

Acetazolamide for Idiopathic Intracranial Hypertension: An Up-to-Date Review in 2025

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Abstract: Idiopathic Intracranial Hypertension (IIH) is characterized by elevated intracranial pressure in the absence of secondary causes. The treatment of IIH entails a combination of lifestyle modifications, medical therapy, and surgical intervention. This article aims to evaluate the role of acetazolamide in the treatment of IIH and identify its mechanism of action, efficacy, and side effect profile. This article also aims to discuss acetazolamide's role in conjunction with other treatment modalities.

Keywords: acetazolamide, idiopathic intracranial hypertension, papilledema

Introduction

Idiopathic Intracranial Hypertension (IIH), previously known as pseudotumor cerebri, is a condition marked by raised intracranial pressure specifically in the absence of intracranial mass lesions, thrombus, or other secondary causes. Prompt diagnosis and management of IIH prevents negative outcomes including impaired quality of life, permanent vision loss, and debilitating headaches.¹ The diagnosis of IIH can be established via the modified Dandy criteria, which includes (1) signs and symptoms of intracranial hypertension, (2) a normal neurological exam (excluding papilledema and cranial nerve VI palsy), (3) neuroimaging that is not suggestive of the aforementioned secondary causes of intracranial hypertension, (4) an increased opening pressure as measured on a lumbar puncture (LP) with normal cerebrospinal fluid (CSF) constituents and (5) no other causes of increased intracranial pressure present.²

Management of IIH entails a combination of lifestyle modifications, medical therapy, and surgical intervention. Weight loss is curative for many patients and is frequently recommended.³ Medical therapy often entails the use of carbonic anhydrase inhibitors to reduce intracranial pressure (ICP). Acetazolamide is a carbonic anhydrase inhibitor that serves this role well. Its role in the management of IIH has been extensively studied, including its effects on subjective parameters such as quality of life and symptomatology as well as objective and sensitive structural effects on the retina and optic nerve defined by eye imaging such as retinal photography and optical coherence tomography (OCT), as well as significant lowering of intracranial pressure.^{1,4} We aim to provide a comprehensive review of acetazolamide including its pharmacological characteristics, efficacy, and side effect profile in isolation and in conjunction with alternative therapies.

Historical Context

The development of acetazolamide traces back to the mid-20th century during the search for an effective carbonic anhydrase inhibitor that would provide adequate diuresis in patients with congestive heart failure and acid-base disturbances.⁵ Carbonic anhydrase was first isolated in red blood cells as early as 1933 by researchers at both the University of Pennsylvania and Cambridge University.⁶ Its role in acid-base regulation and its presence in a variety of tissues became well established shortly after, and the enzyme became a central figure researched in acid-base physiology.

The inhibition of carbonic anhydrase was first noted with the use of sulfonamide antibiotics.⁵ The search for more potent inhibition of carbonic anhydrase ultimately led to the synthesis of acetazolamide in 1952 by William Schwartz and James Clapp.⁵ Its action as a carbonic anhydrase inhibitor led to its discovery as a diuretic. The role of acetazolamide in the treatment of IIH would not be entertained until 1974, after a study published by McCarthy and Reed showed its effect on reducing CSF production.⁷ Prior to this, medical management of IIH entailed the use of corticosteroids and thiazide diuretics, but their side effect profiles and disappointing efficacies limited their use.^{8,9} In 1978, Gücer and Viernstein published a study showing a reduction of intracranial pressure before and after acetazolamide treatment in two patients with IIH.¹⁰ Subsequent studies supported the use of acetazolamide in clinical practice, such as those by Lubow and Kuhr as well as Tomsak,^{11,12} and acetazolamide soon became a common agent in the treatment of IIH. In 2014, its efficacy was further characterized by the Idiopathic Intracranial Hypertension Treatment Trial (IIHTT), showing an improvement in visual field sensitivity, improvement in quality of life, lowering intracranial pressure, and reduction in papilledema.¹

Today, acetazolamide is commonly used in the medical management of IIH. Its role is well established in the management of other medical conditions including, decompensated congestive heart failure,¹³ glaucoma,¹⁴ macular edema secondary to retinitis pigmentosa,¹⁵ and altitude sickness.¹⁶ Since its discovery, its use has expanded to include epilepsy,¹⁷ Meniere's disease,¹⁸ and certain types of myotonia.¹⁹

Acetazolamide: Mechanism of Action in the Choroid Plexus

Cerebrospinal fluid (CSF) is a colorless fluid that encompasses the brain and spinal cord. Its function is multifaceted and includes shock absorption, nutrient delivery, waste elimination, and ionic balance. The level of intracranial pressure abides by the Monro-Kellie doctrine, which states that the sum volume of the brain parenchyma, CSF, and blood flow must remain constant, while a change in each of these compartments results in a compensatory change in the other two.²⁰ An adaptable calvaria may also play a role in the determination of intracranial pressure.²¹ Thus, the rate of production of CSF in conjunction with its rate of resorption helps determine ICP at any given moment. A rise in the intracranial volume due to a pathological state reflects a breakdown of the Monro-Kellie homeostatic mechanism, resulting in an increase in the intracranial pressure.

The production of CSF reflects a filtration process that occurs in the lateral, third, and fourth ventricles via the choroid plexus. The functional unit of the choroid plexus is highly specialized and includes capillaries and cuboidal epithelial cells known as ependymal cells. Ependymal cells are polarized, which allows for the migration of ions including sodium, chloride, and hydrogen, as well as macromolecules such as glucose and protein into the ventricular space, creating the substance of the CSF at a rate of 500 mL/day.²² As such, its composition is like that of plasma. After its production, CSF passes through the ventricular system of the brain and into the subarachnoid space where arachnoid granulations resorb its contents.

Carbonic anhydrase mediates filtration across capillaries and ependymal cells and plays a principal role in the rate of filtration across the choroid plexus. Specifically, carbonic anhydrase catalyzes the reaction $\text{H}_2\text{CO}_3 \rightleftharpoons \text{H}_2\text{O} + \text{CO}_2$.²³ This reaction enables the transport of sodium ions, chloride ions, and water across the cell membranes of ependymal cells into the ventricles. Acetazolamide inhibits carbonic anhydrase, thereby directly reducing the transport of sodium ions across the choroid plexus epithelium and thus the rate of CSF production and intracranial pressure.²⁴

In fact, the effects of acetazolamide on intracranial pressure are multifactorial. Carbonic anhydrase is a ubiquitous enzyme that is also present in the ciliary epithelium of the eye, red blood cells, and the proximal convoluted tubule of the kidney among other locations. As such, its effects on the body are varied and include metabolic acidosis with a compensatory respiratory alkalosis as well as weight loss, both of which independently reduce intracranial pressure. Carbonic anhydrase inhibition by acetazolamide also maximally increases cerebral blood flow to the choroid plexus and is often used in research to increase cerebral blood flow.^{25,26} However, animal study models show that acetazolamide still decreases total CSF production despite increasing cerebral blood flow, suggesting that these two phenomena may be uncoupled with administration of acetazolamide.²⁷

Pharmacology

Acetazolamide is a sulfonamide derivative that acts as a non-competitive inhibitor of carbonic anhydrase throughout the body. It is most commonly administered orally for IIH, although it can be given intravenously. Oral administration typically results in a bioavailability of about 90%. Once absorbed, at least 90% of acetazolamide is bound to albumin which enables its wide distribution throughout the body including the brain. Its onset of action is roughly 1 hour with its peak effect at 2–4 hours. Acetazolamide has a half-life of 10–15 hours in healthy adults and thus sometimes requires twice-a-day dosing for IIH. It is not metabolically altered in the body and is excreted renally.²⁸ A reasonable starting dose of acetazolamide for IIH is 500 mg twice a day. This can be titrated up to 3–4 g daily for maximal effect if needed. Oral administration includes immediate release tablets (250 mg each) and extended-release capsules (500 mg each).

Potential Drug Interactions

Acetazolamide has variable interactions with other medications such as lithium, diuretics, and antiepileptics, potentially leading to adverse effects. Acetazolamide can increase renal excretion of lithium, decreasing its overall efficacy.²⁹ For example, this may be in part why acetazolamide can be considered in the treatment for lithium-induced diabetes insipidus.³⁰ Acetazolamide can also decrease excretion of antiepileptic drugs such as phenytoin and can potentially increase toxicity. This effect was described in a 1977 case report on the effect of acetazolamide and acceleration of anticonvulsant osteomalacia.³¹ In addition, salicylate (aspirin) may increase unbound plasma acetazolamide concentrations, increasing toxicity from acetazolamide.³² This is of particular importance in patients with chronic kidney disease, who are at an increased risk of metabolic acidosis.³³ Careful monitoring of electrolytes and renal function is required in these patients with renal disease during therapy when drug interactions are likely. The IIHTT showed that following electrolytes and CBC was not cost effective as potassium loss is rare when acetazolamide is the only diuretic.¹

Clinical Evidence on Acetazolamide for IIH

The Idiopathic Intracranial Hypertension Treatment Trial (IIHTT) was a landmark, multicenter study published in 2014 that demonstrated improved visual outcomes in patients with IIH who use acetazolamide.¹ The trial comprised 165 participants with IIH and mild vision loss who underwent a low-sodium weight reduction diet coupled with randomization to either acetazolamide or placebo, each given at the maximally tolerated dosage up to and including 4 grams a day as a twice a day schedule. It specifically reported statistically significant improvements in perimetric mean deviation, papilledema grade, quality of life, and lumbar opening pressure. Subsequent studies have corroborated the results of the IIHTT by focusing on structural changes related to papilledema as observed on OCT imaging of the retina and optic nerve. The IIHTT OCT substudy further characterized the effects of acetazolamide compared to placebo on objective parameters of OCT, such as total retinal thickness (TRT), retinal nerve fiber layer (RNFL), optic nerve head volume (ONH), and ganglion cell layer (GCL) measurements.³⁴ Indeed, modern clinical practice extrapolates from the results of the IIHTT in the management of symptoms of patients with IIH, utilizing acetazolamide at an effective dose in combination with weight reduction.

Intracranial Pressure

Intracranial pressure can vary widely over a 24-hour period.³⁵ Nonetheless, a lumbar opening pressure of ≥ 25 cm H₂O is supportive of a diagnosis of IIH in the absence of secondary causes of intracranial hypertension. Given its role in reducing CSF production, it stands to reason that acetazolamide reduces lumbar opening pressure. The IIHTT found a mean reduction in lumbar opening pressure of -112.3 mm H₂O in the acetazolamide + diet group and -52.4 mm H₂O in the placebo + diet group between baseline and 6 months, suggesting that acetazolamide is effective at reducing intracranial pressure. Therefore, many recommend pausing acetazolamide for at least two days prior to a lumbar puncture for the subset of IIH patients who already have been prescribed this medication during their work-up.

Visual Fields

The most common visual field defect from IIH is a concentrically enlarged blind spot, caused by a refractive scotoma from a relative peripapillary hyperopia and in some cases, peripapillary subretinal fluid.^{36,37} Other visual field defects

include arcuate scotomas and nasal steps. Generalized constriction of the visual field from superior and inferior dense arcuate defects implies a greater severity of effect of intracranial hypertension on optic nerve function and suggests a poorer visual prognosis.³⁸ Central visual field defects may also occur due to either direct damage to the maculo-papillary nerve bundle or subretinal fluid tracking from the optic nerve head into the macula.³⁹ The beneficial effects of acetazolamide on improving visual field defects has been well established by the IIHTT. The primary outcome variable of the IIHTT was change in perimetric mean deviation (PMD) from baseline to month 6 in the eye with the most severe vision loss at baseline comparing acetazolamide vs placebo. Indeed, subjects on acetazolamide showed a reduction of PMD of 1.43 dB versus 0.71 dB in the placebo group.¹

Optic Disc Edema Grading

The Frisén grading scale for papilledema is a valuable clinical marker for the severity of papilledema. Higher grades suggest severe intracranial hypertension and confer a higher risk for permanent vision loss.⁴⁰ This may justify aggressive treatment with higher doses of acetazolamide, which may adequately reduce the extent of swelling (Figures 1 and 2). Results from the IIHTT suggest that acetazolamide and weight loss effectively reduce the extent of papilledema as measured by the Frisén grading scale compared with placebo and weight loss in patients with mild to moderate severity of visual field defects.¹

Optical Coherence Tomography

The advent of the OCT provided many useful objective grading measurements for papilledema. These measurements are often used to assess progression of papilledema over the course of treatment and include RNFL thickness, TRT, and ONH volume. These measures provide an alternative, more continuous means of monitoring optic nerve head edema compared with the Frisén grading system. The IIHTT OCT substudy showed a greater reduction of RNFL thickness, TRT, and ONH volume in the acetazolamide group compared with the placebo group.³⁴ However, interestingly, the deflection of Bruch's membrane towards the vitreous was unchanged in the placebo group, even in eyes with reduced optic disc edema. A follow-up study described the presence of peripapillary wrinkles, radial inner retinal folds, peripapillary outer

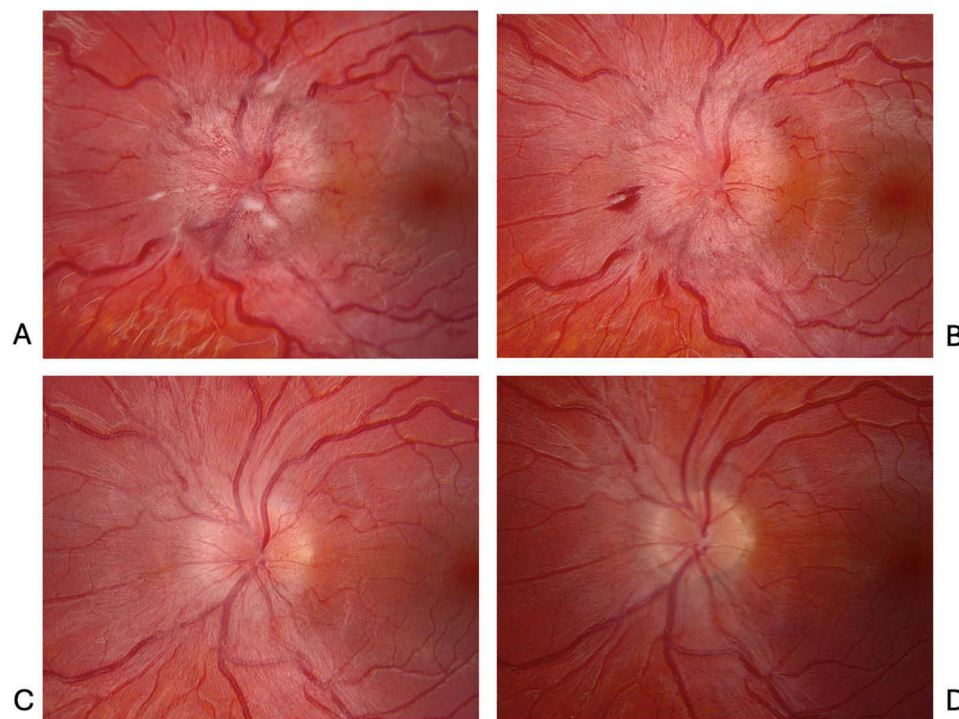


Figure 1 Interval resolution of papilledema in a 17-year-old female on 1.5–2 g of Acetazolamide twice a day. (A) On presentation (B) 9 days later (C) 29 days later (D) 57 days later.

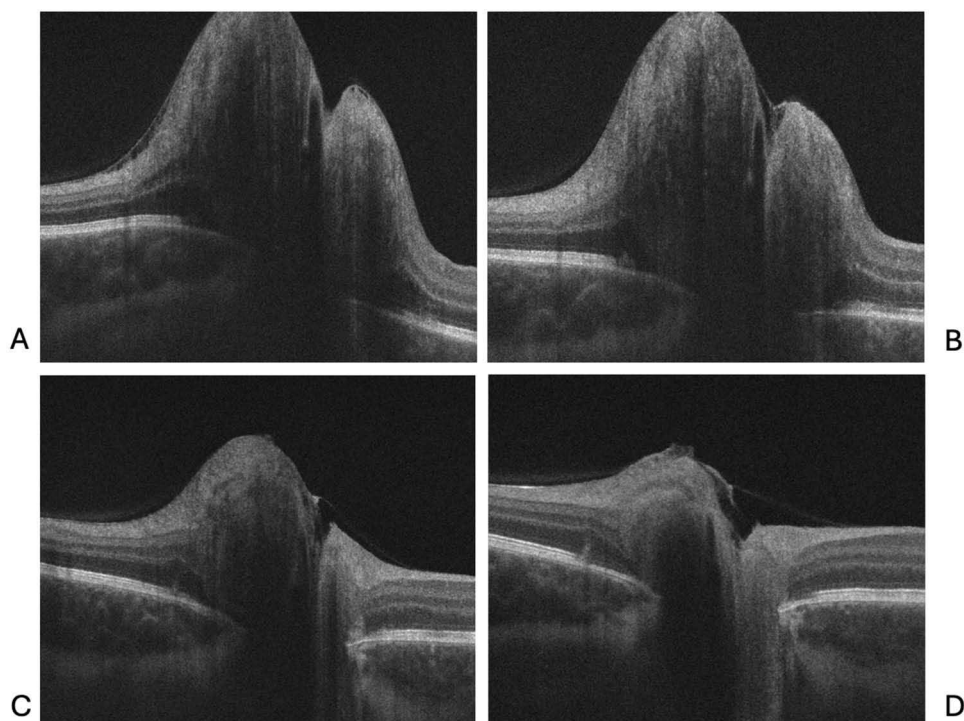


Figure 2 Interval resolution of papilledema of the same patient, OCT images. (A) On presentation (B) 9 days later (C) 29 days later (D) 57 days later.

folds, and choroidal folds, each representing different mechanical deformations. Retinal folds decreased in frequency with resolution of papilledema, a finding that was more notable in the acetazolamide group.⁴ Reduction in choroidal folds also showed a mild correlation with a reduction in the CSF opening pressure. In addition, a change in peripapillary retinal pigment epithelium's and Bruch's membrane's positions away from the vitreous was observed in patients who take acetazolamide and/or have lost weight.⁴¹ In a separate study, it was found that thinning of the retinal ganglion cell inner plexiform layer (GC-IPL) is predictive of poor visual outcome.⁴²

Symptoms and Weight Loss

The most common symptom reported in patients with IIH is headache, followed by transient visual obscurations, pulsatile tinnitus, back pain, dizziness, photophobia, neck pain, and visual loss.¹ The IIHTT showed an improvement in quality of life as measured by National Eye Institute Visual Function Questionnaire (NEI-VFQ-25) and its 10-Item Neuro-Ophthalmic Supplement.

Radiographical Findings

Neuroimaging characteristics of raised intracranial pressure include a partially or empty sella with or without bony remodeling, cerebellar tonsillar ectopia, flattening of the posterior globes, optic nerve sheath distension, and transverse sinus stenosis.⁴³ Although these findings are suggestive of a raised intracranial pressure, they are not diagnostic in the absence of the modified Dandy criteria. Many of the radiographic changes seen reflect permanent structural changes to accommodate intracranial hypertension. For example, some patients with the so-called empty sella syndrome may show radiographic findings of bony remodeling of the sella turcica, which implies chronicity of intracranial hypertension even long after the hypertension has resolved.⁴⁴ Although acetazolamide may reduce ICP in patients with IIH, it has not been shown to reverse most of the structural changes observed on neuroimaging nor is this necessarily a goal in treatment.

Side Effects of Acetazolamide

Acetazolamide induces metabolic acidosis by inhibiting carbonic anhydrase at the level of the proximal convoluted tubule in the kidneys. This can result in metabolic derangements both in the blood and urine, including compensatory

Table 1 Side Effects of Acetazolamide©

Etiology©	Symptoms©
Metabolic acidosis©	Paresthesia© Gastrointestinal disturbance© Dysgeusia© Fatigue© Nephrolithiasis© Shortness of breath©
Idiosyncratic Rare Side Effects©	Aplastic anemia© Stevens Johnsons syndrome© Toxic epidermal necrolysis© Hepatic necrosis©

respiratory alkalosis, hypokalemia, urinary alkalosis, hypocitraturia and hypercalciuria. Metabolic acidosis accounts for most of the common side effects, which include paresthesia, gastrointestinal disturbance, dysgeusia, fatigue, nephrolithiasis and shortness of breath (Table 1).⁴⁵ Although many of these symptoms are transient and may resolve with continued use, their onset can be severe enough to discourage medication compliance. The addition of sodium bicarbonate or potassium chloride supplements sometimes aids in the management of these side effects. However, this is reserved for specific circumstances, and these supplements are not routinely prescribed. A randomized control trial published in 2016 compared the onset of adverse events between participants receiving acetazolamide versus placebo. Adverse events included paresthesia, dysgeusia, vomiting and diarrhea, nausea, and fatigue. The paper concluded that a dosage of up to 4 grams per day of acetazolamide has an acceptable safety profile despite adverse events.⁴⁶

Serious adverse effects include aplastic anemia, Stevens Johnsons syndrome, toxic epidermal necrolysis, and hepatic necrosis. Given their rarity, current literature regarding the onset of these conditions in the setting of acetazolamide is sparse. Nonetheless, the onset of these side effects necessitates immediate discontinuation of these medications.

Special Considerations for Acetazolamide

Sulfa Allergy

Sulfa allergy has often been listed as a contraindication for the use of acetazolamide due to the presence of a sulfa moiety. Indeed, sulfa allergies are well documented in antibiotics that contain an arylamine group at the N4 position. However, this does not predict cross-reactivity with sulfonamide-containing non-antibiotics such as acetazolamide. It has also been suggested that penicillin allergy may serve as a better predictor of acetazolamide allergy.⁴⁷

Pregnancy and Breastfeeding

Acetazolamide is categorized as a class C pregnancy medication. Studies in animal models have shown teratogenic effects on the fetus, but this has not been reliably reproduced in studies involving humans.⁴⁸ Clinically, acetazolamide is often prescribed when needed during pregnancy across all three trimesters. In some cases where the low risk of teratogenic effects on the fetus are of sufficient concern of patients to outweigh a treatment benefit, its use may be delayed until the second trimester. In addition, acetazolamide’s presence in breast milk has been shown to induce metabolic acidosis in feeding infants.⁴⁹ However, this data is limited to independent case reports and warrants further investigation before conclusions can be drawn regarding long-term effects.

Chronic Kidney Disease

Chronic kidney disease increases plasma concentrations of acetazolamide due to its renal excretion.⁵⁰ Thus, it is not recommended if an alternative agent can be used. If necessary, it may be considered under co-management with Nephrology. Dosing depends on the patient’s calculated creatinine clearance (CrCl). A patient with a CrCl > 50 mL/min may take up to 500 mg twice a day. Likewise, a patient with a CrCl < 10 mL/min should not take

acetazolamide. Patients between 10 and 50 mL/min may take up to 250 mg twice a day.⁵¹ Lastly, patients on hemodialysis may take up to 250 mg twice a day, while those on peritoneal dialysis may take up to 125 mg daily.^{52,53}

Nephrolithiasis

Acetazolamide's effects on the proximal convoluted tubule result in metabolic acidosis and relative urinary alkalosis. This also leads to hypocitraturia and hypercalciuria. These metabolic derangements predispose patients to the development of kidney stones. Patients may develop calcium phosphate and/or calcium oxalate kidney stones.⁴⁷ However, the presence of kidney stones was not part of the exclusion criteria in the IIHTT. The development of kidney stones only serves as a relative contraindication rather than an absolute, and acetazolamide may be continued when necessary. In such cases, some recommend adequate hydration and co-management with a Urologist or Nephrologist with regular renal ultrasounds.

Pediatric Dosing

IIH may be present in prepubescent children and adolescents. Acetazolamide can be administered in children in liquid formulation. Dosing can be started at 15–25 mg/kg/day four times a day and can be up-titrated to the equivalent of 2 g/day in children and 4 g/day in adolescents.⁵⁴ When possible, some children and adolescents may be able to tolerate the immediate release tablet or extended-release capsule formulations when applicable.

Further Treatment Considerations and Alternatives

Additional treatment regimens may be considered in the management of IIH and include both medical and surgical options (Table 2). Some patients require adjuvant therapy due to insufficient response or intolerance to acetazolamide. Although acetazolamide is first-line in many patients with IIH, its use in conjunction with other therapies also aims to utilize different mechanisms in lowering intracranial pressure and managing symptoms.

Topiramate

Topiramate's efficacy on the treatment of IIH hinges on its actions as 1) a weak carbonic anhydrase inhibitor, 2) an appetite suppressant to promote weight loss, and 3) its ability to treat migraines, which often has an overlapping symptom profile in IIH patients.⁵⁵ It thus serves as a viable alternative in patients who cannot tolerate acetazolamide and in some cases as adjunctive therapy. Topiramate's role in the management of IIH can be attributed not only to its action as a carbonic anhydrase inhibitor but also its effects on voltage-gated sodium channels, GABA potentiation, and glutamate receptor antagonism, thus offering a broader approach to intracranial pressure reduction and symptom

Table 2 Treatment of Idiopathic Intracranial Hypertension©

Type of Intervention©	Therapies©
Medical©	Acetazolamide© Topiramate© Corticosteroids© Methazolamide© Octreotide© Spironolactone© GLP-I agonists©
Surgical©	Ventriculoperitoneal shunting© Lumboperitoneal shunting© Optic nerve sheath fenestration© Transverse sinus stenting©
Weight Loss©	Diet modification© Bariatric surgery© GLP-I agonists©

management. Its use with acetazolamide is complementary, albeit with different potencies and potentially distinct molecular mechanisms, suggesting a rationale for combination to achieve a more profound reduction in CSF production of ICP. According to a 2007 open-label study, topiramate improves visual field function as effectively as acetazolamide.⁵⁶ The combination of acetazolamide and topiramate also reduces cerebrospinal fluid pressure more than either medication in isolation, according to a 2024 experimental study on rats.⁵⁷ This may be of importance in patients with fulminant IIH in whom acetazolamide monotherapy is ineffective, but this effect has not been reproduced in humans. The same paper also suggested that acetazolamide and topiramate both effectively reduce intracranial pressure through distinct molecular mechanisms. Topiramate is teratogenic and is therefore contraindicated in pregnancy. Further studies are indicated to elucidate its role and efficacy in the management of IIH in comparison to and in conjunction with acetazolamide.

Furosemide

Furosemide is a loop diuretic often employed in the treatment of fluid retention caused by conditions such as congestive heart failure. Specifically, its primary mechanism entails the inhibition of the Na-K-Cl co-transporter within the thick ascending limb of the loop of Henle, leading to increased excretion of sodium, chloride, and water. Thus, furosemide acts systemically to reduce overall fluid volume, which can indirectly contribute to a reduction in ICP. Animal models in dogs have shown a reduction in intracranial pressure when administered furosemide.⁵⁸ Literature to support its use in humans is sparse but includes a pediatric case series in which patients administered both furosemide and acetazolamide demonstrated clinical improvement, implying a possible synergistic effect.^{59,60} This synergy is achieved by the combination of direct reduction in CSF production by acetazolamide and alterations in systemic fluid balance by furosemide. Furosemide therefore may provide some role as an adjunct to acetazolamide, but monotherapy is typically insufficient.

Corticosteroids

Corticosteroids are no longer routinely prescribed for IIH due to their side effect profile and the potential for rebound intracranial hypertension upon their discontinuation. However, their use may be considered in cases of fulminant and refractory IIH as a temporizing measure prior to definitive, surgical intervention.^{61,62}

Other Medications

Methazolamide is another carbonic anhydrase inhibitor that may be considered if patients are unable to tolerate acetazolamide.⁶³ Octreotide is a somatostatin analog that reduces the severity of headaches, extent of papilledema, and intracranial pressure.⁶⁴ Spironolactone reduces intracranial pressure, the extent of which is no more than that of acetazolamide, topiramate, and furosemide.⁶⁵

Cerebrospinal Fluid Diversion

Ventriculoperitoneal (VP) shunting can be an effective measure in providing relief for patients with intractable headaches and for those failing medical treatment. Lumboperitoneal (LP) shunting can also be considered, but this procedure may have a higher incidence of shunt obstruction and revision.⁶⁶ A 2014 study found improved visual outcomes in patients undergoing either VP or LP shunting, as measured by decimal visual acuity and mean radial degrees of the I4e isopter of the Goldmann visual field.⁶⁷ Once shunting provides adequate ICP control, the need for continued acetazolamide therapy is reassessed, with many patients no longer requiring it due to the direct CSF diversion provided by the shunt. However, the placement of shunts is not without risks, which include obstruction, infection, bowel perforation, and intracranial hemorrhage.⁶⁸ It is therefore most often recommended in cases that are either refractory to medical management or fulminant with signs of impending and permanent vision loss.

Optic Nerve Sheath Fenestration

Optic nerve sheath fenestration (ONSF) is a surgical procedure that entails the formation of a fenestration or window through the optic nerve sheath, thereby relieving pressure on optic nerve as it exits the globe in patients with intracranial hypertension. A 1988 study revealed symptomatic improvement of headaches in 11 out of 17 patients (65%) and of transient visual obscurations in 14 out of 16 patients (88%). A 2007 meta-analysis suggested that majority of patients with IIH who undergo ONSF have improvement of visual function compared with those receiving VP and LP shunts.⁶⁹ Lastly, a 2017 meta-analysis by Kalyvas et al that compared different surgical interventions concluded that ONSF is

effective in improving vision as compared with CSF shunting, which instead had a better headache reduction profile.⁷⁰ The role of acetazolamide in conjunction with ONSF has yet to be formally studied, although its use may be considered in the event of ONSF failure.

Transverse Sinus Stenting

Transverse venous sinus stenosis is a common radiographical finding in IIH. A recent meta-analysis suggests that transverse sinus stenting is associated with resolution of subjective symptoms in most patients with pulsatile tinnitus, double vision, and nonspecific visual complaints. It was also associated with a reduction in perimetric mean deviation on automated visual fields, mean retinal nerve fiber layer thickness, and intracranial pressure.⁷¹ Indeed, transverse sinus stenting has shown favorable complication profiles in both vision and headache improvement in the aforementioned 2017 meta-analysis by Kalyvas et al. However, re-stenosis occurs and there are no large long-term studies to give adequate outcomes, especially visual outcomes. Current evidence primarily focuses on stenting as a standalone intervention for select patients. Although transverse sinus stenting directly addresses venous outflow obstruction, the role of adjunctive acetazolamide therapy post-stenting is not well-defined in large long-term studies as current evidence primarily focuses on stenting as a standalone intervention for select patients. Nonetheless, clinicians may consider its use in cases of persistent symptoms or inadequate ICP reduction post-stenting.

Bariatric Surgery

Bariatric surgery has been shown to reduce both lumbar opening pressure and dosage of either acetazolamide or topiramate.⁷² Its role in the management of IIH is indirect in that its promotion of weight loss reduces ICP. As such, bariatric surgery can be considered a definitive, long-term adjunctive therapy that can allow for dose reduction or even discontinuation of acetazolamide. The extent of weight loss has been correlated with a reduction in intracranial pressure in those who have undergone bariatric surgery.⁷³ A 2023 systematic review compared intracranial pressure reduction in patients undergoing bariatric surgery at 24 months with lifestyle intervention with acetazolamide at 6 months and found relatively similar reductions in both groups of patients.⁷⁴ However, it should be noted that a significant portion of patients who have undergone bariatric surgery have experienced a recurrence of obesity.⁷⁵ It stands to reason that patients who regain weight after surgery may develop recurrence of IIH. Thus, careful monitoring for recurrence of obesity and IIH is crucial.

GLP-1 Agonists

Glucagon-like peptide-1 (GLP-1) agonists regulate glucose control by promoting the release of insulin and inhibiting the release of glucagon. GLP-1 receptor agonists are present in the hypothalamus, where their activation controls satiety and promotes weight loss.⁷⁶ GLP-1 receptor agonists are also present in the choroid plexus, where their activation inhibits sodium transport and decreases CSF production. A randomized control trial in 2023 showed a reduction in telemetric intracranial pressures at 12 hours, 24 hours, and 12 weeks following exenatide injections compared to placebo.⁷⁷ A 2023 open-label study showed a reduction in acetazolamide dosing in patients with IIH who received either semaglutide or liraglutide.⁷⁸ The same study also showed a median reduction in headaches and significant weight loss. GLP-1 agonists have also been shown to have direct effects of lowering ICP in addition to its effect in promoting weight loss.^{79,80} In addition, lifelong use of GLP-1 agonists is required for many patients, and its long-term drawbacks are still currently under investigation. In addition, GLP-1 agonists may be used in conjunction with acetazolamide due to their distinct, yet complementary mechanisms. This dual use addresses both direct ICP reduction and the underlying obesity often associated with IIH, potentially allowing for lower doses of acetazolamide and improving long-term outcomes through sustained weight management. Further studies are indicated to investigate the efficacy of GLP-1 agonists with IIH and the need for ICP-lowering medications.

Conclusion

Acetazolamide remains the first-line pharmacologic treatment for IIH due to its ability to reduce ICP. Its function as a carbonic anhydrase inhibitor accounts for its varied effects in reducing intracranial pressure, and it has been shown to improve several clinical parameters including visual function, lumbar opening pressure, and papilledema as measured via Frisén grading or OCT. The use of acetazolamide, starting at 500 mg twice daily in conjunction with weight loss, is recommended as first-line therapy for most patients, with increasing dose as needed and tolerated up to 4 grams a day. Its

use in conjunction with other medical or surgical therapies may be appropriate depending on the clinical context. Clinicians should carefully monitor for side effects and consider acetazolamide in conjunction with lifestyle changes and weight management to optimize treatment outcomes.

Funding

This material is based upon work supported (or supported in part) by the Department of Veterans Affairs, Veterans Health Administration, Office of Research and Development, and the Rehabilitation Research and Development Service including Department of Veterans Affairs (RR&D) grant C9251-C (RX003002) Iowa City Center for The Prevention and Treatment of Visual Loss.

Disclosure

None of the authors have a conflict of interest with the content of this review.

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