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Spontaneous Intracranial Hypotension in a Postpartum Woman

Jelena Sulić[1,2], Nina Mihić[1], Mario Kordić[1], Nikolina Penava[1], Antonio Sesar[1,2], Ivan Ćavar[1,2]

[1]University Clinical Hospital Mostar, Bosnia and Herzegovina; [2]School of Medicine, University of Mostar, Bosnia and Herzegovina

ABSTRACT

Spontaneous intracranial hypotension (SIH) is a clinical syndrome caused by spontaneous cerebrospinal fluid (CSF) leakage, most commonly due to a defect in the spinal dura mater, occurring in the absence of preceding trauma, invasive procedures, or lumbar puncture. The hallmark manifestation is orthostatic headache, characterized by worsening in the upright position and relief upon recumbency. We report the case of a 31-year-old woman who developed symptoms three months after an induced vaginal delivery at 40 weeks of gestation. She presented with a progressively worsening headache exhibiting marked postural dependence, becoming severe upon standing and partially relieved in the supine position. The pain was refractory to oral and intravenous analgesics and was accompanied by photophobia, phonophobia, nausea, repeated vomiting, and neck stiffness, while neurological examination revealed no focal deficits. Initial cranial computed tomography was unremarkable. Brain magnetic resonance imaging (MRI), including MR angiography and MR venography, demonstrated diffuse pachymeningeal thickening, small subdural effusions in the occipital and infratentorial regions, and mild venous sinus engorgement. Cervical spine MRI revealed anterior dural effusion extending from C1 to C7, supporting the diagnosis of spinal CSF hypovolemia. The patient was treated conservatively with bed rest, hydration, and caffeine, resulting in gradual clinical improvement without the need for epidural blood patching. This case highlights the importance of recognizing orthostatic headache as a key diagnostic clue, particularly in the postpartum period. Early MRI evaluation is essential, as timely identification of SIH enables appropriate management and may prevent potentially serious complications.

Key words: spontaneous intracranial hypotension, orthostatic headache, cerebrospinal fluid leak, magnetic resonance imaging

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ORCID IDs of the authors:
J.S. 0000-0002-9427-3215
M.K. 0009-0006-7631-4286
N.P. 0000-0003-3765-3120
A.S. 0000-0003-3176-6052
I.Ć. 0000-0002-0685-3982

Corresponding author:
Jelena Sulić
E-mail: jelena.skoko5@gmail.com

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INTRODUCTION

Spontaneous intracranial hypotension (SIH) is a disorder caused by spontaneous cerebrospinal fluid (CSF) loss, most frequently resulting from a spinal dural defect, and less commonly from meningeal diverticula or CSF–venous fistulas, in the absence of trauma, neurosurgical intervention, or lumbar puncture (1, 2). The condition was first described in 1938 by Schaltenbrand under the term "hypoliquorrhea" (3). The estimated annual incidence is approximately 3–5 per 100,000 individuals, although the condition is likely underdiagnosed (4). SIH predominantly affects women, with a female-to-male ratio of 2:1. Loss of CSF results in reduced intracranial pressure and volume, compromising the brain's normal buoyancy and allowing caudal displacement of intracranial structures, a phenomenon known as »brain sagging«. The resulting traction on pain-sensitive tissues, including the dura mater, cranial nerves, and intracranial vessels, underlies the characteristic neurological manifestations. Predisposing factors include underlying connective tissue disorders, spinal meningeal cysts, and structural abnormalities such as spinal osteophytes (1, 2).

Clinically, the presentation ranges from mild headache to severe neurological impairment, including cognitive deficits, dementia, and even coma (4). Orthostatic headache is the hallmark symptom of SIH, typically worsening in the upright position and improving when lying down. Pain intensity often shows strong postural dependence, similar to headaches following lumbar puncture, although the degree of positional variation may differ between patients. Headaches frequently intensify as the day progresses, particularly in the afternoon or evening (5). Symptom onset may be triggered by activities that markedly increase intra-abdominal pressure, such as vigorous coughing, sneezing, or sudden twisting and stretching movements. Headache may also be accompanied by auditory disturbances, most commonly tinnitus or a sense of muffled hearing (6). Subdural hematoma is the most frequent and potentially life-threatening complication of SIH, occurring in 20–25% of cases, while venous sinus thrombosis and superficial siderosis are much less common (7, 8).

Low CSF pressure is not mandatory for the diagnosis of SIH, as patients may exhibit normal or even elevated values (1). Brain magnetic resonance imaging (MRI) represents the cornerstone of evaluation and plays a crucial role in establishing the diagnosis. Characteristic findings include diffuse pachymeningeal enhancement, engorged venous sinuses, pituitary enlargement, brain sagging as evidenced by effacement of the suprasellar and prepontine cisterns and decreased mamillopontine distance, and the presence of subdural hygroma or hematoma (9).

Management of SIH is guided by the severity and persistence of symptoms. Initial conservative measures, applied over several days to weeks, may include bed rest, increased oral fluid intake, caffeine, and use of an abdominal binder. For patients who do not respond to conservative therapy, epidural blood patching is the primary intervention. Autologous blood is injected into the lumbar epidural space, producing symptomatic relief that may occur immediately due to transient increases in CSF pressure, while long-term resolution is likely achieved through fibrin clot formation at the dural tear (1).

Special clinical circumstances may further obscure the diagnosis and delay appropriate treatment. We report the case of a 31-year-old postpartum woman with SIH and characteristic radiological findings.

CASE REPORT

A 31-year-old woman delivered a healthy baby girl at 40 weeks of gestation following induced labor. This was her second pregnancy, and she had no significant past medical history. The only notable findings were mild thoracic

scoliosis and mild joint laxity, without associated syndromic features. She had no prior history of headaches.

Three months postpartum, she awoke one morning with a headache, which she described as a mild tightening sensation. As the day progressed, the headache intensified. Despite oral analgesic therapy with 800 mg of ibuprofen and 1 g of paracetamol, the pain did not subside. By the end of the day, the headache had become more severe, particularly in the upright position, while it partially improved when lying down. The following day, the pain persisted, although it was milder in the morning. The patient presented to the Emergency Department of the University Hospital Center Mostar, Bosnia and Herzegovina, where laboratory findings were within normal limits. She received intravenous analgesic therapy (diclofenac and orphenadrine ampoule (75mg + 30mg)/250ml) without relief and was discharged with a diagnosis of headache. On the third day, her symptoms worsened. She developed mild photophobia and phonophobia, slight blurred vision, and neck stiffness, and she vomited several times. She returned to the Emergency Department, where an urgent head MSCT (GE Lightspeed VCT 64, General Electric, NY, USA) scan was performed and found to be normal. Neurological examination revealed no focal deficits.

Subsequently, brain MRI with MR angiography (MRA) and MR venography (MRV) was performed (3T Skyra, Siemens Healthineers, Erlangen, Germany). Imaging demonstrated diffuse pachymeningeal thickening with small subdural effusions in the left occipital and infratentorial regions (up to 4 mm), accompanied by mild venous sinus engorgement. The pituitary gland was mildly enlarged, consistent with physiological lactation-related changes. The cerebellar tonsils were in a normal position, with no evidence of herniation. These findings were compatible with mild intracranial hypotension. No signs of ischemia, hemorrhage, demyelination, or mass lesion were observed. MRA showed normal intracranial arterial morphology without stenosis, aneurysm, or arteriovenous malformation. MRV confirmed normal venous sinus patency without evidence of thrombosis (Figure 1).

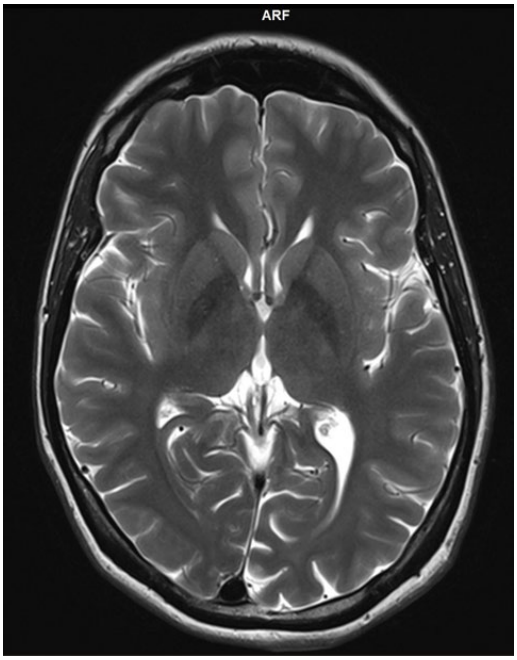

Figure 1. Axial postgadolinium brain MRI demonstrates bilateral diffuse pachymeningeal enhancement accompanied by dural thickening.

MRI of the cervical spine revealed an anterior laminar epidural fluid collection extending from C1 to C7, consistent with spinal CSF hypotension. The spinal cord appeared normal, without demyelinating changes or myelopathy. A right paracentral and foraminal disc protrusion at C5–C6 caused mild compression of the spinal cord and the right C6 nerve root, while a central protrusion at C6–C7 reduced the anterior subarachnoid space without significant compression. No inflammatory or neoplastic lesions were identified (Figure 2).

After the diagnosis of SIH was established, conservative management was recommended, including bed rest, increased fluid intake, and caffeine consumption. An epidural blood patch or other invasive procedures were not required. Her condition stabilized over the following 10

days. Mild residual neck stiffness persisted for several additional days.

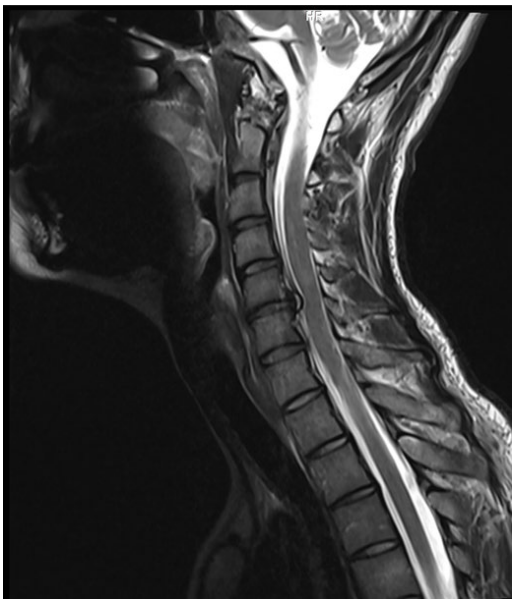

Figure 2. Cervical spine MRI shows an anterior lamellar dural effusion extending from C1 to C7.

DISCUSSION

SIH arises from spontaneous CSF loss, typically due to a spinal dural defect in the absence of prior trauma or medical intervention (1). In our patient, cervical spine MRI demonstrated an anterior epidural fluid collection extending from C1 to C7, supporting the presence of spinal CSF hypovolemia. Mild thoracic scoliosis and joint laxity, although not syndromic, may suggest subtle connective tissue vulnerability, which has been recognized as a predisposing factor for spontaneous dural defects (1, 2).

In response to CSF hypovolemia, compensatory mechanisms are activated to preserve intracranial volume homeostasis. Venous engorgement, including distension of the dural venous sinuses, represents a key adaptive response and contributes to characteristic radiological findings such as diffuse pachymeningeal thickening and subdural fluid collections (2,10). These mechanisms were clearly reflected in our patient's brain MRI, which demonstrated pachymeningeal enhancement, mild venous sinus engorgement, and small occipital and infratentorial subdural effusions. The absence of cerebellar tonsillar herniation or marked brainstem descent in this case suggests an early or relatively mild stage of intracranial hypotension.

The hallmark orthostatic headache, which was prominent in our patient and showed clear worsening in the upright position with partial relief when supine, reflects reduced brain buoyancy and traction on the dura mater and intracranial vessels. Associated symptoms, including nausea, repeated vomiting, photophobia, phonophobia, blurred vision, and neck stiffness, are well-documented manifestations of SIH and further support the diagnosis (1, 11-13). Notably, despite the recognized susceptibility of the abducens nerve to traction-related injury, no cranial neuropathies were observed in this case.

Diagnosis of SIH relies on the integration of characteristic clinical features with supportive imaging findings. Brain MRI remains the most sensitive modality and typically demonstrates diffuse pachymeningeal enhancement, venous sinus distension, and subdural collections, while cerebellar tonsillar descent and brainstem sagging may be present in more advanced cases (2, 3). In our patient, early MRI evaluation was decisive, particularly given the normal findings on cranial MSCT and the absence of focal neurological deficits. Although lumbar puncture may reveal low opening pressure, normal values do not exclude the diagnosis, and the procedure was not required in this case due to the convincing clinical and radiological presentation. Spinal MRI further substantiated the diagnosis by identifying an epidural fluid collection consistent with CSF leakage.

Management strategies range from conservative measures to invasive interventions. Initial treatment typically includes bed rest, hydration, and caffeine, whereas epidural blood patching is considered in patients with persistent or severe symptoms (1). In the present case, conservative therapy alone resulted in gradual clinical improvement

over ten days, without the need for epidural blood patching. Early diagnosis and appropriate management are essential to prevent potentially serious complications such as subdural hematoma, cerebral venous sinus thrombosis, and superficial siderosis (8).

This case illustrates a typical yet diagnostically challenging presentation of SIH in a young postpartum woman, in whom early recognition of orthostatic headache and timely MRI were crucial for establishing the diagnosis and guiding management. The clinical course and radiological findings closely reflect the underlying pathophysiological mechanisms described in the literature.

CONCLUSION

SIH should be considered in postpartum patients presenting with new-onset orthostatic headache, even in the absence of trauma or prior neuraxial procedures. Careful clinical evaluation combined with early MRI facilitates prompt diagnosis and timely initiation of appropriate therapy, often allowing complete recovery with conservative management alone. Increased awareness of SIH across diverse clinical settings is essential to prevent diagnostic delay and reduce the risk of serious complications.

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CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

AUTHORS' CONTRIBUTIONS

All authors contributed equally to all aspects of the preparation of this manuscript and meet the ICMJE criteria for authorship.

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