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AN UNUSUAL CASE OF SPONTANEOUS INTRACRANIAL HYPOTENSION PRESENTING AS MENINGOENCEPHALITIS

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ABSTRACT

Background: Spontaneous intracranial hypotension (SIH) is a rare, underdiagnosed cause of secondary headache due to cerebrospinal fluid (CSF) leakage. While typically presenting with orthostatic headache, atypical manifestations may mimic infectious or inflammatory diseases.

Case description: A 45-year-old man presented with acute alteration of consciousness, expressive aphasia, and nuchal rigidity after 1 month of orthostatic occipital headache. Brain magnetic resonance imaging (MRI) showed diffuse pachy- and leptomeningeal enhancement initially interpreted as infectious meningoencephalitis. Lumbar puncture revealed turbid, blood-stained CSF with elevated protein and neutrophilic pleocytosis, but microbiological and autoimmune studies were negative. Neurological symptoms improved with conservative management (bed rest, hydration, analgesia), and follow-up imaging confirmed full resolution. Retrospective neuroradiological review reclassified findings as SIH.

Conclusion: SIH can mimic infectious meningoencephalitis both clinically and in CSF appearance. Recognition of preceding orthostatic headache and characteristic MRI features is essential to avoid unnecessary antimicrobial therapy and invasive diagnostic procedures.

KEYWORDS

Spontaneous intracranial hypotension, cerebrospinal fluid leak, orthostatic headache, meningeal enhancement, meningoencephalitis

LEARNING POINTS

- Spontaneous intracranial hypotension may present atypically, mimicking infectious meningoencephalitis.
- In any acute neurological presentation, a preceding history of orthostatic headache should prompt reconsideration of spontaneous intracranial hypotension before performing lumbar puncture.
- Magnetic resonance imaging meningeal enhancement is nonspecific; careful correlation with clinical features and exclusion of infectious or autoimmune causes are essential for accurate diagnosis

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INTRODUCTION

Spontaneous intracranial hypotension (SIH) is an uncommon but increasingly recognized disorder caused by spontaneous cerebrospinal fluid (CSF) leakage, most often at the spinal level, resulting in reduced CSF volume and pressure. Its estimated incidence is approximately 5 per 100,000 persons per year, with a clear female predominance and peak occurrence in middle age[1]. The typical clinical picture is dominated by orthostatic headache, often accompanied by nausea, neck pain, and occasionally cranial nerve palsies or cognitive changes[2,3]. However, atypical or non-orthostatic presentations are being reported with growing frequency and may closely mimic other neurological conditions, including infectious or inflammatory meningoencephalitis[4]. Early recognition of SIH is crucial to prevent misdiagnosis, avoid unnecessary antimicrobial or immunosuppressive therapy, and enable timely, conservative management.

CASE DESCRIPTION

A 45-year-old man was admitted to the Internal Medicine ward with a 1-month history of occipital headache and cervicalgia that worsened within 5 minutes of standing and resolved rapidly when supine. He was initially managed conservatively with nonsteroidal anti-inflammatory drugs by his primary physician and orthopaedist, with only partial improvement. Cervical computed tomography (CT) scan showed a vertebral haemangioma at C2 and degenerative disc disease at C5–C7. He also reported a short, self-limited flu-like illness 3 days before admission.

During dinner, he developed sudden alteration of consciousness without involuntary movements, followed by transient language disturbance. At the emergency department, a possible seizure was considered and levetiracetam was initiated. There was no history of trauma, surgery, medication changes, travel, fever, or systemic symptoms.

Vital signs on admission were temperature 36.8°C, blood pressure 132/78 mmHg, heart rate 84 bpm, oxygen saturation 98% on room air. On examination, he exhibited moderate attention deficits, echolalia, impaired naming and repetition, and inconsistent command following. Pupils were equal and reactive; ocular movements were full; facial symmetry and limb strength were preserved; plantar responses were flexor. Nuchal rigidity was present without other meningeal signs or abnormal movements. Laboratory tests revealed mild leucocytosis (12.8 $\times 10^9$/l, 86.6% neutrophils) and elevated C-reactive protein (6.24 mg/dl), with normal renal and hepatic function.

Head CT scan revealed thin bilateral frontal subdural effusions and reduced sulcal and cisternal spaces, suggesting intracranial hypertension without focal parenchymal lesions or haemorrhage *(Fig. 1)*. Brain magnetic resonance imaging (MRI) confirmed bilateral frontoparietal and pericerebellar subdural collections isointense to CSF, diffuse pachymeningeal and leptomeningeal enhancement, and mild effacement of cortical sulci and basal cisterns without

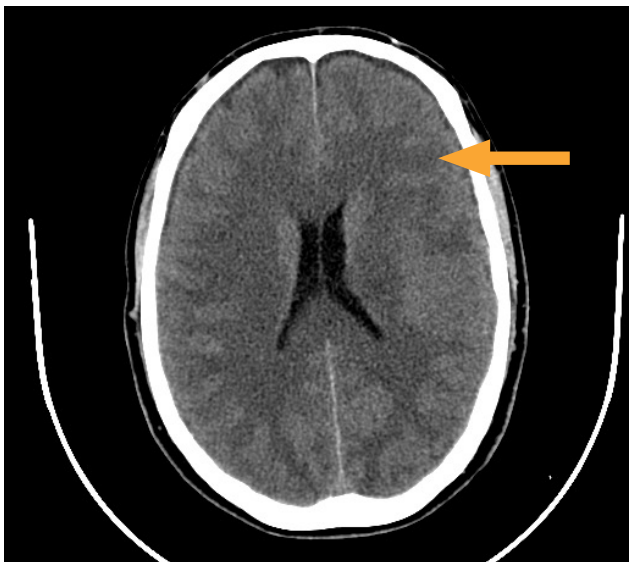


Figure 1. Admission head computed tomography scan demonstrated thin bilateral subdural effusions and sulcal effacement suggestive of reduced intracranial cerebrospinal fluid volume (arrow).

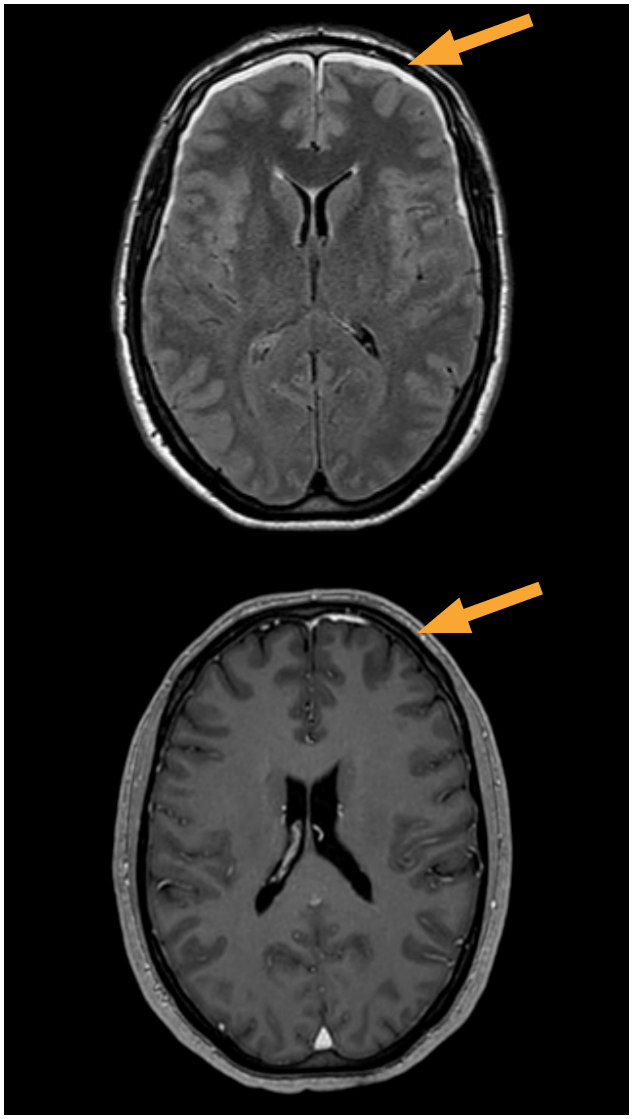


Figure 2. Admission magnetic resonance imaging showing diffuse pachymeningeal and leptomeningeal enhancement, thin bilateral frontoparietal subdural effusions, and mild effacement of cortical sulci (arrows).

venous thrombosis or parenchymal abnormalities *(Fig. 2)*. The initial interpretation favoured infectious meningitis due to the leptomeningeal component. Lumbar puncture (LP) was performed after neuroimaging excluded mass effect or haemorrhage, in accordance with standard diagnostic protocols for suspected central nervous system infection. LP revealed turbid, blood-stained CSF with elevated protein (325 mg/dl), normal glucose (71 mg/dl), and polymorphonuclear pleocytosis (108 cells/µl). Given the acute encephalopathic presentation, nuchal rigidity, and inflammatory CSF profile despite the absence of fever, infectious meningoencephalitis was initially suspected, and empirical antibiotic therapy was initiated while awaiting microbiological results. Stroke or subarachnoid haemorrhage were considered but ruled out by neuroimaging (no vascular-territory lesions or subarachnoid blood). The working diagnosis of infectious meningoencephalitis was therefore most coherent at presentation.

Multiplex polymerase chain reaction (PCR), bacterial and fungal cultures, viral PCR (including herpes simplex and severe acute respiratory syndrome coronavirus 2), *Mycobacterium tuberculosis* PCR, cytology, paraneoplastic antibodies, oligoclonal bands, and extensive serological and autoimmune testing were all negative. Electroencephalogram showed no epileptiform activity. Empirical intravenous ceftriaxone was discontinued after seven days once infection was excluded.

A neuroradiology review later considered the pattern—combined pachymeningeal enhancement, subdural effusions, venous engorgement, and mild "brain sagging"—as more consistent with SIH, particularly given the orthostatic nature of previous symptoms.

The patient improved progressively with conservative treatment (bed rest, hydration, and analgesia) and was discharged asymptomatic after one week.

Follow-up MRI performed 1 month later showed complete resolution of the subdural collections and pachymeningeal enhancement, with re-expansion of the basal cisterns and slight reduction in pituitary size *(Fig. 3)*. The patient remained well and seizure-free at follow-up, allowing gradual levetiracetam tapering. Epidural blood patch was not required.

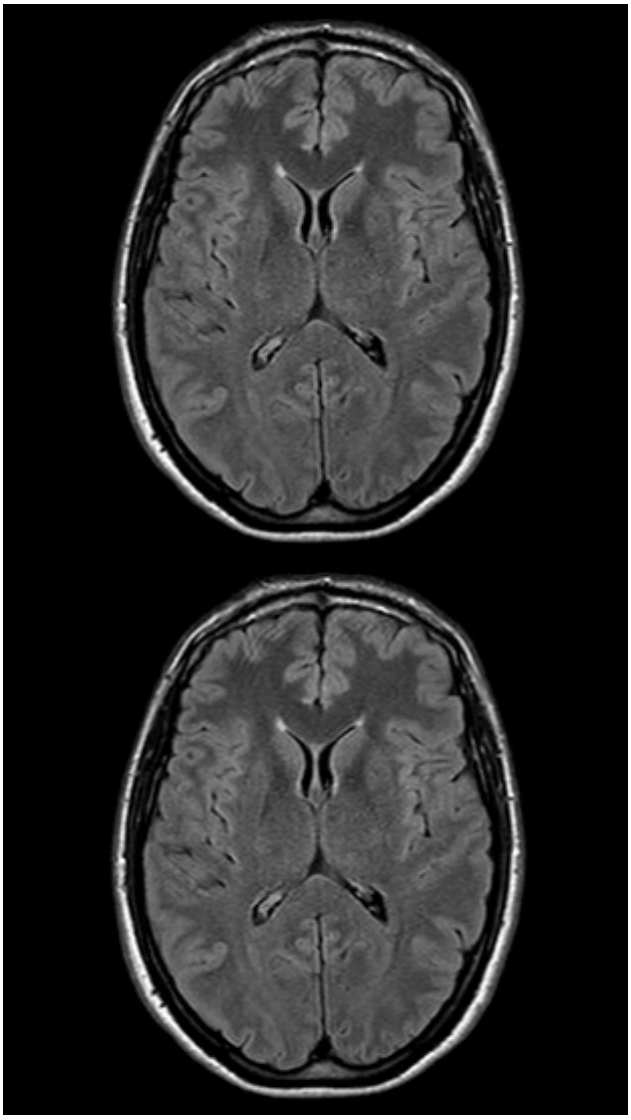


Figure 3. Follow-up magnetic resonance imaging 1 month later demonstrating complete resolution of subdural collections and meningeal enhancement.

DISCUSSION

SIH is a rare but increasingly recognized disorder caused by spontaneous CSF leakage, most commonly at the spinal level. The ensuing reduction in CSF pressure and volume leads to downward displacement of intracranial structures, traction on pain-sensitive meninges, and compensatory venous engorgement[1,2]. Its estimated incidence is around 5 per 100,000 persons per year, with a female predominance and peak onset in fourth decade of life[2].

The classic presentation is an orthostatic headache, occipital or suboccipital, that worsens within minutes of standing and improves when supine[1,3]. Associated symptoms include nausea, neck pain, diplopia, cranial nerve palsies—particularly sixth nerve palsy—and, less frequently, cognitive or behavioural changes[1,3]. However, up to one third of patients may lack the typical orthostatic pattern, especially in advanced stages, leading to diagnostic confusion with meningoencephalitis, subarachnoid haemorrhage, or cerebral venous thrombosis[1,4,5]. In our case, the constellation of acute encephalopathy, language disturbance, and nuchal rigidity with an inflammatory CSF profile suggested infection and justified empirical antibiotic therapy. A vascular event such as stroke was considered less likely because there were no focal motor deficits, cranial nerve palsies, or vascular territory pattern on brain imaging, and both head CT scan and MRI excluded acute ischemia or haemorrhage. Similarly, a subarachnoid bleed was deemed improbable given the absence of severe sudden onset "thunderclap" headache and normal CT scan without subarachnoid blood.

LP was also performed because infectious meningoencephalitis was initially considered the leading diagnosis. The CSF was turbid and blood-stained with markedly elevated protein and neutrophilic pleocytosis—features atypical for SIH but described in isolated reports[6,7].

Although low CSF opening pressure (<60 mmH_2O) is considered characteristic, it is inconsistently observed; up to one third of patients may have normal or even elevated pressures[1,3]. Therefore, CSF parameters must be interpreted within the clinical and radiological context rather than in isolation. In retrospect, lumbar puncture, while diagnostically valuable at the time, could transiently worsen SIH and should be avoided once the diagnosis is suspected[4,5].

Neuroimaging plays a crucial role in the diagnosis of SIH. Typical findings include diffuse pachymeningeal enhancement, subdural collections, venous sinus engorgement, pituitary enlargement, and "brain sagging" with descent of the cerebellar tonsils and reduction of the pontomesencephalic angle[3,8]. In our patient, brain MRI demonstrated bilateral subdural effusions, diffuse pachy- and leptomeningeal enhancement, and mild effacement of cortical sulci and basal cisterns—initially interpreted as infectious involvement due to the leptomeningeal component. However, the absence of parenchymal lesions, cerebral venous thrombosis, or true meningeal infiltration, together with the orthostatic nature of the headache and the spontaneous clinical improvement with conservative measures, favoured the diagnosis of SIH. Follow-up MRI one month later confirmed complete resolution of the subdural collections and meningeal enhancement, with re-expansion of basal cisterns and partial normalization of pituitary size, consistent with radiological recovery.

The underlying cause of spontaneous CSF leaks is often unidentified, although dural weakness or minor trauma may predispose to rupture of spinal meningeal diverticula[5]. In our patient, degenerative cervical spine changes could have contributed to local dural fragility and predisposed to spontaneous leakage.

The differential diagnosis of diffuse meningeal enhancement is broad and includes bacterial, viral, or autoimmune meningoencephalitis, inflammatory disorders, neoplastic meningitis, and subarachnoid haemorrhage[4]. As illustrated here, extensive microbiological and autoimmune testing is essential to exclude these entities, but careful correlation with imaging and clinical evolution remains decisive. The prompt reassessment of MRI by an experienced neuroradiologist was key to avoiding unnecessary antimicrobial or immunosuppressive therapy.

Management of SIH follows a stepwise approach. Conservative measures—bed rest, hydration, caffeine, and analgesia—are first-line and effective in many cases[3,9]. When symptoms persist or are disabling, an epidural blood patch is the treatment of choice, achieving success rates exceeding 70%[9,10]. In refractory cases, targeted leak repair or surgical closure may be required[9]. Early recognition is crucial to prevent complications such as subdural haematoma, superficial siderosis, or persistent cognitive impairment[2,9].

This case highlights a rare and diagnostically challenging presentation of SIH with encephalopathy, meningeal signs, and inflammatory CSF findings. The overlapping clinical and radiological features with infectious meningoencephalitis emphasize the importance of maintaining a high index of suspicion. Multimodal assessment—combining clinical history, CSF analysis, and expert neuroradiological review—is essential for accurate diagnosis. Greater awareness among internists and neurologists can facilitate early identification, guide appropriate conservative management, and prevent unnecessary interventions. This report contributes to the limited literature on atypical presentations of SIH and underscores that, in patients presenting with acute encephalopathy and meningeal signs, a careful search for preceding orthostatic headaches and characteristic MRI features is key to distinguish SIH from infectious meningoencephalitis and avoiding unnecessary lumbar puncture or antimicrobial therapy.

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