk factor shown to affect the ICP. One of the first studies treated patients with a low-calorie rice diet to treat their IH. (14) Study participants lost 11 to 56 kg over the treatment period, resulting in papilledema resolution without medication use. The 2 symptomatic patients reported complete resolution of symptoms in addition to resolution of their papilledema.

The goal of pharmacologic management is to decrease CSF production and in turn lower the ICP. Acetazolamide is considered the first-line treatment for IH due to its relative tolerability and lack of need for repeated laboratory monitoring. Patients may complain of food having a metallic taste to it, especially carbonated beverages. Transient paresthesia, metabolic acidosis, and slight increase in urination are other common adverse effects. The metabolic acidosis is often asymptomatic, and patients tolerate serum carbon dioxide levels down to 15 mmol/L without the need for supplementation. The typical adolescent dosing is 1 to 2 g divided twice daily (BID). Doses greater than 2 g failed to show much added benefit at the risk of increased adverse effects. In younger children, doses of approximately 20 to 25 mg/kg per day divided BID are typical, although doses as high as 100 mg/kg per day can be tolerated.

Furosemide is the second-line agent due to its diuretic nature and requirement to monitor for hypokalemia. Fortunately, most pediatric patients taking furosemide do not need potassium replacement above increased dietary intake. Typical dosing is 1 to 2 mg/kg per day divided BID. Studies have shown a synergistic effect when used in combination with acetazolamide.

Due to similar chemical structures to sulfonamides, there is a potential risk of cross-reactivity to acetazolamide and furosemide in patients with reported allergy. An adult study reviewed the medical records of patients with self-reported sulfa allergy who were given regimens of acetazolamide, furosemide, or a combination for treatment of IH. (15) No evidence of cross-reactivity was found, and the study suggests that patients with a reported sulfa allergy can safely take these medications.

Topiramate is also used in patients who are not able to tolerate acetazolamide or furosemide. It is similar to acetazolamide and has a similar mode of action. In an open-label study, 40 adult patients were alternately assigned to receive topiramate (100–150 mg/d) or acetazolamide (1,000–1,500 mg). Visual field grades were followed, and both treatment groups demonstrated statistically significant improvement.

Long-term use or rapid weaning of corticosteroids can result in SIH, although they used to be the first-line choice for treatment of PIH before acetazolamide. Their use has declined given the improved outcomes and fewer adverse effects with medications used today. However, in instances of severe visual deficits at presentation, corticosteroids used in conjunction with acetazolamide does result in a better outcome. The recommended dose is largely anecdotal,

mirroring what is used in optic neuritis and other inflammatory neurologic disorders. Patients are initially given intravenous methylprednisolone 20 mg/kg (maximum 1 g) daily for 5 days followed by an oral taper.

In pediatrics, surgical interventions for IH are rarely necessary. Surgery is considered in patients for whom medical therapy cannot be tolerated, patients with severe optic nerve swelling with concerns of permanent vision loss, or patients with persistently elevated ICP with associated papilledema or pain despite maximizing pharmacologic options. The main procedures considered involve CSF shunting and optic nerve sheath fenestration (ONSF).

It is believed that ONSF preserves optic nerve function by redirecting pressure away from the optic nerve head, the area of the most tenuous blood supply. It does not directly address the elevated ICP. Surgery involves exposing the optic nerve and making openings in the sheath to relieve the pressure. Risks include ischemic optic neuropathy (especially in severely edematous or ischemic nerves), transient blindness, pupillary mydriasis and retrobulbar hemorrhage. Unilateral fenestration can result in resolution of edema in the unfenestrated eye; however, common practice is to perform bilateral fenestration. Typically, ONSF is not performed on an acutely swollen nerve but rather when the swelling is more under control. Signs of optic nerve ischemia, including cotton wool spots, increase the risk to the optic nerve and thus should resolve before the procedure is performed.

A CSF diversion is more effective in patients when pain is the primary symptom. In addition, it is used to protect vision in the acute presentation, where the risk of ischemic neuropathy due to ONSF is high. There are 2 options for shunt placement: lumboperitoneal or ventriculoperitoneal. The goal is to mechanically reduce the CSF pressure by direct CSF diversion. Unfortunately, the myth that ventricular diversion is superior still persists. In one study, the outcomes of 25 patients shunted for their IH were reviewed. (16) Shunt failure was defined as continuation or return of presenting symptoms with radiographically verified shunt placement and normal opening pressure. Those requiring revision had radiographic evidence of shunt disturbance, blockage, or increased opening pressure. Failure rates were similar at 11% and 14% for lumbar and ventricular shunts, respectively. Revisions were more common in lumbar versus ventricular shunts (60% versus 30%).

Attempts have been made to stent sinus venous stenosis in adults with varying success. Headache resolution is seen in up to 84.6% of patients, and resolution of papilledema in 62.5% to 100%. Transient postoperative headache or partial hearing loss on the treated side are the most common adverse effects reported. Rarer adverse effects include hemorrhage, venous perforation due to the guidewire, and temporary unsteadiness. Use of stenting has not been studied in pediatrics.

PROGNOSIS

Despite the previously held belief that this is a relatively benign condition, the potential for permanent vision loss and chronic pain are significant morbidities. When properly treated, headaches typically resolve within the first few weeks. Resolution of papilledema averages 4.2 to 5 months. Minor visual field deficits often fully resolve, sometimes lagging behind resolution of the papilledema. There is literature to suggest that grade 3 or higher papilledema is a high predictor of permanent visual deficits. Another study found that postpubertal status resulted in a worse visual outcome. Recurrence is seen in 18% to 20% of patients. A series of adult patients were followed after weaning off acetazolamide for recurrence of their optic nerve edema. (17) All the patients lost weight from the time of diagnosis to medication wean, and those with recurrence had an average BMI 5.5% higher than at diagnosis. This was compared with an average decrease of 17.9% in BMI in those without recurrence.

Up to two-thirds of patients develop new headache symptoms that are reported as different from their IH headache. Most are diagnosed as having migraine without aura or episodic tension-type headache and respond to common prophylactics used for these conditions.

Summary

- Based on research evidence, obesity does not play as large of a role in primary intracranial hypertension in prepubertal patients. (6)(7)
- Consensus supports the crucial role of the ophthalmic examination in diagnosis and subsequent management of intracranial hypertension. This includes pupillary and ocular motility assessments, color vision testing, visual acuity, funduscopic examination, Humphrey or Goldmann visual field testing, and optical coherence tomography.
- Based on strong research and consensus, the lumber puncture opening pressure measurement should be performed with the patient in the lateral decubitus position with the legs and head

extended. Sedated patients should have spontaneous respirations with normal arterial carbon dioxide concentrations.

- The literature supports the consensus that minocycline, and the related tetracycline class of medications, can cause secondary intracranial hypertension. This is of concern because many pediatricians use minocycline in the treatment of acne and are not aware of this potential adverse effect.
- Based on strong research studies, aside from removing an offending medication, weight loss is the only modifiable risk factor shown to affect the intracranial pressure and prevent recurrence. (11)(14)
- Based on research and consensus, acetazolamide is the first-line treatment for intracranial hypertension due to its relative tolerability and lack of need for repeated laboratory monitoring.
- Some research has shown that lumbar shunts have equal failure rates compared with ventricular shunts. (13)

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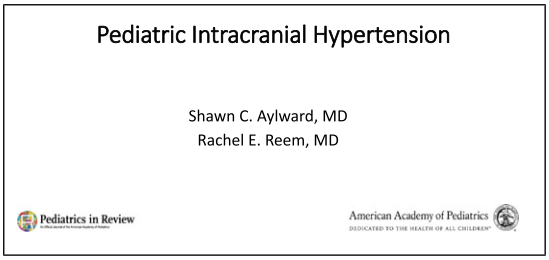

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1. A 14-year-old girl presents to the clinic for a health supervision visit. She has a history of primary intracranial hypertension, which is now resolved. She is concerned about facial pustular acne, especially as she starts high school. Which of the following medications would be the most appropriate first-line treatment for this patient?
   A. Clindamycin/tretinoin.
   B. Erythromycin/benzoyl peroxide.
   C. Minocycline.
   D. Oral contraceptives.
   E. Retinoic acid.
2. A 9-year-old obese boy is seen in an urgent care clinic for headaches and vision problems for the past 2 weeks. According to the modified Dandy criteria, the presence of which of the following would exclude a diagnosis of primary intracranial hypertension?
   A. Abducens palsy.
   B. Enlarged blind spot.
   C. Localizing neurologic signs.
   D. Spontaneous venous pulsations on funduscopic examination.
   E. Transient visual obscurations.
3. You refer a 5-year-old boy to the ophthalmology clinic because his mother is concerned that he dislikes reading and holds his book too far away from his face. You receive a call from the ophthalmologist stating that his visual acuity is normal but he has bilateral optic disc edema. Which of the following best represents the percentage of prepubertal pediatric patients that have optic disc edema incidentally discovered on routine eye examination?
   A. 1%.
   B. 10%.
   C. 20%.
   D. 25%.
   E. 30%.
4. After receiving a lumbar puncture and confirming that his cerebrospinal fluid pressure is elevated at 30 cm H_20, the patient described in Question 3 is started on acetazolamide. His mother would like to know how long the papilledema will take to resolve. Which of the following represents the average time that it takes for papilledema to resolve?
   A. 2 weeks.
   B. 6 weeks.
   C. 2 months.
   D. 5 months.
   E. 1 year.
5. A 13-year-old girl who presented with headaches at age 12 years and was diagnosed as having primary intracranial hypertension returns to the clinic with recurrence of her headaches after weaning her off acetazolamide. She also notes nausea and photophobia. You suspect a diagnosis of migraines but wonder whether you should check a cerebrospinal fluid opening pressure. Which of the following represents the recurrence rate of primary intracranial hypertension?
   A. 10%.
   B. 20%.
   C. 30%.
   D. 40%.
   E. 50%.

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