Medical Termination of Pregnancy Precipitating Idiopathic Intracranial Hypertension

Dear Editor,

Raised intracranial pressure (ICP) without any identifiable secondary cause characterizes idiopathic intracranial hypertension (IIH), which has been associated with chronic anemia.[1] However, IIH precipitated by acute anemia presenting with isolated visual impairment has not been reported earlier. We hereby report a young thin-built woman, with acute anemia secondary to massive hemorrhage following an attempted first-trimester medical termination of pregnancy (MTP), after which she presented with bilateral diminution of vision and was diagnosed with IIH.

A 28-year-old thin-built lady presented with bleeding per vaginum for 7 days following an attempted first-trimester MTP with self-administration of mifepristone and misoprostol. Five days later, she had a mild holocranial headache, with recurrent episodes of vomiting and bilateral painless blurring of vision. Visual acuity was 6/24 in the right eye and 6/18 in the left eye. Fundus examination revealed grade 3 papilledema

bilaterally. Magnetic resonance imaging (MRI) of the brain with contrast and venogram were normal without any findings of raised ICP. The cerebrospinal fluid (CSF) opening pressure was 340 mm of H_2O with normal composition. She had a hemoglobin of 6.9 g/dL with iron deficiency. Thyroid, renal, and hepatic profiles were normal. Ultrasound of the abdomen showed retained products of conception, suggesting an incomplete abortion. Based on these findings, she was diagnosed with IIH, precipitated by acute hemorrhage-related anemia. She was transfused with two packed red blood cell transfusions to escalate the hemoglobin to 10 g/dL. She was also administered mannitol 0.5 g/kg eighth hourly and acetazolamide 1 g/day. Evacuation of the retained products of conception was performed, along with iron supplementation. Headache and vomiting subsided within 3 days; however, visual acuity did not improve. Thus, methylprednisolone 1 g/day for 3 days was initiated for vision-threatening IIH. A significant improvement was noted by the first week, and her visual acuity had become 6/6 bilaterally. On follow-up post-discharge, the patient was asymptomatic. The steroids were tapered over 4 weeks and stopped, and acetazolamide was gradually tapered to 500 mg per day.

IIH refers to the syndrome of raised intracranial tension with normal parenchyma, after reasonable exclusion of alternative causes. It usually presents with headache and features of raised ICP, with visual involvement secondary to long-standing papilledema. Rarely, it may present with acute severe diminution of vision, with minimal signs of raised ICP.[1] A definite diagnosis of IIH requires the presence of papilledema, a normal neurological examination with the exception of sixth cranial nerve involvement, no identifiable structural etiology, and cerebrospinal fluid before (CSF) pressure >250 mmH_2O with normal CSF composition.[2] MRI may show findings of raised ICP, such as empty sella, flattening of posterior globe, distended perioptic subarachnoid space, and transverse venous sinus stenosis. These findings are classically reflective of chronically raised ICP; however, it is not known how long these features take to develop in the setting of raised ICP. These findings may be absent initially, and their absence cannot rule out IIH.[2-4] This explains the lack of MRI findings of raised ICP, in cases of acute IIH, as in our patient.

The demographics of IIH patients classically involve obese women of childbearing age.[1] Headache is usually the most common symptom, followed by transient visual obscurations and pulsatile synchronous tinnitus. Subclinical visual defects are often identified on perimetry in >90% of patients although the loss of vision is reported in only 32% of cases.[1] Our patient primarily presented with bilateral painless diminution of vision without any forthcoming symptoms of raised ICP.

It has been hypothesized that iron deficiency anemia (IDA), being a hypercoagulable state, can increase venous pressure. The reduced oxygen-carrying capacity in IDA can lead to cerebral hypoxia, followed by cerebral edema. In acute anemia, the compensatory protective mechanisms protecting against acute hypoxia may be overpowered, resulting in cerebral edema.[5]

Reports on the association of acute anemia with IIH are sparse. Acute anemia secondary to parvovirus 19-related and autoimmune hemolytic anemia has been reported to cause IIH, but vision loss has not been described.[5] Only two cases of fulminant IIH following an abortion have been reported, both of whom had presented with predominant diminution of vision and minimal headache.[6,7]

In the majority of patients with IIH, insidious vision loss occurs secondary to chronic disc edema and progressive optic nerve damage. In <3% of cases, patients may present with acute deterioration of visual acuity, due to severe papilledema and optic neuropathy, and are referred to as having fulminant IIH.[8]

Management of IIH focuses on decreasing ICP and protecting vision. Medical management to control ICP includes acetazolamide and topiramate. A short course of high-dose systemic corticosteroids is used in rapidly progressive acute vision loss resulting from fulminant papilledema, especially as a temporary measure while awaiting surgery. Surgical interventions are required for progressive visual loss unresponsive to medical therapy. Additionally, any secondary causes of IIH should be identified and treated.[5]

Studies in mouse models with intracranial hemorrhages have demonstrated protective effects of these drugs against cerebral edema and cytotoxic damage.[9] Furthermore, the presence of a temporal effect, previous associations, no alternative explanation of the adverse effect, and improvement upon discontinuation are required to satisfy a definite causation relationship between the drug administration and IIH. While a temporal relationship cannot be denied, the presence of anemia, as a confounding factor, which has been undisputedly associated with IIH as detailed above, compounds the clinical scenario. Case reports of IIH following abortion[7,10] similarly have attributed the IIH to acute anemia, which seems plausible in light of the available evidence and unclear influence of these drugs on cerebral flow dynamics.

In conclusion, IIH can be precipitated by acute anemia and can present with isolated visual impairment. Furthermore, the absence of findings of raised ICP on neuroimaging cannot rule out IIH. Anemia correction and urgent lowering of ICP may circumvent surgery, leading to improved visual outcomes.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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References

1. Wall M. Idiopathic intracranial hypertension. Neurol Clin 2010;28:593-617.
2. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. Neurology 2013;81:1159-65.
3. Bidot S, Saindane AM, Peragallo JH, Bruce BB, Newman NJ, Biousse V. Brain imaging in idiopathic intracranial hypertension. J Neuroophthalmol 2015;35:400-11.
4. Maralani PJ, Hassanlou M, Torres C, Chakraborty S, Kingstone M, Patel V, *et al*. Accuracy of brain imaging in the diagnosis of idiopathic intracranial hypertension. Clin Radiol 2012;67:656-63.
5. Yu CW, Waisberg E, Kwok JM, Micieli JA. Anemia and idiopathic intracranial hypertension: A systematic review and meta-analysis. J Neuroophthalmol 2022;42:e78-86.
6. Deb AK, Ghag G, Gera P, Kasturi N, Sarkar S. Bilateral non-arteritic anterior ischemic optic neuropathy with idiopathic intracranial hypertension secondary to attempted abortion related hemorrhage: A rare case report. Indian J Ophthalmol Case Rep 2021;1:137-9.
7. Selvaraj J, Veeranki V, Kommaraju SY, Ravi P, Kaliaperumal S. Abortion and fulminant idiopathic intracranial hypertension. Cureus 2021;13:e13501. doi: 10.7759/cureus. 13501.
8. Thambisetty M, Lavin PJ, Newman NJ, Biousse V. Fulminant idiopathic intracranial hypertension. Neurology 2007;68:229-32.
9. Wu H, Wu T, Hua W, Dong X, Gao Y, Zhao X, *et al*. PGE2 receptor agonist misoprostol protects brain against intracerebral hemorrhage in mice. Neurobiol Aging 2015;36:1439-50.
10. Waisberg E, Yu C, Sverdlichenko I, Micieli J. New onset severe anemia and fulminant idiopathic intracranial hypertension. Can J Neurol Sci 2022;49:713-5.

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