Case Report

Delayed Radicular Pain Following Two Large Volume Epidural Blood Patches for Post-Lumbar Puncture Headache: A Case Report

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Free full manuscript: www.painphysicianjournal.com **Introduction:** Postdural puncture headache (PDPH) is a known complication of diagnostic lumbar puncture. Multiple factors including needle size, type, and needle bevel orientation, have been postulated to contribute to the development of PDPH. The presentation of PDPH tends to have classic symptoms that include a postural headache, nausea, vomiting, tinnitus, and ocular disturbances. Conservative treatment measures include bed rest, intravenous hydration or caffeine, and analgesics. Resistant cases might require an epidural blood patch (EBP). Though complications are rare, cases of immediate post-procedural pain and subdural epidural hematoma have been reported. Here we present a case of PDPH treated with sequential EBPs that resulted in delayed radicular pain.

Case Report: A 29-year-old female presented to the emergency room with a severe frontal headache of several days duration. She underwent a diagnostic lumbar puncture as a part of her work-up. Then, 24-48 hours later she developed a severe postural headache unresponsive to conservative care. Two days later she underwent an epidural blood patch with 20 mL of autologous blood. Her symptoms did not abate, prompting a repeat EBP within 24 hours with an additional 20 mL of autologous blood. Five days later the patient began experiencing muscle spasms and radicular pain in the buttocks and left posterior leg that radiated to her posterior calf. The patient was initially started on pregabalin 25mg 3 times daily, and underwent a gadonlinum-enhanced MRI of the lumbar spine. She followed up 5 days later with unchanged symptoms and a negative MRI. She was then started on a methylprednisolone taper and continued the pregabalin. At the 10-day follow-up, there was 90% resolution of symptoms and a pain intensity of 1/10 on NRS. At this time she is continuing the pregabalin with plans to discontinue medication.

Discussion: Although EBP is typically a safe procedure, complications might occur. An inflammatory response, secondary to the injection of blood, or mechanical compression, due to the total volume of blood injection, are highlighted as possible causative agents in the development of this complication. The role of fluoroscopic imaging, particularly in patients who have failed an initial EBP, must also be examined. Given the rates of false loss of resistance (17-30%) reported in the literature, the use of real-time imaging to ensure proper needle placement and subsequent injectate spread should be considered.

Key words: Blood patch, epidural, radiculopathy, postdural puncture headache, complications, fluoroscopy, epidural

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ostdural puncture headache (PDPH) is a known complication of diagnostic lumbar puncture. Multiple factors including needle size, type, and needle bevel orientation have been postulated to predispose to the development of PDPH (1). The

presentation of PDPH tends to include classic symptoms such as a postdural headache that is relieved by lying down, as well as nausea and vomiting. Patients might also experience tinnitus and ocular disturbances. The initial treatment for this condition includes bed rest, caffeine, and analgesics. An epidural blood patch (EBP) might be considered if the previously mentioned therapies do not alleviate the symptoms. Typically, conservative options are exhausted prior to utilization of EBP due to persistent concerns regarding efficacy, optimal timing, safety, and mechanism of action particularly as the majority of PDPHs resolve within 6-7 days (1). Furthermore, when an EBP is employed, the optimal volume of injection and the use of image guidance are relatively controversial. Various theories abound regarding the ideal volume of blood injectate to achieve maximal effectiveness for an EBP, however consensus is fleeting (2). Image-guidance with fluoroscopy does not appear to be routine and is often institutionally driven. Here we present the first reported case of PDPH treated with sequential highvolume blind epidural blood patches that resulted in delayed radicular pain.

CASE DESCRIPTION

A 29-year-old female presented to the Emergency Room with severe non-positional frontal headache and fever of several days duration. Due to the severity of her symptoms and her low grade fever (99.6° F) she underwent a diagnostic lumbar puncture with a 20-gauge Whitacre needle (BD, Franklin Lakes, NJ). The number of attempts prior to successful puncture were unrecorded. Her original symptoms abated during her emergency room visit, she was treated with hydromorphone, oxycodone, ibuprofen, metoclopramide and intravenous hydration. The results of the lumbar puncture were normal (Table 1). Neurological evaluation considered her primary headache to be a mixed vascular and muscle contraction variant. Then, 24-48 hours later, she developed a severe postural bifrontal and occipital headache unresponsive to bed rest, intravenous and oral fluids, intravenous caffeine, and analgesics (oxycodone and acetaminophen, butalbital, and caffeine). Two days

Table 1. Cerebrospinal fluid cell count.

Test	Result
Appearance Fluid	Clear
Color CSF	Colorless
WBC CSF	0 mm ³
Lymph CSF	0 %
Seg CSF	0 %
RBC CSF	11 mm ³

later, she underwent a non-fluoroscopically guided EBP with 20 mL of autologous blood. Her symptoms only partially resolved, resulting in a repeat non-fluoroscopically guided EBP within 24 hours with an additional 20 mL of autologous blood.

Five days later, the patient began to experience muscle spasms in the left buttock and radicular pain in the left posterior leg that radiated down to her posterior calf. She described the sensation as aching and gnawing. Her pain was rated as a 5.5/10 on the numerical rating scale. Positions that aggravated her symptoms included standing, sitting, and bending. Her pain was alleviated with lying down and walking. She denied any bowel or bladder dysfunction or muscle weakness.

At the time of presentation to the University Pain Center, the patient was taking acetaminophen, butalbital, caffeine, ducosate, and metoclopramide. Her past medical history was significant for asthma, gastrointestinal ulcer, and irritable bowel syndrome.

The patient's physical examination was significant for a positive seated straight leg raise test reproducing her left lower extremity symptoms. Otherwise, her neurological exam demonstrated that cranial nerves II-XII were grossly intact, sensation to light touch and pinprick was present in all dermatomes, and reflexes were 2+ equal and symmetric throughout. Her musculoskeletal exam was significant for 5/5 strength in her upper and lower extremities bilaterally at all myotomes tested.

A gadolinium-enhanced magnetic resonance image of the lumbar spine was ordered and the patient was started on pregabalin 25 mg, 3 times daily. She followed up 5 days later with unchanged symptoms; the MRI did not demonstrate any relevant pathology; epidural hematoma and arachnoiditis were excluded (Figs. 1-3). A methylprednilisone taper was added to her medication regimen at this visit. At the 10-day follow-up, there was 90% resolution of symptoms. She subsequently discontinued her pregabalin at her one month follow-up and did not report any residual symptoms.

Discussion

Although the exact mechanism of the postdural puncture headache (PDPH) is unknown, it is postulated that downward traction on intracranial structures occurs due to cerebrospinal fluid (CSF) leakage from a dural puncture site, resulting in pain. Alternatively, the modified Monroe-Kelly theory hypothesizes that there is a reciprocal relationship between CSF volume



Fig. 1. Lumbar spine magnetic resonance image-sagittal view, normal.

and intracranial blood volume. A decrease in CSF volume should therefore result in vasodilation leading to migraine-like headaches (3).

PDPH occurs in approximately 10-40% of patients who undergo a lumbar puncture (4). Spinal puncture might also be a consequence of widely utilized interventional techniques (5-16). A recent Cochrane review suggested there are inadequate studies evaluating PDPH response to EBPs; however, clinically, EBPs have been accepted as a gold standard treatment (17,18). Originally developed in 1960 by Gormley (19), it was found that patients who experienced a "bloody tap" had a lower incidence of headaches. Currently, 10-20 mL of autologous blood is typically injected at or below the site of the lumbar puncture until the patient reports a pressure sensation. The mode by which EBP relieves PDPH is not well understood; however, there are various theories which might explain the mechanism of action (20).

The "plug" theory suggests that preventing further CSF loss by plugging the dural puncture site with blood allows the dural hole to heal through normal reparative processes (18). While this hypothesis is supported by evidence of headache relief during the time frame of CSF regeneration, it does not explain the role that other therapies, such as caffeine, colloids, and crystalloids, play in alleviating symptoms of PDPH. The pressure theory suggests that injected fluid increases the epidural pressure and subsequently elevates the subarachnoid CSF pressure. This displaces



Fig. 2. Lumbar spine magnetic resonance image-sagittal, gadolinium-enhanced, normal.



Fig. 3. Lumbar spine magnetic resonance image-axial view, L4-5 disc level, normal.

spinal CSF into the cranium and restores CSF volume and pressure, resulting in reduced traction on pain fibers, resolving painful symptoms. Another theory suggests that EBP reverses vasodilatation, resulting in an immediate decrease in cerebral blood flow, possibly mediated by deactivation of adenosine receptor responses as intraspinal pressures rise or by release of vasoactive substances. Yet another theory states that EBP blocks the normal lymphatic and arachnoid villae drainage of CSF from the subarachnoid space into the venous system, allowing rapid replenishment of CSF volume. Complications following EBP, although rare, have been reported. Most commonly, acute paresthesias, neck and back pain, and transient temperature elevations have been observed. Additionally, rare complications including spinal subdural hematoma, spinal subdural epiarachnoid hematoma, intrathecal hematoma, chronic back pain secondary to a calcified epidural blood patch, and arachnoiditis are noted in the literature. Neurologic deterioration secondary to increased intracranial pressure following EBP has also been cited (21-27). Delayed radicular pain has not yet been noted as a complication to EBP.

We postulate that these symptoms might be due to either an inflammatory response, secondary to the injection of blood, or mechanical compression, due to the total volume of blood injection. The inflammatory role of heme has been well documented in animal and human models. In cases of ruptured aneurysms in the subarachnoid space, hemolysis of blood incites an inflammatory response that chronically irritates the leptomeninges in the post-hemorrhage period (28). The fibroproliferative reaction that ensues in the intrathecal compartment might lead to arachnoiditis anywhere in the neuroaxis (29-31). Furthermore, the common causes of noninfectious arachnoiditis include the intrathecal injection of blood—which has been shown to produce neurological deficits in dogs (32) — and entry of blood into the intrathecal space during spinal surgical interventions (33). Although there was no evidence of arachnoiditis on magnetic resonance imaging, the inflammatory role of blood cannot be ignored as a possible etiology for this patient's radicular symptoms. The total volume of injected blood and hence, the mechanical role of blood in this setting, might also explain her symptoms, although this is less likely given the MRI findings. In combining her 2 EBPs, 40 mL of blood was injected into this patient's lumbar epidural space within 24 hours. There is research to suggest that this amount of blood might have resulted in compression on the spinal roots emerging from the cauda equina, producing the radicular pain (34).

Beards et al (35) demonstrated via magnetic resonance imaging that epidural blood patches produce a "mass effect" compressing the thecal sac. Over time, some of the red cells in the clot pressing on nerve roots might be removed by phagocytosis and the serum might be absorbed through the epidural lymphatics, resulting in relief of radicular pain. Although there was no evidence of persistent blood or clot on MRI, complete pain relief might have been obtained once the whole blood clot was organized and absorbed, which is postulated to occur as early as 10 days, or more likely via an anti-inflammatory mechanism following treatment with methylprednisolone (36).

Optimal performance of EBP might lead to a decreased rate of complications. First, needle placement with image guidance and volume of delivery might be important factors in maximizing safety and efficacy. Use of image guidance during EBP is inconsistent and institutionally driven; fluoroscopic guidance during EBP is not uniformly utilized. Given the published failure rates (14-63%) during blind epidural injections, the use of real-time imaging and contrast medium when available might be prudent particularly during repeat EBPs (37-41). Furthermore, use of fluoroscopy might allow more targeted delivery of injectate close to or at the site of the dural puncture, possibly reducing the need for high volume injections.

The volume of autologous blood used during EBP might also predispose to the development of symptoms. While the optimal volume of autologous blood to inject during EBP remains controversial, the amounts used in clinical practice have increased over time. Presently, 20 mL is cited as the target volume to enhance patch efficacy (42). However, in a study performed by Taivanen (43) comparing effectiveness of epidural blood patches in patients who received 10 mL versus a height-adjusted volume of 10-15 mL, no difference in efficacy could be detected. The use of large volumes might be unnecessary, particularly in comparison to fluid injection as the injection of autologous blood maintains CSF and epidural pressures for an extended period of time. It was demonstrated by Coombs (44) that an injection of 15 mL of blood produced a threefold increase in CSF pressure, which was sustained at 70% of its peak injection pressure approximately 15 minutes later. While repeat epidural blood patches are advocated in the setting of persistent symptoms (7), further studies should evaluate the time frame between the injections and the efficacy of repeating the procedure. This patient received two 20 mL EBP injections less than 24 hours apart. Although there does not appear to be a contraindication to repeating the procedure, there are no controlled studies to examine the adverse effects of a short interval between high-volume EBPs.

CONCLUSION

Delayed radicular pain might occur as a complication of EBP. While the exact etiology is unclear, it might be secondary to hemolytic irritation of the nerve roots or cord or nerve root compression. Maximizing EBP effectiveness might be the best way to prevent these symptoms. We recommend the use of fluoroscopy during repeat EBPs to ensure proper needle placement and subsequent spread of injectate. We also recommendfurther studies to evaluate both the optimal blood volume for the procedure as well as the efficacy of repeat injections.

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