

Idiopathic Intracranial Hypertension in Older Population: Do We Need to Modify Our Approach?

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<p>Abstract: Older adults have a higher incidence of secondary headaches. The aim of this paper is to highlight that idiopathic intracranial hypertension (IIH), one of the causes of secondary headache, is not so uncommon in older adults, and if missed, may lead to considerable morbidity and, in unfortunate cases, drastic consequences.</p>	<p style="text-align: center;">Letter to Editor</p> <p style="text-align: center;">*Corresponding Author: <i>Debabrata Chakraborty</i> Department of Neurology, Apollo Multispeciality Hospitals, Kolkata, West Bengal, India</p>
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TEXT

IIH (idiopathic intracranial hypertension) is a neurological disorder which is still evolving, and the diagnostic criteria may require revision in the near future. We need to keep in mind the wide variation of its clinical manifestations and a significant number of patients not obeying the classical clinical criteria. Hence revisiting this topic is important and objective of this paper is to prevent misdiagnosis in this group of population.

Older adults are more likely to have a secondary headache than younger adults, with rates in clinical settings around 15% of all headache cases, compared to 1% to 6% in younger patients.

Idiopathic intracranial hypertension (IIH) is not so uncommon in older adults, but rather less detected and treated. In a study, out of 165 patients with IIH, 135 patients were in the younger group and 32 were in the older group [1].

IIH is still evolving, and the diagnostic criteria may require revision in the near future. We need to keep in mind the wide variation of its clinical manifestations and a significant number of patients not obeying the classical clinical criteria. This often leads to a misdiagnosis or late diagnosis in different departments:

Neurology (18.2%), Ophthalmology (16.1%), Internal Medicine (20%), and Neurosurgery (7.7%) and 33.3% in the Paediatrics department [2].

Given that up to 24% of IIH patients experience irreversible vision loss and 4% experience bilateral blindness, a delayed diagnosis can have disastrous results. For this reason, it is crucial to identify and treat IIH as soon as possible.

With time we are getting acquainted with the atypical presentations [3] of the disease and we need to be more suspicious about the entity. Some of the atypical presentations are:

1. Ocular motor disturbances from third or fourth cranial nerve impairment and skewed deviation of eyes.
2. Unilateral papilledema and without papilledema.
3. Disorder of olfaction (up to 80%).
4. Trigeminal nerve dysfunction (in the context of elevated intracranial pressure (ICP), this dysfunction/neuralgia may be caused by pathophysiologic mechanisms such as direct compression of the trigeminal root at the petrous apex by the petrosal sinus).
5. Facial nerve dysfunction. (The coincidence of IIH and Bell's palsy is possible).

6. Hearing loss and vestibular dysfunction—pulsatile tinnitus is the most common otological symptom, reported by 52%–61%, while hearing impairment is reported in 30%–85% of patients with IIH.
7. Lower cranial nerve dysfunction, including deviated uvula, torticollis, and tongue weakness.
8. Cognitive impairment.
9. Temporal lobe encephalocele in case of drug-resistant epilepsy.

Regional variation in cerebrospinal fluid (CSF) pressure is another important point that needs to be considered in clinical decision-making. Compared to Caucasian patients, Asian IIH patients were shown to have lesser obesity and papilledema. While the phenotype of individuals with CSF pressures between 200 and 250 mm of H₂O was less severe, the risks of headache or visual loss were similar to those in the group with CSF pressures greater than 250 mm of H₂O. Although further research is required, it's probable that Asians may benefit more from a diagnostic limit of > 200 mm of H₂O [4].

IIHWOP (Idiopathic Intracranial Hypertension without Papilledema) patients are a subgroup of patients who have no papilledema or optic atrophy, neuroimaging results indicate elevated ICP, and lumbar puncture reveals evidence of elevated CSF opening pressure. This is possibly either due to anatomic variation in the optic nerve sheath or its trabeculations that limits transmissions of raised intracranial pressure to the optic nerve or because the fluctuating elevation in intracranial pressure is below the threshold required to develop papilledema.

Idiopathic intracranial hypertension (IIH) is strongly associated with hormonal and metabolic dysregulation which has implication in diagnosis and management. Some evidences are as follows:

1. The incidence of polycystic ovarian syndrome in patients of IIH ranged from 15.5% to 57% [5].
2. Women with IIH exhibit a distinct "androgen excess signature" that is marked by noticeably higher amounts of testosterone and androstenedione in their blood and CSF. By activating ion transporters in the choroid plexus, elevated testosterone levels may boost the generation of CSF.
3. Leptin, a hormone released by fat cells, is frequently found in high concentrations in the serum and CSF of IIH patients, and its persistent elevation in IIH points to "leptin resistance," which could lead to weight gain and

possibly raise CSF secretion by interfering with salt pumps.

4. The enzyme 11 β -HSD1, which converts inactive cortisone into active cortisol, is often more active in IIH patients. This increased local cortisol in the brain may drive excess CSF production. Increased estrogen levels may also increase the permeability of water channels in the brain.
5. Growth hormone therapy, especially in children, is a recognized trigger for increased intracranial pressure.
6. Conditions like Addison's disease and the use or withdrawal of systemic corticosteroids have been implicated in secondary intracranial hypertension.
7. Both hypothyroidism and thyroid replacement therapy (particularly in children) are potential risk factors.

Pituitary hormone dysfunction can be brought on by IIH, which can lead to subclinical hormonal problems like elevated prolactin or disturbed gonadotropin levels, though these typically improve when the ICP is lowered; if diagnosis is delayed, it may have influence in both pregnancy and on fertility.

All patients suspected of having IIH, particularly those exhibiting unusual signs or symptoms, require CSF investigation, contrast-enhanced brain MRI with MRV to rule out a meningeal process, and exclusion of cerebral venous sinus thrombosis and dural arteriovenous fistula.

In case of a diagnostic dilemma, we may need to take help of some modified imaging techniques. A sensitive method for assessing the integrity of the blood-brain barrier is the dynamic contrast-enhanced perfusion measure K_{trans} [5]. This method has been widely applied to a number of disorders linked to aberrant glymphatic pathways, and it may be crucial to comprehending the function of the IIH. Diffusion tensor image analysis along the perivascular space is a new technique to detect the interstitial fluid and CSF imbalance along the perivascular space.[6] The use of these sophisticated MR imaging methods is probably going to be quite important in doubtful cases but more importantly when it comes to measuring therapy response in IIH patients.

Interestingly, IIH is not that uncommon. It has been estimated that between 5% and 14% of migraineurs have IIHWOP. According to the above-mentioned current diagnostic criteria for IIHWOP, 2.5% of patients with persistent headaches who were not responding to treatment had IIHWOP [3]. In elderly population when cognitive impairment may be on board along with

multiple comorbidities including other reason for headache the presentation of IIH may be more challenging. Thus, in doubtful cases, keeping in mind the evolving nature of the disease, we may give a trial of acetazolamide and observe for improvement in the clinical signs and symptoms. More clinical trials in this regard are welcome.

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