# Idiopathic Intracranial Hypertension Masquerading as Cerebral Venous Sinus Thrombosis: A Diagnostic Challenge

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Idiopathic intracranial hypertension (IIH) is a syndrome identified by raised intracranial pressure (ICP), with absence of a mass lesion/hydrocephalus, typically affecting obese women of childbearing age.[1] It may present with similar symptoms such as headache, visual disturbances, and papilledema, making clinical differentiation challenging.[1] IIH primarily threatens vision, and treatment focuses on lowering ICP using medications like acetazolamide, serial lumbar punctures, or surgical interventions such as optic nerve sheath fenestration or CSF shunting in refractory cases.[2] Misdiagnosing IIH as CVST can lead to unnecessary anticoagulation, which may prove to be harmful for the patient.

A woman in her early 30s, from Noida, presented with a 15-day history of diplopia, floaters, and holocranial headache. Ophthalmologic evaluation revealed esotropia in the right eye and bilateral optic disk edema with hemorrhages (Fig. 1). The MRI brain was normal, while the MRI orbit showed cupping of the bilateral optic nerves—findings suggestive of bilateral papilledema. A lumbar puncture was done, which showed raised opening pressure. CSF biochemistry and cell counts were within normal limits. The patient also reported improvement in visual symptoms following the procedure. Given suspected cerebral venous sinus thrombosis (CVST, the patient was started on low-molecular-weight heparin (dalteparin) and high-dose acetazolamide to reduce cerebrospinal fluid pressure. After 2 weeks, the patient presented to our hospital with persistent symptoms. Re-evaluation of laboratory tests, including a detailed history focusing on potential thrombosis risk factors, was done. Physical and systemic examination were normal. A comprehensive thrombophilia panel was also sent, which was normal (Table 1). Fundus examination was done, which showed bilateral papilledema (Fig. 1). Repeat contrast-enhanced MR venography showed no evidence of CVST but revealed bilateral arachnoid granulations and hypoplastic left transverse sinus (Fig. 2). Repeat lumbar puncture demonstrated an opening pressure of 350 mm $H_2O$ with normal CSF biochemistry. Based on the above findings, she was diagnosed with idiopathic intracranial hypertension (IIH). Dalteparin was discontinued, and the patient was continued on high-dose acetazolamide with significant symptomatic improvement on follow-up. Follow-up visits included serial fundus examinations, which showed gradual resolution of papilledema in both eyes (Fig. 3), confirming clinical and therapeutic response.

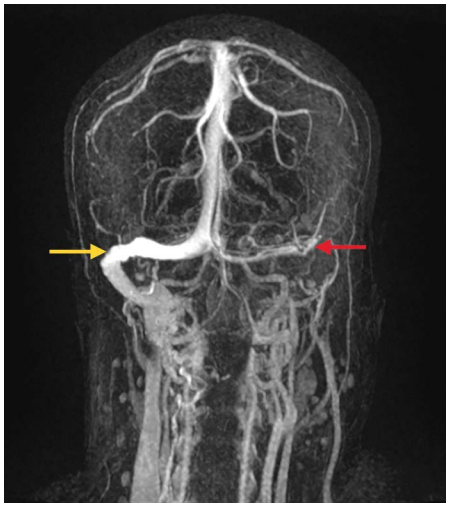

**Fig. 2:** Contrast-enhanced magnetic resonance venography showing a hypoplastic left transverse sinus (red arrow) and patent right transverse sinus (yellow arrow), with no evidence of thrombus

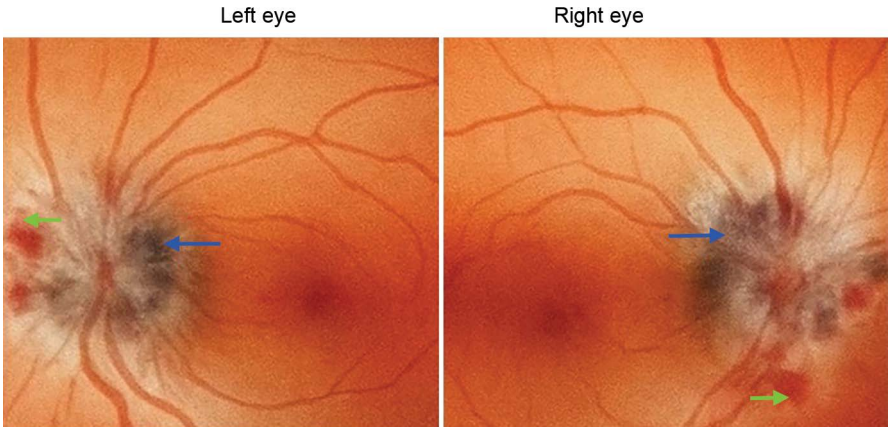


**Fig. 1:** Fundus photographs of both eyes showing disk edema (blue arrow) and peripapillary hemorrhage (yellow arrow)

## Discussion

Idiopathic intracranial hypertension (IIH), formerly known as pseudotumor cerebri, is characterized by elevated intracranial pressure (ICP) in the absence of an intracranial mass, hydrocephalus, infection, or hypertensive encephalopathy.[3] In adults, standard ICP is generally 15 mm Hg, and values >25 cm $H_2O$ are considered elevated. IIH predominantly affects young, overweight women of childbearing age, with an annual incidence of 1–2 per 100,000 population, rising to 4–21 per 100,000 in obese females aged 15–44 years. Obesity is the most significant modifiable risk factor. Other associated factors include systemic conditions (e.g., Addison's disease, hypoparathyroidism), medications

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**Table 1:** Laboratory parameters

| *Parameter* | *Patient value* | *Reference range* |
|---|---|---|
| Hemoglobin | 10.0 gm/dL | 13.5–17.5 gm/dL (male), 12.0–15.5 gm/dL (female) |
| Hematocrit (%) | 31.6 | 41–53 (male), 36–46 (female) |
| Total leukocyte count | 9250/µL | 4,000–11,000/µL |
| Platelet count | 359000/µL | 150,000–450,000/µL |
| Differential leukocyte count (%) | 75/20/4/1 | Neutrophils 40–75, lymphocytes 20–45, monocytes 2–10, eosinophils 1–6 |
| Total bilirubin | 0.45 mg/dL | 0.1–1.2 mg/dL |
| Direct bilirubin | 0.35 mg/dL | 0.0–0.3 mg/dL |
| Total protein | 7.5 g/dL | 6.0–8.3 g/dL |
| Serum albumin | 4.2 g/dL | 3.4–5.4 g/dL |
| AST | 29 U/L | < 40 U/L |
| ALT | 29 U/L | < 40 U/L |
| GGT | 16 U/L | 9–48 U/L |
| ALP | 135 U/L | 44–147 U/L |
| TSH | 3.01 mIU/L | 0.4–4.0 mIU/L |
| Free T3 | 3.44 pg/mL | 2.0–4.4 pg/mL |
| Free T4 | 1.51 ng/dL | 0.9–2.3 ng/dL |
| BUN | 23 mg/dL | 7–20 mg/dL |
| Creatinine | 0.7 mg/dL | 0.6–1.3 mg/dL |
| $Na^+$ | 139 mEq/L | 135–145 mEq/L |
| $K^+$ | 4.23 mEq/L | 3.5–5.0 mEq/L |
| $Ca^{2+}$ | 9.2 mg/dL | 8.5–10.5 mg/dL |
| Phosphorus | 3.2 mg/dL | 2.5–4.5 mg/dL |
| CSF opening pressure | 350 mm $H_2O$ | 100–250 mm $H_2O$ |
| Cell count | 0 cells | 0–5 cells/µL |
| Protein | 22.2 mg/dL | 15–45 mg/dL |
| Glucose | 73.9 mg/dL | 45–80 mg/dL |
| ADA | 2.0 U/L | < 10 U/L (for TB screening) |
| TB PCR (Xpert MTB) | Not detected | Negative |
| CSF Gram stain | No organism seen | No organism seen |
| CSF AFB stain | Negative | Negative |
| ANA, ANCA | Negative | Negative |
| Protein C/protein S | Normal | Normal |
| Antithrombin III | 105% | 80–120% |
| Lupus anticoagulant/dRVVT | Negative | Negative |
| β2-GPI, ACA IgG/IgM | Negative | Negative |
| Homocysteine | 7.23 µmol/L | 4–15 µmol/L |
| Factor V Leiden mutation | Negative | Negative |

AST, aspartate aminotransferase; ALT, alanine aminotransferase; GGT, gamma-glutamyl transferase; ALP, alkaline phosphatase; TSH, thyroid-stimulating hormone; BUN, blood urea nitrogen; $Na^+$, sodium; $K^+$, potassium; $Ca^{2+}$, calcium; CSF, cerebrospinal fluid; ADA, adenosine deaminase; TB, tuberculosis; PCR, polymerase chain reaction; MTB, Mycobacterium tuberculosis; AFB, acid-fast bacilli; ANA, antinuclear antibody; ANCA, antineutrophil cytoplasmic antibody; dRVVT, dilute Russell viper venom time; β2-GPI, beta-2 glycoprotein I; ACA, anticardiolipin antibody; IgG, immunoglobulin G; IgM, immunoglobulin M

(e.g., tetracyclines, hypervitaminosis A), and more recently, abrupt cessation of GLP-1 receptor agonists such as semaglutide, which may result in rapid weight regain—a known risk factor for IIH development.[4] In this patient, clinical presentation with headache, visual floaters, diplopia, and bilateral papilledema with hemorrhages suggested raised ICP.[5] The initial noncontrast MRV, which falsely indicated cerebral venous sinus thrombosis (CVST), illustrates a frequent diagnostic pitfall. Flow-related artifacts and normal anatomical variants, such as hypoplastic sinuses or prominent arachnoid granulations, can mimic thrombosis on imaging. This underscores the need for contrast-enhanced MRV, which remains the gold standard in differentiating true thrombus from anatomical variants.[6] The diagnostic confirmation of IIH in this case followed the modified Dandy criteria, which include: Symptoms of increased IC pressure (e.g., headache, visual obscurations, papilledema), normal CNS examination (excluding VI nerve palsy), normal neuroimaging (excluding mass or hydrocephalus), raised opening pressure on lumbar puncture (>25 cm $H_2O$) with normal CSF parameters as well as alert and oriented mental status.[7] This patient's CSF opening pressure was significantly elevated (350 mm $H_2O$), with normal cell count and biochemistry, aligning well with diagnostic criteria. The mainstay of medical treatment is acetazolamide, a carbonic anhydrase inhibitor that causes a reduction in production of CSF by its action at the choroid plexus.[7] As used in our case, it showed significant efficacy in improving both symptoms and fundoscopic findings. The patient showed marked improvement on serial fundus evaluations, confirming resolution

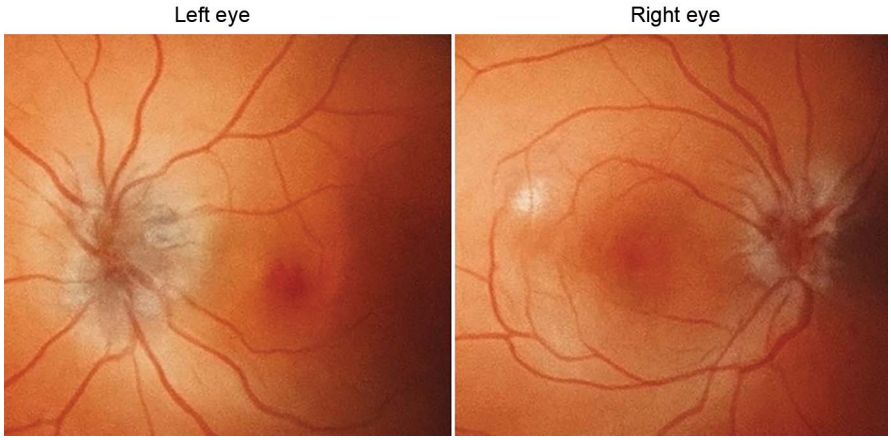


**Fig. 3:** Fundus photographs after 2 weeks of follow-up on acetazolamide therapy showing resolving disk edema

of papilledema. In refractory cases, options include topiramate and/or furosemide. Invasive interventions, such as CSF shunting or optic nerve sheath fenestration, may also prove to be helpful, especially when vision is threatened. Long-term care involves maintaining weight reduction and periodic vision monitoring to prevent relapse.

Idiopathic intracranial hypertension can mimic cerebral venous sinus thrombosis on imaging. High clinical suspicion, detailed thrombophilia workup, CSF pressure analysis, and imaging with contrast MRV are crucial for accurate diagnosis. Ophthalmologic findings and symptom improvement after LP strongly support a diagnosis of IIH. Avoiding misdiagnosis prevents unnecessary anticoagulation and directs appropriate treatment.

## Patient Consent Statement

Participant's consent has been obtained.

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## References

1. Saposnik G, Barinagarrementeria F, Brown RD Jr, et al. Diagnosis and management of cerebral venous thrombosis: a statement for healthcare professionals from the American Heart Association/American Stroke Association. Stroke 2011;42(4):1158–1192.
2. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 2013;81:1159–1165.
3. Mollan S, Davies B, Silver NC, et al. Idiopathic intracranial hypertension: consensus guidelines on management. J Neurol Neurosurg Psychiatry 2018;89:1088–1100.
4. Heckel B. Idiopathic intracranial hypertension after abrupt cessation of medication: a case report of abrupt glucagon-like peptide-1 (GLP-1) receptor agonist cessation and review of the literature. Curr Pain Headache Rep 2024;28:453–456.
5. Wall M. Idiopathic intracranial hypertension. Neurol Clin 2010;28:593–617.
6. Leach JL, Fortuna RB, Jones BV, et al. Imaging of cerebral venous thrombosis: Current techniques, spectrum of findings, and diagnostic pitfalls. Radiographics 2006;26 Suppl 1:S19–S41.
7. Ball AK, Clarke CE. Idiopathic intracranial hypertension. Lancet Neurol. 2006;5:433–442.