Case Report: Post-Traumatic Intracranial Hypertension in the Setting of Traumatic Brain Injury

Justin Weppner DO, Michael Bova MD, Justin Tu MD, Emily Hillaker DO

University of Virginia, Department of Physical Medicine and Rehabilitation

CASE DIAGNOSIS

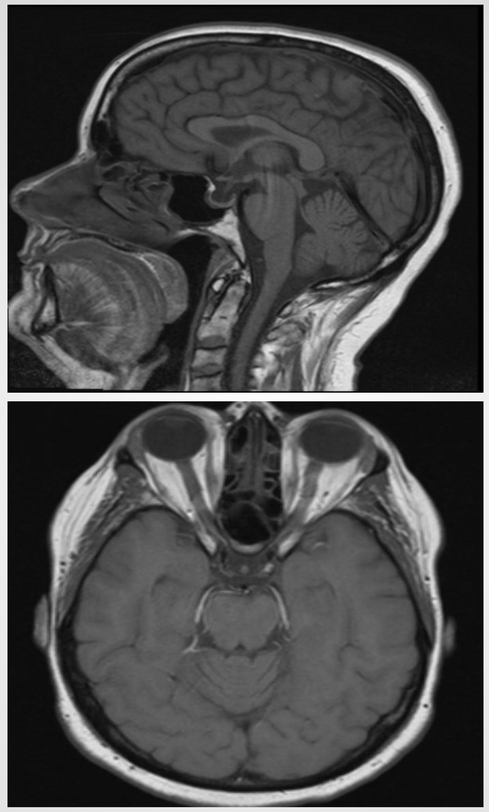
- 59-year-old female with moderate traumatic brain injury (TBI) and subarachnoid hemorrhage from a motor vehicle collision with delayed-onset idiopathic intracranial hypertension (IIH).

CASE DESCRIPTION

- Patient with no past medical history, BMI 23 kg/m2 presents two months following a moderate TBI with headaches, blurred vision, tinnitus, and impaired balance.
- Neurologic exam was unremarkable. She was prescribed vestibular rehabilitation and preventative and abortive medications for two weeks, then began experiencing significantly worse symptoms.
- Ophthalmologic examination revealed bilateral optic nerve edema. MRI revealed flattening of the adenohypophysis and symmetric fluid signal along the optic nerves without papilledema. Lumbar puncture (LP) revealed an opening pressure of 36 cm/H2) with normal CSF composition.
- The patient was prescribed acetazolamide and experienced complete resolution of symptoms. After weaning acetazolamide, repeat LP revealed a normal opening pressure, and the patient remained asymptomatic.

FIGURE 1

Brain MRI Without Contrast: Moderate to severe flattening of the adenohypophysis and symmetric fluid signal along the optic nerves without papilledema.



DISCUSSION

- The pathophysiology of IIH is hypothesized to involve the fluid dynamics of cerebrospinal fluid (CSF), including increased CSF production and impaired resorption of CSF.
- Symptoms of IIH include severe headache, vision loss, and pulsatile tinnitus. Physical exam findings may include papilledema.
- As IIH is a diagnosis of exclusion, other causes of increased ICP need to be excluded with imaging and LP.
- Treatment involves decreasing CSF production with acetazolamide, surgical shunting, or optic nerve sheath fenestration.

CONCLUSIONS

- As far as we know, there has been only one other documented case of delayed onset IIH after TBI.
- Practitioners should have a high index of suspicion for IIH in patients with TBI experiencing delayed-onset progressive neurologic deterioration.

REFERENCES

Jensen, Rigmor Højland, et al. “The Diagnosis and Management of Idiopathic Intracranial Hypertension and the Associated Headache.” *Therapeutic Advances in Neurological Disorders*, vol. 9, no. 4, 2016, pp. 317–326., doi:10.1177/1756285616635987.

Mollan, Susan P, et al. “Evolving Evidence in Adult Idiopathic Intracranial Hypertension: Pathophysiology and Management.” *Journal of Neurology, Neurosurgery & Psychiatry*, BMJ Publishing Group Ltd, 1 Sept. 2016, jnnp.bmj.com/content/87/9/982.

Rahman MI, Raveendran S, Kaliaperumal C, Marks C. Pseudotumor cerebri following traumatic brain injury in a 29-year-old man. J Nat Sci Biol Med. 2012;3(1):105-107. doi:10.4103/0976-9668.95987

Thurtell, Matthew J., and Michael Wall. “Idiopathic Intracranial Hypertension (Pseudotumor Cerebri): Recognition, Treatment, and Ongoing Management.” *Current Treatment Options in Neurology*, vol. 15, no. 1, 2012, pp. 1–12., doi:10.1007/s11940-012-0207-4.