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Unexpected Therapeutic Response to Lumbar Puncture Leading to the Diagnosis of Idiopathic Intracranial Hypertension in a Patient Previously Diagnosed with TMJ dysfunction: A Case Report

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Abstract

Background: Idiopathic intracranial hypertension (IIH) typically presents with headache, papilledema, pulsatile tinnitus, transient visual obscurations, and elevated cerebrospinal fluid (CSF) opening pressure. Diagnosis may be delayed when patients have a pre-existing primary headache disorder such as migraine, particularly when neuroimaging is unrevealing and papilledema is subtle or absent.

Case Presentation: A 40-year-old woman with a history of migraine with aura and temporomandibular joint (TMJ) dysfunction presented with a new severe occipital headache accompanied by pulsatile tinnitus, visual disturbances, diplopia, and worsening positional head pressure. Initial evaluation for aneurysmal subarachnoid hemorrhage (SAH) included CT angiography followed by lumbar puncture, with an opening pressure of 23 cm H2O. Although the diagnostic evaluation was negative for SAH, the patient experienced immediate and marked improvement in headache, and jaw pain following CSF removal. A second, therapeutic lumbar puncture several weeks later again resulted in substantial symptomatic improvement lasting approximately two weeks. Progressive recurrence of symptoms, particularly worsening when supine and during Valsalva maneuvers, ultimately led to a diagnosis of idiopathic intracranial hypertension. Treatment with acetazolamide resulted in further clinical improvement.

Conclusion: This case illustrates how IIH may masquerade as Jaw pain, TMJ-related pain, or worsening migraine and demonstrates that dramatic symptomatic improvement following

diagnostic lumbar puncture may provide an important clinical clue to elevated intracranial pressure. Clinicians should maintain a high index of suspicion for IIH in patients with new positional headaches, pulsatile tinnitus, transient diplopia, or visual symptoms despite normal structural neuroimaging.

Introduction

Idiopathic intracranial hypertension is characterized by elevated intracranial pressure without an identifiable intracranial mass, hydrocephalus, or secondary cause. The disorder most commonly affects women of childbearing age and typically presents with headache, pulsatile tinnitus, transient visual obscurations, diplopia due to sixth cranial nerve dysfunction, and papilledema.[1]

Headaches associated with IIH often resembles migraine and can manifest as facial pain, creating a diagnostic challenge in patients with history of migraine, face pain, and TMJ dysfunction.[2] Furthermore, some patients exhibit minimal or absent papilledema, further delaying diagnosis. Recognition of distinguishing clinical features is essential because untreated intracranial hypertension may lead to irreversible visual loss.[3]

We describe a patient who was previously diagnosed with TMJ dysfunction, whose diagnosis became apparent after two diagnostic lumbar punctures unexpectedly produced significant therapeutic benefit.

Case Presentation

A 40-year-old woman, with a BMI 27.3 and a history of migraine with sensory aura and TMJ dysfunction presented with a new headache syndrome that differed substantially from her baseline migraine.

Her prior migraines occurred approximately six days per month before topiramate therapy and improved to approximately two days monthly on topiramate. Typical attacks consisted of unilateral right-sided throbbing pain preceded by facial numbness and burning sensations, associated with photophobia, phonophobia, osmophobia, nausea, vestibular symptoms, and cognitive slowing. Rizatriptan consistently aborted these attacks.

Approximately one year before presentation, she started to experience increasing ear pressure, jaw pain and headache associated with facial numbness that prompted stroke evaluation, which was unrevealing. Eventually she was diagnosed with TMJ dysfunction

Few months later, she developed an abrupt new headache described as "being punched in the back of the head," accompanied by visual disturbance and several weeks of worsening right-sided pulsatile tinnitus, and jaw pain. CT angiography raised concern for a possible intracranial aneurysm, prompting diagnostic lumbar puncture. CSF analysis excluded subarachnoid hemorrhage and revealed an opening pressure of 23 cm H2O in the lateral decubitus.

Unexpectedly, immediately after lumbar puncture she experienced dramatic improvement in headache and right jaw pain that had previously been attributed to temporomandibular dysfunction.

Several weeks later she developed recurrent symptoms, including intermittent diplopia. During an emergency evaluation, a repeat lumbar puncture was performed with an opening pressure of 12 cm H2O, and she again

experienced marked symptomatic improvement lasting approximately two weeks.

Symptoms gradually returned and evolved into a classic intracranial pressure syndrome characterized by: morning headache, daily pressure-type headaches, marked worsening while lying flat, improvement with elevation of the head of the bed, exacerbation during bending forward and physical exertion, persistent right-sided pulsatile tinnitus, intermittent visual disturbances and episodic diplopia

Neurological examination was largely normal. Cranial nerves were intact without focal deficits. Ophthalmoscopic examination demonstrated only mildly blurred optic disc margins without frank papilledema.

Brain MRI, MRA, CTA, and multiple CT examinations demonstrated no intracranial hemorrhage, aneurysm, infarction, mass lesion, hydrocephalus, or vascular abnormality.

Because of the characteristic positional headache, pulsatile tinnitus, transient diplopia, repeated symptomatic improvement after CSF drainage, and exclusion of secondary intracranial pathology, idiopathic intracranial hypertension became the leading diagnosis.

Neuro-ophthalmologic evaluation was unremarkable. Acetazolamide was initiated and titrated, while the topiramate dosage was reduced, resulting in improvement of her symptoms.

Discussion

IIH has been associated with orofacial pain presentations, including persistent idiopathic facial pain and, less commonly, trigeminal neuralgia. However, to our knowledge, a comparable association with TMJ pain has not been reported in the literature.[4, 5]

This case highlights several important clinical lessons. First, IIH can closely mimic worsening migraine and present as jaw pain in its early phase. Although the patient had a longstanding history of migraine with aura, the evolution of a distinctly different headache phenotype—including occipital pressure, positional worsening, pulsatile tinnitus, and visual symptoms—suggested an alternative diagnosis.

Second, the dramatic response following lumbar puncture served as a valuable diagnostic clue. While lumbar puncture is performed primarily for diagnosis, transient symptom relief after CSF drainage offers an important clinical clue to intracranial hypertension when considered together with the other clinical symptoms. In this patient, therapeutic improvement after two independent lumbar punctures preceded formal recognition of IIH.

Third, papilledema was not a dominant clinical finding. Increasing recognition of IIH without papilledema (IIHWOP) emphasizes that absence of optic disc edema does not exclude elevated intracranial pressure, particularly in patients with compatible symptoms and elevated opening pressure. Such patients frequently undergo prolonged evaluation for migraines or headache and face pain disorders before the correct diagnosis is established.

Finally, pulsatile tinnitus proved to be an important localizing symptom. The rhythmic "whooshing" sound synchronized with the heartbeat is increasingly recognized as one of the most characteristic symptoms of IIH and should prompt consideration of elevated intracranial pressure after vascular abnormalities have been excluded.

The patient's transient but reproducible improvement following CSF drainage further reinforces the role of intracranial pressure in symptom generation with referred pain to the

jaw. Jaw pain is thought to be from CSF pressure–mediated mechanism rather than true temporomandibular joint dysfunction.

Conclusion

This case illustrates the diagnostic challenge of distinguishing IIH from chronic migraine in patients with pre-existing headache and TMJ disorders. The emergence of positional headache, pulsatile tinnitus, visual symptoms, and reproducible improvement after diagnostic lumbar puncture should prompt evaluation for intracranial hypertension even when neuroimaging is normal and papilledema is subtle or absent. Early recognition is essential to prevent permanent visual complications and initiate appropriate therapy.

Declarations/Disclosures

Consent: Consent to publish this case report has been obtained from the patient in writing.

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