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Chronic migraine, neuroimaging findings suggestive of idiopathic intracranial hypertension without papilledema, and response to fremanezumab: a case report

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Introduction

Neuroimaging findings suggestive of idiopathic intracranial hypertension (IIH) may be incidentally identified in patients with migraine, particularly in those with chronic and treatment-refractory forms. Interpretation of these findings is challenging in the absence of papilledema, when the causal relationship between elevated intracranial pressure and headache is not always evident.

Case presentation

A 41-year-old woman with normal body weight had a history of migraine with typical aura since the age of 13. In February 2025, she experienced increased headache frequency and intensity, without new neurological or visual symptoms. Brain magnetic resonance imaging (MRI) showed a partially empty sella and bilateral transverse sinus stenosis. Cerebrospinal fluid opening pressure was 42 cmH_2O, and a repeated measurement after a few weeks was 30 cmH_2O. Serial ophthalmological evaluations, including fundoscopy and optical coherence tomography, showed no evidence of papilledema. She had a history of recurrent nephrolithiasis associated with topiramate and was unable to tolerate acetazolamide. In September 2025, she underwent lumboperitoneal shunt placement. After the procedure, she developed orthostatic headache, improving in the supine position. A new MRI showed signs of intracranial hypotension, including pachymeningeal thickening, engorgement of the dural venous sinuses, reduced ventricular size, and decreased mamillopontine distance, as well as a subdural collection adjacent to the tentorial membrane, extending to the left side at the C3 level. The valve was closed and subsequently removed, followed by an epidural blood patch, with improvement of the orthostatic headache. However, migraine-like headache recurred on most days. In November 2025, follow-up MRI again showed findings suggestive of IIH, now including optic nerve tortuosity, while ophthalmological assessment remained normal. Given the persistent absence of papilledema and the predominance of a migrainous phenotype, fremanezumab was initiated, with a marked clinical response, reducing headache frequency to approximately 2 days per month.

Discussion

This case illustrates the complexity of evaluating patients with chronic migraine and findings compatible with IIH, particularly when elevated opening pressure is present without papilledema or visual impairment. Distinguishing migraine with incidental findings from IIH without papilledema, or an overlap between both conditions, has important therapeutic implications. The robust response to CGRP blockade supports the predominance of a migrainous mechanism, although it does not exclude a possible contribution of pathways related to CSF dynamics and CGRP signaling in meningeal lymphatic vessels.

Conclusion

In chronic headache with imaging findings suggestive of IIH but no papilledema, management should prioritize headache phenotype, CSF pressure, and visual monitoring. Phenotype-guided migraine treatment may be appropriate in selected cases.

Keywords:
Chronic migraine
Idiopathic intracranial hypertension
Papilledema
CGRP
Fremanezumab

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