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Case Report

Radiology Section

Spontaneous Intracranial Hypotension with Bilateral Subdural Haematomas and Posterior Cerebral Artery Infarcts with Delayed Epidural Blood Patch Response: A Case Report

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ABSTRACT

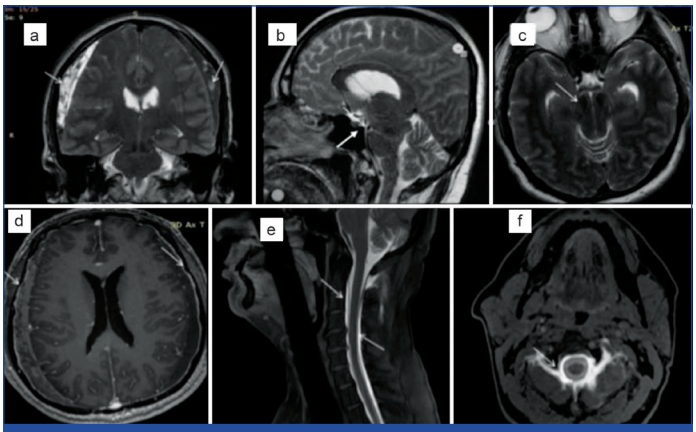
Spontaneous Intracranial Hypotension (SIH) is a rare but treatable cause of orthostatic headache due to spontaneous leakage of Cerebrospinal Fluid (CSF). A 48-year-old male presented with orthostatic headache with bilateral subdural haematomas. The patient was found to have bilateral subdural haematomas with mass effect on Brain Magnetic Resonance Imaging (MRI), and a cervical dural tear with active CSF leakage on contrast-enhanced MR myelography, suggestive of SIH. The late diagnosis was complicated by progressive mass effect and infarcts involving the bilateral posterior cerebral arteries. A delayed Epidural Blood Patch (EBP) had favourable clinical and radiological outcomes. Early diagnosis and prompt EBP were needed to avoid neurological sequelae.

Keywords: Cerebral arteries, Cerebrospinal fluid leak, Dural tear, Neurological sequelae, Orthostatic headache

CASE REPORT

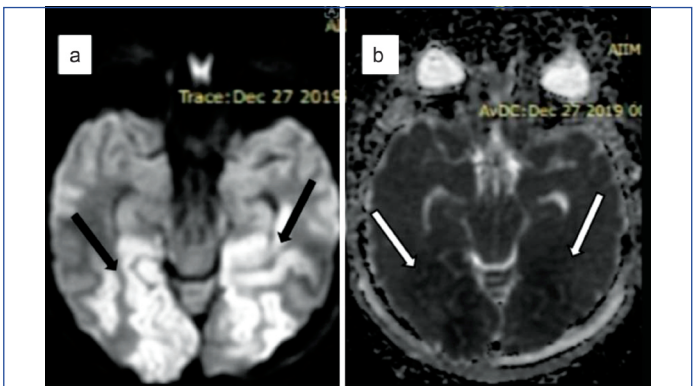
A 48-year-old male presented with a history of persistent severe headache for five weeks. The severity of headache was rated 8/10 on the Numeric Rating Scale (NRS) at presentation. The headache was predominantly orthostatic, worsening on sitting or standing and relieved on lying down. There was no history of head trauma, lumbar puncture, spinal surgery, or known connective tissue disorder. On neurological examination, the patient was conscious and oriented, with normal motor strength, intact deep tendon reflexes, and no focal neurological deficits.

The Brain MRI bilateral subdural haematomas of varying ages with associated mass effect. Additional imaging findings included diffuse pachymeningeal enhancement, anteroposterior elongation of the midbrain, effacement of the perimesencephalic cisterns, reduced pontomesencephalic angle, and decreased mammillopontine distance, consistent with intracranial hypotension. MRI of the spine demonstrated spinal longitudinal extradural collections in the cervical region. Contrast-enhanced MR myelography clearly demonstrated a cervical dural tear with active CSF leakage, confirming the diagnosis of SIH [Table/Fig-1].



[Table/Fig-1]: a) Coronal T2-weighted image showing bilateral subdural haemorrhages (white arrows) of different ages; (b) Sagittal T2-weighted image demonstrating effacement of the CSF space between the midbrain and pons (white arrow), with decreased pontomesencephalic angle and mammillopontine distance; (c) Axial T2-weighted image showing anteroposterior elongation of the midbrain with effacement of the perimesencephalic cistern (white arrow); (d) Axial post-contrast T1-weighted image showing diffuse, smooth dural enhancement with bilateral subdural haemorrhages (white arrows); (e) Sagittal spinal T2-weighted image demonstrating spinal longitudinal extradural collections in the cervical spine (white arrow); (f) Contrast MR myelography showing contrast leakage at the cervical spine level (white arrow).

An EBP was advised as definitive management; however, the patient initially declined the procedure and was managed conservatively. The patient was initially managed conservatively with bed rest, adequate oral hydration, caffeine intake, and analgesics during hospital admission. During the observation period, the patient's headache progressively worsened. Follow-up imaging demonstrated increasing mass effect from the bilateral subdural haematomas, along with the development of bilateral posterior cerebral artery territory infarcts. These infarcts showed diffusion restriction on diffusion-weighted imaging with corresponding low apparent diffusion coefficient values, consistent with acute ischemia and likely secondary to severe brain sagging and posterior circulation compromise [Table/Fig-2].

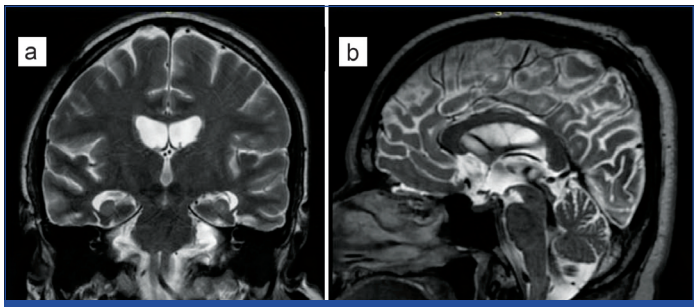


[Table/Fig-2]: Diffusion-weighted imaging showing (a) diffusion restriction in the bilateral posterior cerebral artery (PCA) territory (Black arrows) , (b) corresponding low signal on apparent diffusion coefficient (ADC) maps (White arrows).

In view of the increasing mass effect, the patient underwent bilateral burr-hole drainage of the subdural haematomas. Postoperatively, there was only minimal reduction in haematoma volume, with no

significant improvement in headache, indicating persistence of the underlying low CSF pressure state.

Given persistent symptoms and imaging evidence of an ongoing CSF leak, the patient subsequently consented to EBP. The EBP was performed as a blind lumbar epidural procedure under strict aseptic precautions. Approximately, 15-20 mL of autologous blood was injected into the epidural space. The patient was monitored closely following the procedure and was advised strict supine rest for several hours post-intervention. Following the procedure, the patient experienced dramatic relief of headache. Follow-up MRI after 72 hours demonstrated marked radiological improvement, including complete resolution of bilateral subdural haematomas, normalisation of the pontomesencephalic angle and mamillopontine distance, and disappearance of pachymeningeal enhancement [Table/Fig-3]. The CSF leak was no longer visualised. The patient remained clinically stable throughout hospitalisation and was discharged after eight days. No recurrence of symptoms was noted during the 3-month follow-up. The timeline of events from patient presentation to discharge are summarised in [Table/Fig-4].



[Table/Fig-3]: (a) Coronal T2-weighted image showing complete resolution of bilateral subdural haematomas; (b) Sagittal T2-weighted image demonstrating normalisation of the pontomesencephalic angle and mammillopontine distance.

Timeline	Clinical course/Intervention
Day 0	Patient presented with orthostatic headache.
Day 1-7	Managed conservatively with supportive treatment.
Day 8	Clinical deterioration with development of new cerebral infarcts on imaging.
Day 9	Bilateral burr-hole drainage performed due to worsening neurological status.
Day 20	Epidural Blood Patch (EBP) administered because of persistent symptoms despite surgical intervention.
90-Day follow-up	Significant clinical recovery observed, accompanied by marked radiological improvement.

[Table/Fig-4]: Summary of timeline.

DISCUSSION

Spontaneous CSF leakage was most often secondary to spinal dural defects. It typically presents with orthostatic headache, characterised by worsening pain on standing and relief on recumbency. Agarwal P et al., described two similar cases with CSF leak and similar symptoms [1]. In case one, a 25-year-old man presented with a three-week history of severe orthostatic headache. MRI demonstrated bilateral subdural hygromas, diffuse pachymeningeal enhancement, brain sagging, and a thoracic (T6) CSF leak. He responded completely to conservative management with bed rest and hydration, with sustained clinical and radiological improvement on follow-up. In case two, a 50-year-old man presented with orthostatic headache, progressive cognitive decline, urinary incontinence, and worsening encephalopathy. MRI showed features consistent with SIH without an identifiable CSF leak. Following failure of conservative treatment, two EBPs resulted in rapid neurological recovery and complete resolution of symptoms [1].

Characteristic Magnetic Resonance Imaging (MRI) features include diffuse pachymeningeal enhancement, venous sinus engorgement, descent of the brain (brain sagging), and, in some cases, subdural haematomas [2].

Subdural haematomas associated with SIH are believed to occur due to stretching and rupture of bridging veins in the setting of low CSF pressure and downward displacement of intracranial structures [3]. While many cases improve with conservative measures or EBP, severe or prolonged intracranial hypotension may rarely lead to ischemic infarctions, particularly involving the posterior circulation, due to vascular stretching and compromised cerebral perfusion [4]. EBP remains the treatment of choice for SIH, as it addresses the underlying CSF leak and restores normal CSF dynamics.

One such case was described by Agarwal P et al., which was managed conservatively, while another patient required EBP as his symptoms worsened [1].

Another study published by Kim YS et al., described 15 cases of Subdural Haematoma (SDH) and SIH. Initial management consisted of conservative treatment in 4 (26.7%) patients, EBP in 3 (20%) patients, and burr-hole drainage with subsequent Trendelenburg positioning in 8 (53.3%) patients. Clinical improvement was achieved in seven patients without the need for additional EBP, whereas one patient subsequently required EBP [5].

Mohamad Ebrahim Ketabforoush AH et al., reported another case of SIH in which they concluded that management often requires a combination of clinical evaluation, imaging, and other diagnostic procedures [6].

EBP remains the cornerstone of treatment for SIH, as it seals the dural defect, restores CSF volume, and reverses intracranial hypotension. Even when performed late in the disease course, as in the present case, EBP can result in dramatic clinical and radiological improvement [7]. The complete resolution of headache, disappearance of imaging features of intracranial hypotension, and closure of the CSF leak following EBP emphasise its pivotal therapeutic role [8].

Differential diagnoses for SIH include post-dural puncture headache, chronic subdural haematoma, idiopathic intracranial hypertension, cerebral venous sinus thrombosis, posterior reversible encephalopathy syndrome, Chiari I malformation, and meningitis. SIH is associated with orthostatic headaches along with specific imaging features, such as diffuse meningeal enhancement and brain slumping [9]. Post-dural puncture headache is similar to SIH, except for a documented history of lumbar puncture or spinal anaesthesia [4]. A diagnosis of chronic subdural haematoma does not include orthostatic headache or specific MRI features of SIH [10]. Idiopathic intracranial hypertension is associated with supine worsening of headaches, papilloedema, and increased CSF opening pressure [11]. Cerebral venous sinus thrombosis can be confirmed through MR venography showing venous sinus thrombosis [12]. Posterior reversible encephalopathy syndrome can be defined by parieto-occipital vasogenic oedema seen in MRI [13]. A diagnosis of Chiari I malformation is defined by congenital cerebellar tonsillar hernia without a CSF leak [14]. Meningitis is associated with fever and abnormal CSF features, as well as leptomeningeal rather than pachymeningeal enhancement [15].

CONCLUSION(S)

The SIH, though rare, can lead to severe and potentially irreversible neurological complications if not treated promptly. This case emphasises the importance of early recognition and timely EBP to prevent secondary complications such as subdural haematomas and posterior circulation infarcts. Addressing the underlying CSF leak is essential for optimal clinical and radiological recovery.

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