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Case Report

Early use of targeted blood patch in spontaneous intracranial hypotension presenting with bilateral subdural hematomas and acute infarcts

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ABSTRACT

Background: Spontaneous intracranial hypotension (SIH) is a rare but important condition characterized by cerebrospinal fluid (CSF) leakage, typically presenting with postural headaches. In severe cases, SIH can result in subdural hematomas (SDHs), brain herniation, and acute infarcts. The 2023 SIH guidelines recommend starting with nontargeted epidural blood patches as the initial treatment, with up to two attempts before considering a targeted patch. Nontargeted patches are effective in most cases by distributing blood widely in the epidural space. However, in rapidly deteriorating patients, targeted blood patches may be necessary, especially when a specific leak site has been identified. This report highlights the importance of timely, targeted interventions in severe SIH cases.

Case Description: A 34-year-old male presented with a 6-month history of worsening headaches, impaired mobility, and stupor. On admission, his Glasgow Coma Scale was 12, which rapidly deteriorated to 5. Imaging revealed bilateral chronic SDHs, cerebellar tonsillar herniation, and signs of intracranial hypotension despite no history of trauma or anticoagulant use. Initial surgical evacuation of the hematomas failed to improve the patient's neurological status. Subsequent magnetic resonance imaging and computed tomography myelogram identified a CSF leak at the T12 level. Given the patient's critical state and rapid neurological decline, we opted for a targeted epidural blood patch at the L1/2 level. This intervention led to significant clinical improvement, with follow-up imaging demonstrating a reduction in the subdural collections and resolution of the leak. The patient fully recovered and remained asymptomatic at a 6-month follow-up.

Conclusion: This case highlights the need for flexibility in SIH management, particularly in severe cases with acute neurological decline. While nontargeted blood patches are typically recommended, early use of a targeted patch when imaging identifies the leak can lead to faster resolution and improved outcomes. Personalized treatment strategies are essential for managing complex SIH presentations and preventing further neurological complications.

Keywords: Acute infarct, Blood patch, Cerebrospinal fluid leak, Spontaneous intracranial hypotension, Subdural hematoma

INTRODUCTION

Spontaneous intracranial hypotension (SIH) is a rare but important condition characterized by cerebrospinal fluid (CSF) hypovolemia, which can lead to debilitating symptoms such as postural

headaches, neurological deficits, and, in severe cases, acute neurological deterioration.[1-3,11]

The 2023 guidelines for the management of SIH recommend initial treatment with nontargeted epidural blood patches, which involve injecting blood into the epidural space at a lumbar level without specifically targeting the CSF leak site.[2,11] This approach is preferred due to its simplicity, safety, and high success rate of the first patch, with up to 64% of patients responding. The rationale behind nontargeted patches lies in their ability to distribute blood widely within the epidural space, potentially sealing leaks across multiple spinal levels, even when the exact location is not identified.[2,11] Targeted blood patches, however, where blood is injected precisely at the identified site of the CSF leak, are recommended when the leak can be localized through imaging modalities such as computed tomography (CT) myelography or magnetic resonance imaging (MRI).[2,11] This approach is more invasive and technically demanding and thus often reserved for patients who do not respond to nontargeted patches. The current guidelines for the management of SIH also recommend that nontargeted patches should be attempted twice before considering a targeted approach.[2,11]

Although chronic postural headaches are the typical presentation of SH, some cases may exhibit acute neurological deterioration, posing diagnostic challenges.[3,5] We describe a unique case of a young male who presented with a 6-month history of declining physical and mental health. The subsequent finding of subdural hematomas (SDHs), acute infarcts, and intracranial hypotension highlights the diagnostic complexity of SIH and our rationale to bypass a nontargeted blood patch and opt straight for a targeted patch instead.

CASE REPORT

A 34-year-old male presented with a six-month history of deteriorating physical and mental health. He complained of headaches, poor sleep, declining mobility, requiring a wheelchair, and needing a soft diet as he was unable to chew food. Over the past 2 months, he developed occipital headaches, ultimately becoming stuporous over the 5 days before his presentation. There was no history of head trauma or use of blood thinners/coagulation abnormalities. On admission, he had a Glasgow Coma Scale (GCS) score of 12 (E3V3M6) with hyperreflexia, which later declined to GCS 5 (E1V1M3) with associated rigidity and hypertension. The patient's pupils were bilaterally sluggish, reactive, and isochoric, and he was loaded with antiepileptics.

A CT scan of the head revealed bilateral chronic SDHs, with a right-sided hematoma measuring 13 mm and a left-sided hematoma measuring 4 mm, accompanied by a marked midline shift to the left. The patient underwent a right craniotomy due to septations and left-sided burr hole evacuation of the SDHs. The operating surgeon noted no brain expansion after the dural opening. Postoperatively, a CT scan with contrast and CT angiography showed no evidence of vascular abnormalities. New areas of low attenuation in the right parietal, temporal, and occipital lobes were seen, suggesting infarcts [Figure 1].

MRI with contrast was performed on the 2nd postoperative day to investigate the areas of low attenuation with a working diagnosis of lymphoma or encephalitis. The MRI revealed bilateral subdural collections, pneumocephalus, increased right SDH volume with mass effect, and midline shift to the left. Additional findings included diffusion restriction and high fluid-attenuated inversion recovery/T2 signal in the right hemipons, indicative of acute infarcts secondary to right uncal herniation. Smooth pachymeningeal thickening bilaterally, cerebellar tonsillar herniation, pituitary congestion, and flattening of pons were consistent with intracranial hypotension as the underlying cause of the SDHs [Figure 2].

The patient showed no evidence of sustained clinical improvement, with a GCS score fluctuating between E1V1M5 and E2V3M5, and there had been no chronological improvement in the mass effect or midline shift. He was kept intubated and ventilated and underwent an MRI of the whole spine, which showed a ventral longitudinal epidural collection extending from C2/3 to T2, with features consistent with a CSF leak, including paucity of CSF around cauda equina and dilatation of lumbar venous plexus [Figure 3]. He was managed initially with a flat bed rest.

A repeat CT scan of the head performed 4 days later for deteriorating motor score showed an ongoing 8 mm right SDH with a midline shift to the left and a 3 mm left SDH. A CT myelogram revealed an opening pressure of 8.5 cm H_2O and a suspicious area on the right lateral dural surface at T12, but without any obvious prominent disc or bony spurs. The delayed study showed contrast in renal pelves, confirming CSF leak.

Due to the rapidity of neurologic decline resulting in brainstem infarcts and lack of clinical improvement, the decision was made to proceed directly with a targeted blood patch. A targeted blood patch (20 mL) was performed at the L1/2 interspace. The region of suspected CSF leak was at T12; however, previous imaging had demonstrated that the conus was at T12, so L1/2 was deemed more appropriate. Under fluoroscopic guidance, an 18 G Tuohy needle was advanced to the L1/L2 epidural space. A small volume of contrast (2 mL of Omnipaque 300) was injected through the needle. This demonstrated "dirty" opacification of the spinal canal, which confirmed the epidural position [Figure 4]. This was followed by an injection of the blood patch (20 mL).

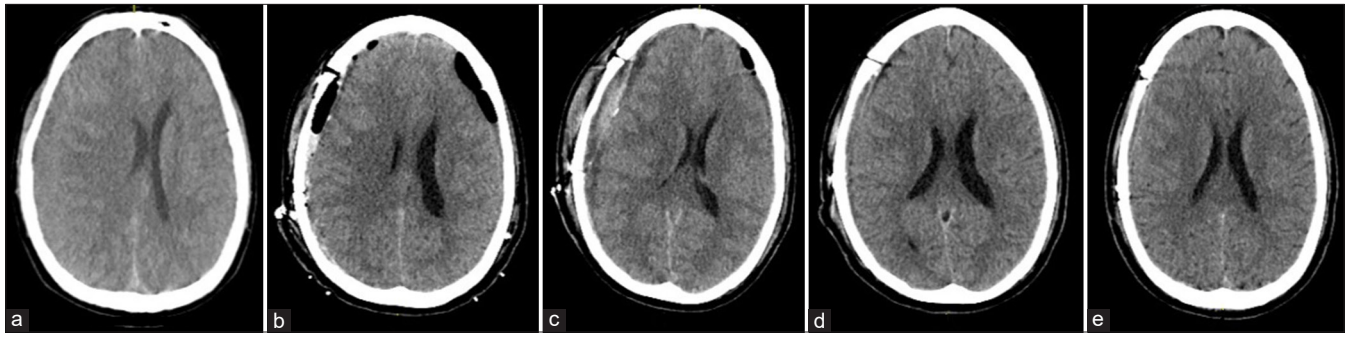

Figure 1: Computed tomography (CT) scans of the brain axial slices. (a) Initial scan showing bilateral subdural haematomas with marked midline shift to the left. (b) Day one postoperative CT scan showing adequate evacuation of the subdural haematomas. (c) Day 7 postoperative CT scan showing recurrent bilateral subdural collections. (d) CT scan 1 week and (e) CT scan 6 weeks after targeted blood patch.

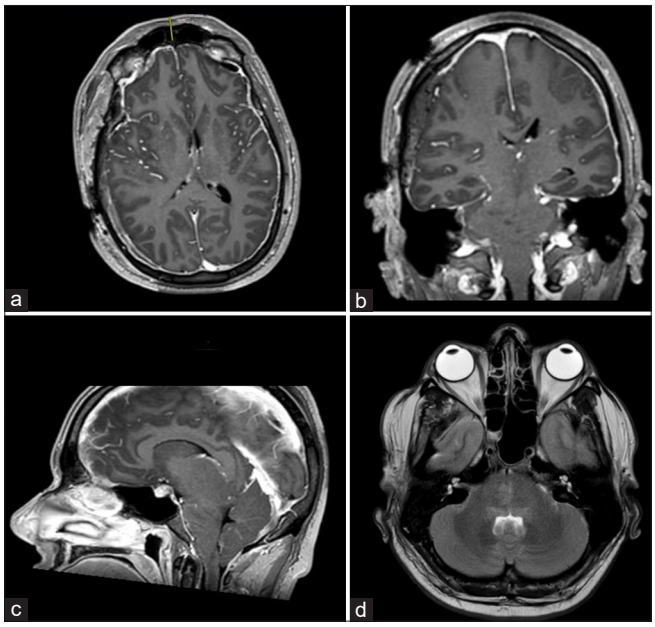

Figure 2: Magnetic Resonance Imaging (MRI) brain showing (a and b) pachymeningeal thickening bilaterally, (c) cerebellar tonsillar herniation, pituitary congestion, flattening of pons, and (d) high T2 signal in the right hemipons indicative of acute infarct.

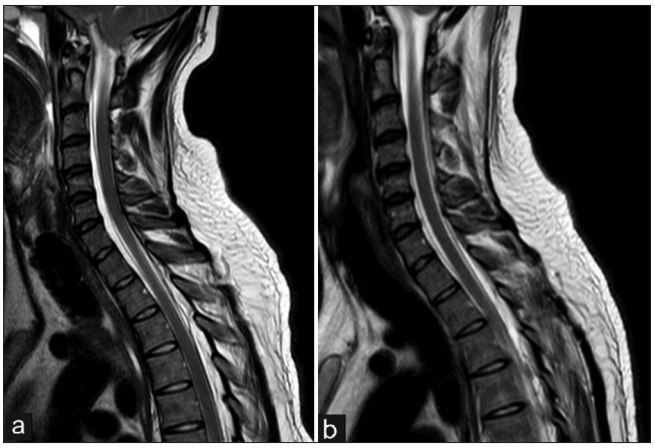

Figure 3: (a) Magnetic resonance imaging (MRI) spine showing ventral longitudinal epidural collection from C2/3 to T2. (b) MRI spine 1 week after the targeted blood patch showed reduced size of the ventral epidural collection.

The patient was kept on flatbed rest for the first 5 days after the blood patch procedure. CT scan of the head at this stage revealed a decrease in the size of the right SDH to 5 mm, with complete resolution on the left. MRI of the whole spine showed further reduction in the size of the ventral epidural collection. The patient's condition improved, reaching a GCS of 15, and he was discharged with physiotherapy and occupational therapy support. He reattended 2 weeks later for a repeat CT scan, which showed further reduction in the size of the right SDH, and the patient remained asymptomatic at the 6-month follow-up.

DISCUSSION

This case report presents a compelling clinical scenario involving a patient who initially experienced a 6-month history of declining physical and mental health, marked by headaches, poor sleep, mobility deterioration, and impaired chewing. His condition took a critical turn as he developed occipital headaches and rapidly deteriorated into a semi-comatose state, leading to emergency admission. The clinical sequelae underscore the complexities that can occur when dealing with a case of SIH.

The 2023 SIH guidelines suggest the use of nontargeted high-volume epidural blood patches while implementing conservative strategies as the first step.[2] However, our decision to proceed directly with a targeted blood patch rather than relying on the broader nontargeted patch was important for several reasons. First, the urgency of the clinical presentation was a factor, as the patient had a rapid neurological decline, which warranted timely intervention. Nontargeted patches, while effective in many cases, can take time to show results and may require multiple attempts. Second, we identified the leak site at the T12 level. When a specific leak site is known, a targeted blood

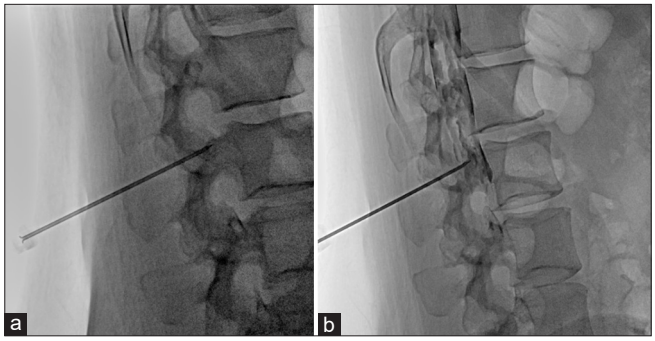

Figure 4: (a) Tuophy needle advanced to L1/2 epidural space. (b) Epidural position confirmed with contrast injection.

patch may have a higher success rate as it will address the leak directly, preventing ongoing CSF loss. The choice to perform a targeted patch in this case was supported by the localization of the leak. Finally, there may be potential risks in delaying treatment as nontargeted patches can lead to diffuse coverage of the epidural space, but in severe cases such as ours, where rapid deterioration is occurring, delaying focused intervention may increase the risk of complications such as worsening of subdural hematomas of herniation. The targeted approach provided an opportunity to halt the ongoing CSF leakage more efficiently, leading to quicker clinical improvement.

The surgical evacuation of SDHs before addressing the CSF leak may have contributed to the worsening clinical course, potentially due to further accumulation of air and an increased downward tractive force on the brain.[4] Thus, in cases where SIH is suspected, it is imperative to prioritize the identification and repair of the CSF leak before proceeding with surgical hematoma evacuation.[5] This may not always be possible if the hematoma is causing mass effects and neurological symptoms, and in such cases, surgical evacuation should be followed by flat bed rest and appropriate imaging to identify features of SIH.[8]

Typically, SIH is associated with a chronic course, where patients predominantly present with postural headaches that worsen over time.[2] However, as observed in our patient, SIH can lead to acute neurological deterioration.[3] The pathophysiology underlying this rapid decline is likely attributed to the development of bilateral subdural collections, which, when they become acute, can exert a significant impact on intracranial pressure and cerebral blood flow.[3,10,12] The sudden loss of a substantial volume of CSF can trigger a cascade of events, leading to decreased intracranial compliance and increased downward tractive forces on the brain, which can result in the development of pontine and other cerebral infarcts, as observed in our patient.[1]

Our case report aligns with the findings of Kim *et al*.[6] who noted that rapid neurologic decline is noted in 40% of patients, necessitating SDH drainage to prevent irreversible neurologic damage. They also noted that severe sagging and SDH thickness were strong indicators of clinical deterioration. Our case also supports the assertion by Kim *et al*. that the severity of SDH does not solely depend on hematoma thickness but rather a combination of radiological and clinical findings. However, while Kim *et al*. found SDH drainage alone to be sufficient for sustained improvement in most deteriorated patients, this was not evident in our case. In addition, our approach differed in using flat bed rest postprocedure, whereas Kim *et al*. utilized the Trendelenburg position to promote CSF redistribution.[7]

Our case highlights the importance of considering SIH in younger patients presenting with bilateral chronic SDHs in the absence of a history of trauma or coagulation disorders.[4] SIH may be overlooked when evaluating patients with SDHs, notably if the patient lacks the typical orthostatic headache presentation.[3,4] Failure of the brain to expand following surgical evacuation of SDHs, as observed in our patient, should raise suspicion of underlying CSF leakage and intracranial hypotension.[9]

CONCLUSION

Whilst nontargeted blood patches remain the recommended initial strategy for managing SIH, this case underscores the importance of recognizing suitable candidates for early targeted interventions. Patients presenting with rapid neurological decline, imaging-confirmed CSF leaks, and poor response to initial management may benefit from prioritizing the use of advanced imaging and targeted blood patches to achieve faster resolution and improved outcomes.

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