

Case Report

Chronic Subdural Hematoma after Spontaneous Intracranial Hypotension : A Case Treated with Epidural Blood Patch on C1-2

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Spontaneous cerebrospinal fluid (CSF) leak is a recognized cause of spontaneous intracranial hypotension (SIH). Subdural hematoma (SDH) is a serious but rare complication of SIH. An autologous epidural blood patch at the CSF-leak site can effectively relieve SIH. We report a case of bilateral SDH with SIH caused by a CSF leak originating at the C1-2 level. A 55-year-old male complained of orthostatic headache without neurological signs. His symptoms did not respond to conservative treatments including bed rest, hydration and analgesics. Magnetic resonance imaging showed a subdural hematoma in the bilateral fronto-parietal region, and computed tomography (CT) myelography showed a CSF leak originating at the C1-2 level. The patient underwent successful treatment with a CT-guided epidural blood patch at the CSF-leak site after trephination for bilateral SDH.

Key Words : Blood patch · Epidural · Intracranial hypotension · Subdural hematoma.

INTRODUCTION

Spontaneous intracranial hypotension (SIH) is characterized by an orthostatic headache in the absence of a history of trauma or dural puncture. SIH is caused by spontaneous cerebral spinal fluid (CSF) leakage of unknown etiology at the level of the spine. Most SIH patients recover after bed rest, hydration, applying an abdominal binder and administration of caffeine and steroids. Application of epidural blood patches (EBP) at the CSF-leak site offers another treatment option. Some SIH patients encounter a subdural hematoma (SDH) as severe complication with neurologic deficits.

In SIH with CSF leak at the high cervical region, EBP has traditionally been performed in the lumbar area or in the thoracic and lower cervical area^{4,13,14}. Because a direct EPB at the leak site may present challenges due to the narrow space of region and its proximity to important neural structures, the medical literature has reported only two cases involving an EBP procedure performed at the C1-2 level^{7,13}.

We report the case of a bilateral SDH patient with SIH who came to our hospital and was discharged two weeks later with

no neurologic deficit after trephination and EBP treatment. In addition, the patient had no residual symptoms or recurrence at six-month follow-up.

CASE REPORT

A 55-year-old male had a three-month history of progressive headaches and neck pain without history of trauma. Brain magnetic resonance (MR) imaging revealed a subdural hematoma in the fronto-parietal region, and cervical MR imaging at that time showed dural thickening enhancement of the spinal canal (Fig. 1). This patient was diagnosed with a chronic subdural hematoma after SIH. The patient underwent a computed tomography (CT) myelography of the entire spinal column, which localized a generous leak at the C1-2 level, with contrast extravasation in the epidural space (Fig. 2). The patient had progressive symptoms and increased subdural hematoma in subsequent CT imaging.

A trephination of the subdural hematoma was performed and the hematoma was drained for three days. After two days of trephination, a direct cervical blood patch was performed under CT guidance at the C1-2 level to prevent a recurrence of subdural hematoma. With the patient resting in a supine position on the CT gantry, we guided a 22-gauge needle into the left lateral epidural compartment at the C1-2 level. We positioned the needle adjacent to the thecal sac, matching the site of the leak seen on the CT myelography (Fig. 3). We also confirmed the needle position with an injection of 0.5 mL of iohexol (Om-

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nipaque 240, Nycomed Amersham, Oslo, Norway). We then performed the blood patch by an injection of the patient's autologous blood obtained from the right brachial vein. The injection was stopped at 5 mL, at which point the patient noticed an increased pressure sensation in his neck. Findings of a neurologic examination performed after the procedures were normal. The patient's neurological signs were observed for a period of two weeks. At the time of discharge, his headache was almost completely relieved. His headache had completely dissipated three months after the EBP.

An MR imaging of the brain taken three months after the procedure showed no more dural enhancement and no fluid collection in the subdural space (Fig. 4). A CT myelography at the six-month follow-up revealed no contrast extravasation in the epidural space (Fig. 5). At present, the patient is in good health condition and reports no headaches.

DISCUSSION

Spontaneous intracranial hypotension, as the name implies, is caused by low CSF pressure, usually secondary to an occult leak. A CSF leak occurs in weak areas around the dura mater and

nerve root sheaths and around small defects due to small traumas, a fall, severe exercise, or a cough that tears the dura or arachnoid^{1,12}.

Some studies have reported that connective tissue disorders such as Marfan syndrome, Ehlers-Danlos syndrome type 2, and autosomal dominant polycystic kidney disease play a significant role in causing SIH^{9,14}.

While the pathophysiology of SDH in patients with SIH remains unknown, studies have proposed several mechanisms. Downward displacement of the brain due to low CSF pressure may produce tears in the bridging veins of the dural border cell layer, causing these veins to rupture. Alternatively, as subdural CSF collections gradually enlarge the subdural space, the bridg-

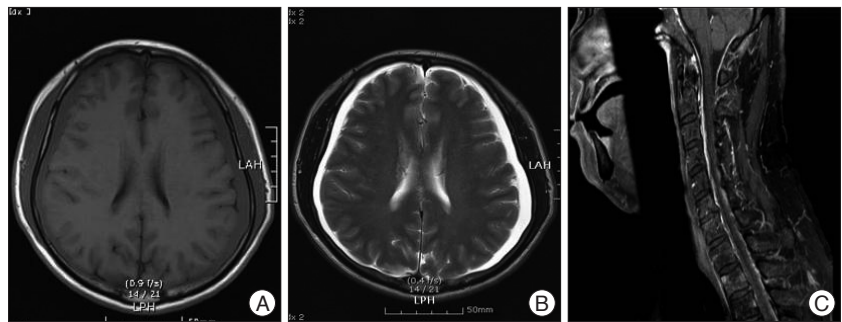


Fig. 1. Preoperative magnetic resonance imaging. Brain non-contrast T1-weighted (A) and T2-weighted (B) axial images show a subdural hematoma over both frontal and parietal convexity. Cervical contrast-enhanced T1-weighted (C) sagittal image shows dural thickening enhancement.



Fig. 2. Coronal (A) and axial (B) computerized tomographic scanning shows epidural contrast material accumulation at the C1-2 levels.

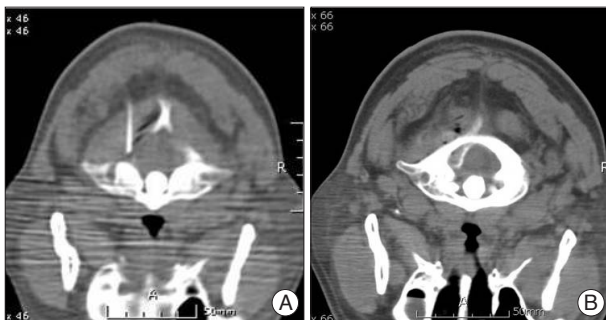


Fig. 3. An axial computerized tomographic (CT) scanning (A) shows needle placement in the left lateral epidural compartment at the upper C2 level followed by administration of the blood patch. An axial CT scanning (B) shows that contrast material injected to confirm the epidural location is identified with mild flattening of the lateral thecal sac margin.

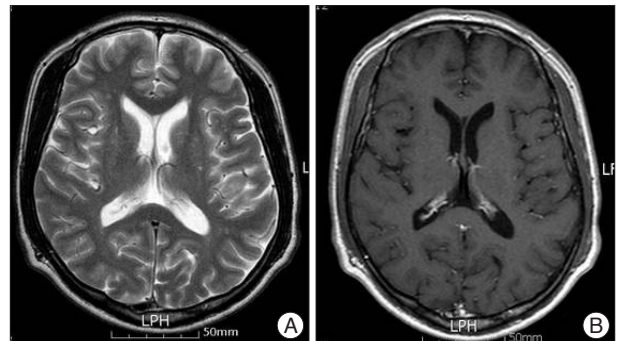


Fig. 4. Brain magnetic resonance imaging three months after epidural blood patch. Axial T2-weighted (A) and contrast-enhanced T1-weighted (B) images show no subdural hematoma and pachymeningeal thickening enhancement.

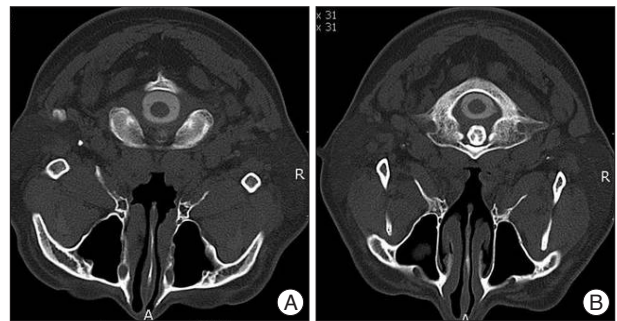


Fig. 5. Axial computerized tomographic scanning (A and B) six months after epidural blood patch show no epidural contrast material accumulation at the C1-2 levels.

ing veins may stretch and rupture in some cases¹⁴.

Although the most common presenting symptom in SIH is orthostatic headaches, the exact mechanism of orthostatic headaches in CSF leak is unknown. The total volume of the brain, CSF, and the intracranial blood remains constant inside the rigid skull. Therefore, a decrease in one of these components should cause a reciprocal increase in either or both of the remaining two⁸). The intracranial venous structures are pain-sensitive, and their dilatation in turn may lead to headaches.

MR imaging represents the method of choice to depict intracranial manifestations; the neuroimaging features include diffuse meningeal enhancement, acquired Chiari malformation, and subdural fluid collections¹¹). The Monro-Kellie hypothesis is the mechanism frequently used to explain MRI findings with aforementioned conditions⁹). A reduction in the volume of the CSF requires an increase in volume of one or both of the other components. The most reliably demonstrated area of increased volume on imaging is the pachymeninges, which show diffuse thickening and enhancement with gadolinium-enhanced MRI due to lack of a blood-brain barrier and an increase in the volume of venous blood in this compartment¹⁵). In cases of SIH, the site of the CSF leak rests predominantly in the cervical or thoracic region, and the diagnosis is typically established by CT myelography or radionuclide imaging^{4,6}). In our patient, CT myelography was instrumental in identifying the leak site.

Although supportive measures and medical therapy such as hydration, bed rest, caffeine, steroid and parenteral fluid may provide temporary relief, a more durable treatment is to seal the site of the leak. The mainstay of the treatment is the injection of autologous blood (10-20 mL) into the spinal epidural space. Relief of symptoms is often dramatic after EBP. If EBP fails the first time, it can be repeated³). Complications of cervical EBP include spinal cord and nerve root compression, chemical meningitis, intrathecal injection of blood, seizures, and stiffness of the neck²). Cases of large subdural hemorrhage require surgical drainage and treatment of the underlying cause of SIH¹⁰). With the current technology, we can perform imaging-guided procedures in the spine with relative safety and minimal discomfort to the patient. In cases of cervical leaks, it is reasonable to offer a cervical blood patch as the initial treatment¹³). In our patient, after trephination of subdural hematoma, we performed EBP at the C1-2 level.

CONCLUSION

We report the case of a bilateral SDH as a severe complication of SIH with a CSF leak originating at the C1-2 level. The authors believe that an EBP performed directly at the site of the leak as the initial treatment can more effectively seal the defect.

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