COMPLETE RESOLUTION OF CHRONIC POSTDURAL PUNCTURE HEADACHE FROM CERVICAL DECOMPRESSION AFTER LUMBAR EPIDURAL BLOOD PATCH ADMINISTRATION: A CASE REPORT

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Background:	Neuraxial procedures have the risk of causing dural compromise that leads to postdural puncture headaches (PDPH). PDPH is normally treated conservatively with oral agents, such as nonsteroidal anti-inflammatory drugs and caffeine, or invasively with epidural blood patches (EBP). There is a paucity of evidence for the use of lumbar EBP in cases where the suspected chronic dural defect is at the cervical level.
Case Report:	A 47-year-old patient who underwent C4-C6 posterior extension of fusion as well as right-sided C5-C6 foraminotomy subsequently developed chronic PDPH symptoms that were refractory to conservative interventions. A lumbar EBP was performed for suspected cervical dura compromise, with near-immediate resolution of symptoms that lasted for multiple months.
Conclusion:	Lumbar EBP should be considered in patients with suspected PDPH from cervical dural compromise, especially in the context of a prolonged clinical course or failure of conservative means.
Key words:	Case report, cervical dura compromise, lumbar epidural patch, postdural puncture headache

BACKGROUND

An epidural blood patch (EBP) involves the instillation of autologous blood into the epidural space in order to treat inadvertent or iatrogenic dural puncture and its clinical sequelae (1). This invasive procedure, which has been performed since the 1960s, remains the most effective treatment for postdural puncture headaches (PDPH) in patients who have otherwise failed conservative management with intravenous fluid hydration, bedrest, oral caffeine, and nonsteroidal anti-inflammatory drugs (2,3). While the use of lumbar EBP has been well described and studied in the context of lumbar-level dural defects, there is a paucity of evidence for the use of lumbar EBP in cases where the suspected chronic dural defect is at the cervical level. Here we present the unusual case of a patient who underwent cervical spine decompression, developed the prolonged symptoms classically associated with intracranial hypotension, and, despite negative imaging suggestive of durotomy or puncture, experienced complete resolution of her symptoms after a lumbar EBP.

CASE

The patient is a 47-year-old right-hand dominant woman with a past medical history of axial and radicular low-back pain managed with dorsal column stimulation that was placed in 2014, depression, fibromyalgia, and prior C4-C5 anterior fusion performed in 2006 for left-sided symptoms of cervical radiculopathy and weakness; she presented with progression of these

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same symptoms. Given her progressive symptom burden and failure to respond to conservative management that included cervical epidural steroid injections, she underwent C4-C6 posterior extension of fusion as well as right-sided C5-C6 foraminotomy in April of 2021.

A review of her chart showed multiple visits to the emergency department for this chief complaint. She was admitted to the hospital at the beginning of May for severe holocephalic orthostatic headaches and neck swelling. She reported onset of symptoms one day prior to her presentation, when she described developing a severe headache with standing, as well as clear drainage from her wound after her surgical staples were removed in clinic. She endorsed concomitant photophobia, phonobia, nausea, and vomiting. Her symptoms failed to improve after administration of acetominophen, metoclopramide, ketorolac, diphenhydramine, and intravenous dexamethasone. A computed tomography (CT) scan of her neck was obtained and did show an ill-defined fluid collection. A neurology consultation recommended increase of caffeine intake as well as consideration of empiric lumbar blood patch given the suspicion of intracranial hypotension. However, the patient experienced partial relief of her symptoms on the fourth day of admission after administration of dexamethasone and diazepam and was discharged to home with a recommendation for modified bedrest. No EBP was attempted.

She presented again to the hospital 2 weeks later, in the beginning of June. She reported ongoing symptoms of positional, orthostatic headaches, nausea, vomiting, and vertigo. She was admitted for management of symptoms, and a CT myelogram was obtained which showed no obvious cerebrospinal fluid (CSF) leakage in the cervical, thoracic, or lumbar spine. Neurology was consulted again on this admission and made a recommendation for consideration of empiric EBP. Before the procedure could be scheduled, however, the patient experienced improvement in her symptoms after administration of diazepam and intravenous dexamethasone. She was discharged home.

She thus presented to our clinic at the end of June for initial evaluation of these symptoms and consideration of lumbar EBP. The procedure was scheduled for the beginning of July.

Briefly, for this procedure, we obtained blood from her left antecubital vein in a sterile fashion; with the patient in a sitting position, the skin was prepped in a sterile fashion and anesthetized with 1% lidocaine at the approximate L4-L5 level, and an 18-gauge Tuohy needle was advanced into the posterior epidural space via a midline interlaminar approach using a loss-ofresistance technique. Approximately 19 mL of blood was injected into the epidural space. The patient was asked to lay in a supine position for 30 minutes. After 30 minutes, the patient was happy to report a dramatic improvement in her symptoms while sitting and standing and near-complete resolution of her headache and nausea.

Further review of her chart in the intervening months showed a clear endorsement of resolution of her symptoms without noted adverse effects from the EBP. Institutional policy did not require informed consent for this case report due to lack of patient identifiers.

DISCUSSION

PDPH, as a complication of intracranial hypotension, has been described since the first spinal anesthetic was administered in 1898 (4). The overall incidence of PDPH from neuraxial procedures ranges from 6% to 36%, with noted variable ranges depending on the type of procedure and spinal needle size used for specific interventions (3,5).

There are a number of risk factors for the development of PDPH which include female gender, history of headaches, age between 31 and 50 years old, low body mass index, and use of a cutting spinal needle for accessing the neuraxis, as well as an orientation of the needle bevel that is perpendicular to the long axis of the spinal column at the time of dural puncture (3,6). An awareness of these risk factors explains why PDPH is commonly seen among young, non-obese, parturients who undergo neuraxial anesthesia for labor or delivery (7). Other common procedures that may result in symptoms of PDPH are diagnostic lumbar punctures, epidural steroid injections, intrathecal pump placement, spinal cord stimulator placement, and spine surgery (5).

Symptoms of PDPH are characterized by an orthostatic frontal or occipital headache that is alleviated by recumbent positioning, with associated symptoms of neck pain, tinnitus, changes in hearing – usually hyperacusis, photophobia, and nausea (5,7). The diagnosis of PDPH can be made clinically in those patients with a high suspicion of dural puncture or trauma. The International Classification of Headache Disorders offers specific criteria for diagnosis which includes headache that develops within 5 days of dural puncture, is consistent with the symptoms listed above, and is not better explained by another etiology. Neuroimaging is not required but may be helpful since intracranial hypotension can be suggested by evidence of CSF leakage on imaging. Alternatively, an opening pressure of < 60 mm CSF during lumbar puncture can be suggestive of intracranial hypotension (5).

Treatment for PDPH ranges from conservative to invasive measures. In general, PDPH has a benign course, arising usually 24 to 48 hours after dural compromise and is self-limiting (8). Bed rest and hydration tend to be sufficient until its resolution, which tends to occur 2 weeks or less after dural compromise (5,11). However, various medications have been utilized for symptom management. Medications commonly used include acetaminophen, gabapentin, pregabalin, and steroids with each having variable, but positive efficacy in decreasing symptoms of PDPH compared to placebo (5,9). EBP, while an invasive procedure, has been shown to improve symptoms immediately or with a delay 70% to 90% of the time. Due to its high efficacy, it is considered the method of choice for treatment of PDPH, especially when conservative measures fail or when symptoms are severe (9,10). However, EBP is not without complications with reports of back pain, radicular symptoms from blood products causing irritation, and pyretic reactions. In less common cases, chronic adhesive arachnoiditis, subdural or spinal hematoma, seizures, and cerebral ischemia can be seen (8).

The mechanism by which EBP resolves PDPH symptoms is still up for debate. One such explanation has to do

with increased thecal pressures. The administration of blood products causes volume expansion within a confined space and consequently normalizes intracranial pressures. Another explanation is a rapid coagulation cascade leading to clot formation around the defect, thus leading to cessation of CSF leakage (12). Both processes could occur simultaneously, but further investigation is necessary.

CONCLUSION

For persistent or severe PDPH, EBP has been proven to reduce the burden of symptoms, allowing for a better quality of life. In this case report, we present the case of a patient who underwent cervical decompression and subsequently developed long-lasting, debilitating symptoms of PDPH which completely resolved after undergoing empiric treatment with a lumbar EBP. Based on our review of the literature, there are limited reported cases similar to this one, thus raising a unique consideration for physicians. Given the response the patient experienced in the context of her history, we would consider lumbar EBP in patients with suspected chronic PDPH from cervical dural compromise, with or without imaging confirmation, and especially in the context of a prolonged clinical course or failure of conservative means. Such treatment can provide patients with improvement in their quality of life.

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