

TREATMENT OF INTRACRANIAL HYPERTENSION-RELATED HEADACHE CAUSING LOW CEREBROSPINAL FLUID PRESSURE REQUIRING EPIDURAL BLOOD PATCH

Austin Eells, BS¹, Philip Shumsky, MD², John Freeman, DO², Christopher Wie MD²,
Alaa Abd-Elseyed, MD³, and Natalie Strand, MD²

Background: Patients can have a lumbar puncture for numerous reasons, including therapeutic cerebrospinal fluid (CSF) drainage in patients with idiopathic intracranial hypertension (IIH). Aggressive CSF drainage to alleviate the IIH headache can inadvertently cause a low-pressure postdural puncture headache (PDPH).

Case Report: We report the novel case of a patient with an IIH headache, which was treated with CSF drainage resulting in a PDPH and subsequent return of the IIH headache. Physical examination findings of papilledema and the nonpositional character after the epidural blood patch (EBP), and improvement of symptoms with topiramate and dexamethasone, show that IIH was the likely cause of her post-EBP headaches.

Conclusions: The best-case scenario is to prevent severe cases of PDPH in this patient population. The authors recommend targeting intracranial pressure (ICP) after the treatment of IIH to the high-normal range to prevent PDPH. If IIH headache symptoms arise after EBP, conservative measures to reduce ICP can be utilized, such as acetazolamide, topiramate, and dexamethasone.

Key words: Epidural blood patch, idiopathic intracranial hypertension, lumbar puncture, postdural puncture headache

BACKGROUND

Postdural puncture headache (PDPH) is characterized by positional headache exacerbated by sitting or standing, and occurs within a few days of dural puncture (1). Other common symptoms include neck pain, nausea, photophobia, tinnitus, vertigo, and neck stiffness (2). PDPH is a relatively common complication after intentional lumbar puncture (LP) with an incidence of 0.1% to 36% (1). Intentional lumbar punctures are performed as a diagnostic tool for neurological conditions such as meningitis, hemorrhage, and inflammatory diseases. However, unintentional LP can occur during epidural

catheter placement or epidural steroid injection therapy. Risk factors for PDPH include young age (18-30 years), low body mass index, and use of larger diameter needles (2). Mild PDPH is treated conservatively, while moderate to severe PDPH is often treated with an epidural blood patch (EBP). This case study details a symptomatic rise in intracranial pressure (ICP) following an EBP, causing a new type of headache from the one it was intended to treat.

CASE PRESENTATION

A 27-year-old woman with a history of systemic lupus

From: ¹Mayo Clinic Alix School of Medicine, Mayo Clinic, Scottsdale, Maricopa County, AZ; ²Department of Anesthesiology and Perioperative Medicine, Mayo Clinic, Phoenix, Maricopa County, AZ; ³Anesthesiology, University of Wisconsin School of Medicine and Public Health, Madison, WI

Corresponding Author: Natalie Strand, MD, E-mail: strand.natalie@mayo.edu

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erythematous (SLE), postural orthostatic tachycardia syndrome, intractable headache with suspected idiopathic intracranial hypertension (IIH), and polycystic ovarian syndrome (PCOS) underwent multiple LP procedures during diagnosis and treatment of pulmonary coccidioides with initial suspicion of fungal meningitis. Fungal serologies were persistently negative. She had elevated opening pressures of 20 to 30 cm H₂O with previous LPs. She experienced PDPH following each previous LP. Also, the patient tried conservative management to treat PDPH following 2 previous LPs, once for 2 days and another time for 3 days, with no improvement. The patient had a history of resolution of headaches with EBP procedures following the failure of conservative management in the previous LPs. Several months after her last treatment for coccidioides, a LP was performed to reassess cell count and recheck fungal studies. A 20-gauge spinal needle was advanced to the L2-3 interspace. Opening pressure was found to be highly elevated at 55 cm H₂O, and 22.5 mL of cerebrospinal fluid (CSF) was removed to a closing pressure of 15 cm H₂O. The patient experienced PDPH symptoms, and a blood patch was performed the following day. A total of 12 mL of sterile blood was slowly injected into the epidural space during the EBP with continuous monitoring of the patient for any pain response.

Two days later, the patient was hospitalized with complaints of neck stiffness, pain in the neck and shoulders, malaise, nausea, and vomiting with a concern for increased ICP or iatrogenic meningitis. The patient had blurred vision, light sensitivity, and a nonpostural headache. On physical examination, the patient had papilledema. IIH was diagnosed following normal magnetic resonance imaging (MRI). Her symptoms improved over 3 days with conservative treatment involving daily topiramate 50 mg, dexamethasone 4 mg, and migraine cocktails (ketorolac 15 mg, prochlorperazine 5 mg, diphenhydramine 25 mg). Acetazolamide was not given due to patient allergy. Repeat LP and serologies revealed an opening pressure of 12 cm H₂O with negative serologies before discharge. The patient was discharged on topiramate and methylprednisolone and scheduled for infusions of the migraine cocktail for 3 weeks, by which point her headache had returned to preadmission severity.

CONCLUSIONS

Previous reports show that IIH is a known complication of EBP (3). However, these cases reported that

the IIH onset was weeks to months after first resolving orthostatic headaches from EBP treatment. The authors hypothesized that “rebound” intracranial hypertension may be due to increased CSF production from chronic CSF volume depletion or disrupted CSF resorption with a slowing of CSF flow and subsequent increased pressure following CSF leak repair. However, our patient was not having orthostatic headaches due to chronic CSF leakage, but instead was experiencing an acute PDPH. Additionally, she did not experience headache relief before the onset of the IIH headache 2 days post EBP, indicating that a different mechanism may have been at play. A separate case reported IIH immediately after EBP, presumably due to the direct injection of fluid into the subarachnoid space (4). In contrast to these reports, our patient was previously diagnosed with IIH but was asymptomatic in the months before this event. Her history of IIH may have predisposed her to the development of rebound IIH or recurrence following EBP.

IIH primarily affects overweight women of childbearing age. Other risk factors include PCOS, SLE, recent weight gain, and a family history of IIH (2,5). The patient reviewed in this study had several risk factors for IIH, including a recent 25-pound weight gain, SLE, recently diagnosed PCOS, female gender, and being of childbearing age. Unfortunately, at the time of treatment, this patient’s history of IIH was unknown to the injection provider. A single-center retrospective analysis of 146 patients with IIH who underwent therapeutic CSF removal concluded that low-volume CSF removal to a high-normal range of approximately 18 cm H₂O resulted in IIH-related headache relief with a lower incidence of post-LP headache (6). In this particular patient, iatrogenic IIH after EBP could have been avoided with a lower CSF amount removed. If the patient did not have a severe PDPH after the CSF removal, the EBP could have been avoided.

EBP procedures are generally well tolerated, with transient back pain being the most common complication (25%-35%)(7). The exacerbation of IIH demonstrated in this case study may have been caused by a temporary increase in ICP following the procedure. Careful evaluation of EBP candidates should also include assessing their medical history for previous signs or symptoms of IIH. Suspicion for reflex IIH may be warranted when evaluating patients with complications after an EBP who have predisposing factors to IIH. Consideration should be given to patients most at risk

for worsening side effects from transient increases in ICP, including those with previous intracranial pathology (mass lesion, infection, ventricular defects) (8) and those who do not present with classical PDPH symptoms (9,10). Risk stratification should be assessed on a case-by-case basis. PDPH following LP in patients with a history of IIH should be treated conservatively for longer durations than usual to avoid increases in ICP following the EBP.

Those patients are at higher risk of increased ICP due to the increased production of CSF.

Patients with IIH may be at increased risk of developing increased ICP and headache following EBP for treating PDPH. Patients may require longer durations of conservative management if they develop PDPH following LPs for treating increased ICP.

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