Case Report

THORACIC EPIDURAL BLOOD PATCH IMPROVES NEUROLOGIC SYMPTOMS FOLLOWING SURGICAL EVACUATION OF BILATERAL SUBDURAL HEMATOMAS: A CASE REPORT

Anthony Halupa, DO, Michael Dorbad, DO, Mary Herman, MD, PhD, and Abdel Ragab, MD

Subdural hematomas (SDHs) are a common entity occurring after major trauma, often in elderly patients. Rarely, SDHs have been reported following dural tears from spinal or epidural anesthetics. A decrease in cerebrospinal fluid (CSF) volume and resulting intracranial hypotension may cause orthostatic headache, nausea, vomiting, visual changes, and seizures. Intracranial hypotension has been reported as a source of SDH, following CSF volume loss (1). We report a case in which bilateral chronic SDHs, likely resulting from multiple spontaneous thoracic dural tears, were relieved by thoracic epidural blood patch (EBP) following surgical evacuation of the SDH.

CASE

A 29-year-old female presented to her primary care physician (PCP) with a 2-month history of a progressively worsening 10/10 occipital headache and associated nausea, vomiting, tinnitus, bilateral ear pain, occasional loss of hearing, blurry vision, and intermittent numbness of her extremities. Four days prior to presentation, the patient fell on ice onto her shoulder. She denied hitting her head or loss of consciousness. Her previous medical history consisted of gastroparesis of unknown etiology and a family history of granulomatosis with polyangitis. Computed tomography (CT) and magnetic resonance imaging (MRI) of her brain with and without contrast revealed bilateral extra-axial collections present around both

cerebral convexities measuring up to 10 mm, which were consistent with chronic SDHs (Fig. 1). A CT angiogram of the head and neck showed no aneurysm, vascular malformation, flow-limiting stenosis, occlusion of the major intracranial vessels, nor evidence of dural arteriovenous fistula or sinus thrombosis.

The following day, the patient experienced worsening headaches, pupillary changes, and diplopia. A repeat CT scan showed new multifocal areas of increased density within the bilateral extra-axial collections consistent with acute-on-chronic SDH. MRI of the spine showed fluid between the dura and thecal sac throughout the entire thoracic spinal canal, consistent with a dural tear as the likely cause of the SDH. The origin of the CSF leak, however, was not able to be identified.

The patient underwent a bilateral craniotomy to evacuate the SDHs with placement of a right exter-

Fig. 1. Perioperative MRI demonstrating bilateral SDHs.

From :Department of Anesthesiology, Geisinger Medical Center, Danville, PA

Author for correspondence: Michael Dorbad, DO
Address: Department of Anesthesiology, Geisinger Medical Center, 100 N Academy Ave., Danville, PA 17821
E-mail: Mdorbad1@geisinger.edu

nal ventricular drain and bilateral subgaleal drains. Postoperatively she developed worsening headache, with right hand paresthesias. A CT myelogram of the spine showed focal dense contrast seen at the dorsal lateral right T8-T9 and dorsal lateral right T10-T11 levels, thus identifying sites of potential dynamic CSF leakage (Fig. 2). Following discussion of potential treatment options, the patient elected to have an EBP, knowing that it may not be able to repair both CSF leak sites.

Using the loss of resistance technique, an EBP was performed at T10-T11 in the sitting position using an 18-gauge Tuohy needle and aseptic technique. Thirty mL of autologous blood was delivered to the epidural space in 10 mL increments, with immediate improvement of her symptoms. Surprisingly, she experienced no back pain with epidural blood injection. The patient laid flat for one hour following the procedure. She remained headache free overnight. The morning after the blood patch repair, the patient developed numbness and tingling in the right hand and face with headache, nausea, and an unsteady gait. A CT of the head and electroencephalography (EEG) were ordered. The CT showed no new SDH, a slight increase in the overall CSF density secondary to the recent intrathecal administration of contrast, and a hyperdensity within the region of the occipital horns of both lateral ventricles representing residual contrast within the CSF. The EEG showed no seizure-like activity. The neurological deficits resolved within 24 hours, along with improvement of her headache and nausea.

One week after the initial epidural blood patch procedure, the patient had a return of her headache symptoms. A second blood patch was performed at T8-T9 using an 18-gauge Touhy needle to access the epidural space under direct fluoroscopic guidance. An epidurogram with 6 mL of Isovue 200 dye (Bracco Diagnostics Inc., Cranbury, NJ) was performed to confirm the placement of the needle, and 30 mL of autologous blood was injected into the epidural space. The patient remained flat for one hour and was positioned at 45 degrees for another hour. The procedure was well-tolerated by the patient, with her only complaint being a mild headache. The patient was discharged home 6 days after the second blood patch procedure.

Approximately 2 months following discharge, the patient was evaluated for worsening nonpositional headaches with nausea, vomiting, and fever. CT and MRI of the head were performed, which showed no evidence of interval changes compared to previous CT scans as well as no evidence of intracranial hypotension. A fluoroscopy-guided lumbar puncture was subsequently performed, which was negative for infection. Her symptoms subsided during a short hospital course. One month after discharge, the patient presented to an outside hospital and was admitted for seizure-like activity as well as persistent nausea, vomiting, and dehydration. A CT scan determined she developed bilateral subdural hygromas without evidence of CSF leak, and she was started on Depakote 500 mg (AbbVie Inc., North Chicago, IL) twice a day. She continues to have

Fig. 2. CT myelogram showing tear at T10-11 (left), clearance of dye at T9-10 (middle), and second tear at T8-9 (right). Arrows demonstrate areas of increased, bright, punctuate contrast indicating contrast leak into epidural space.

chronic headaches along with occasional episodes of blurred vision and nausea.

DISCUSSION

Spontaneous intracranial hypotension (SIH) is an uncommon condition, with a prevalence of about one per 20,000 patients (2). SIH is characterized by positional headaches and decreased CSF pressure without trauma or procedurally-induced dural insult. Although the pathophysiology of SIH may be unclear, one theory states that CSF volume loss may be due to the weakening and subsequent rupture of the dura. Supporting this theory, people with certain connective tissues diseases, such as Ehler's-Danlos syndrome and Marfan's syndrome, have an increased risk of SIH (3).

Orthostatic headache is the paramount complaint with SIH and is often associated with symptoms such as nausea, dizziness, and changes in vision. Orthostatic headaches are frequently attributed to low CSF volume secondary to leak. The International Headache Society defines headaches secondary to SIH as being orthostatic with either low CSF opening pressure, sustained improvement after EBP, demonstration of an active spinal cerebrospinal fluid leak, or cranial MRI associated with changes of intracranial hypotension (e.g., brain sagging or pachymeningeal enhancement), without recent history of dural puncture nor attributable to another disorder (4). In SIH, as the CSF volume decreases, the brain loses its buoyancy, which results in pressure on pain-sensitive venous sinuses and subsequent headache. With progressive egress of CSF, there is greater downward displacement of the brain, which places increased tension on the bridging veins and increases risk of SDH.

As discovered through neuraxial anesthesia, increasing needle thickness and the resulting size of dural tear positively correlate to incidence of headache and intracranial hypotension that may complicate dural puncture (5,6). Our patient appeared to have developed spontaneous dural CSF leaks which led to intracranial hypotension and a caudal shift of the brain, with traction on the arachnoid mater and dural veins. This likely caused lesions to the blood vessels, blood extravasation, and formation of SDHs. The timing of her fall is likely secondary to her underlying pathology. Large dural tears can distort several major inputs to a patient's balance including auditory,

vestibular, and ocular function due to traction of cranial nerves (7). While interrogation of our patient's auditory function was unyielding, she did complain of increased headache with sound, tinnitus, occasional hearing loss, and bilateral ear pain. She was noted to have a diplopia during an ophthalmology exam, secondary to cranial nerve VI palsy, suspected due to traction on the nerve. Additionally, she mentioned intermittent peripheral numbness of her extremities and ataxic gait which may have been an overall decrease in her proprioception.

The vast majority of dural tears and subsequent CSF leaks are self-limiting and treated conservatively with bed rest, caffeine, and increased fluid intake. Although EBP may provide immediate relief of SIH symptoms, it may not always lead to resolution of the CSF leak. The EBP can be repeated, several times if necessary, and a larger volume of blood, up to 30 mL, can be considered for injection. The ideal blood volume to be used has been controversial. One study showed that the success rate of the EBP increases slightly with the amount of blood used, however this did not reach statistical significance (8).

While EBPs can be performed at any level, most are placed at the level of the dural tear, which most commonly occurs in the lumbar region secondary to dural tears from laminectomy, spinals, and epidurals. Although placing the EBP at the levels of the cervical and thoracic spine are associated with higher risks, specifically-directed EBP placement is thought to be more successful in providing long-term relief (9).

In patients with SDH secondary to dural tears it may be unclear whether to continue with evacuation and drainage of the hematoma vs. EBP as the first step in management. Most commonly, as in our case, the SDH is noted first and treated to avoid herniation secondary to displacement of brain by the expanding hematoma. However, excess drainage of CSF may decrease CSF volume further and create a potential re-bleed phenomenon; thus, some recommend treating the CSF leak as primary treatment (10). Cost, extended recovery, and lowered success rates following craniotomy without repair of the dural tear may make surgical intervention alone less appealing. In our case, the patient had worsening neurologic symptoms and an acute-on-chronic hemorrhage diagnosed with repeat imaging prior to surgical drainage. EBP was performed shortly after the evacuation, minimizing

the potential for re-bleeding, and since she had a ventricular drain placed, there was little risk of causing increased intracranial pressure with EBP. As noted in a similar case, delay in EBP after craniotomy can lead to a clinical deterioration (1,11).

While specific mortality data about SDH from dural insult is difficult to estimate, SDH is often seen in a much older patient population and has a fairly high mortality rate (12). In patients with a history of orthostatic headaches who develop SDH, diagnosis of SIH and potential CSF leak needs to be ruled out. If missed, the patient is at greater risk for re-bleeding due to unresolved pathology initiating the SDH. Despite advanced imaging, it may still be difficult to identify the specific site of the dural tear (13). In patients with symptoms refractory to conservative

management, EBP is the treatment of choice for dural tear due to its low invasiveness, decreased risk profile relative to surgery, and high improvement rate. Caution must be exercised in patients with SDH receiving an EBP because these patients may already have intracranial hypertension due to an increased blood mass in the calvaria. Timing of EBP in relation to SDH evacuation is critical to provide the safest treatment course.

The seemingly benign condition of CSF loss, often undiagnosed and usually self-limiting, on rare occasions can result in devastating neurologic dysfunction ranging from chronic headaches, cranial nerve palsy, and SDH, as exemplified in our patient. EBP may prevent progression of symptoms following surgical intervention.

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