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Case Report

Balancing the System: Occult Spinal CSF Leak Leading to Spontaneous Intracranial Hypotension

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ABSTRACT

Spontaneous intracranial hypotension (SIH) is an underdiagnosed cause of headache and neurological dysfunction, often missed when classical orthostatic features are absent. We present a 43-year-old woman with chronic, non-specific neurological symptoms ultimately found to have an occult spinal cerebrospinal fluid (CSF) leak. Brain MRI demonstrated diffuse pachymeningeal enhancement, and CT myelography confirmed extraspinal CSF egress. The patient experienced complete resolution following targeted epidural blood patching. This case highlights the diagnostic challenges of SIH and emphasises the importance of maintaining suspicion and using appropriate imaging, even when routine studies appear normal.

INTRODUCTION

The causes and consequences of spontaneous cerebrospinal fluid (CSF) hypertension are generally well known, with a relatively well-established set of criteria available to aid diagnosis (Dandy criteria).(1,2) Spontaneous occult CSF hypotension, on the contrary, is an entity that remains underdiagnosed,(1,2) particularly in middle- and lower-income countries.

Under diagnosis is principally due to a lack of awareness of the condition. A history of headache, worse in the erect position and relieved by recumbence, suggests the presence of spontaneous intracranial hypotension (SIH). Afflicted patients, however, do not always present with a posture-dependent headache, particularly when the disease process has become chronic. They may present solely with several non-specific and disparate symptoms and signs.

Early diagnosis requires a high index of suspicion, as routine brain and spinal imaging may appear unremarkable. We present a case of occult CSF leak causing SIH, followed by a focused review to improve its clinical recognition and reduce misdiagnoses.

CASE

A 43-year-old female patient presented with a three-month history of a feeling of fullness in her head associated with visual and hearing impairment, dizziness, and headaches that were worse in the erect position. Prior ophthalmological and otorhinolaryngological consultation and clinical examination were normal. A non-contrast brain MRI was unremarkable. Symptomatic treatment in the form of anti-vertigo medication and analgesics was ineffective. The patient, a counselling psychologist, was unable to function at work and consequently sought neurological consultation.

No lateralising signs were noted on neurological examination. Neck stiffness was absent, and no focal neurological deficits were detected. MRI of the brain performed six months later demonstrated diffuse pachymeningeal enhancement (Figure 1A). A lumbar puncture was performed as meningitis and neurosarcoidosis were initially considered. The CSF opening pressure was 6 mmHg. Cytological and microbiological analyses were normal. Chest radiography, serum inflammatory markers, and angiotensin-converting enzyme levels were normal, effectively excluding sarcoidosis.

MRI examination of the entire spine was normal. Subsequent CT myelography revealed multiple arachnoid outpouchings (Tarlov cysts) and extraspinal CSF egress, particularly in the thoracic spine (Figure 1B, C). At this level, CSF was seen tracking along the intercostal nerves. A thoracic epidural blood patch was performed, resulting in partial symptomatic relief. The procedure was repeated one month later, with complete resolution of symptoms.

DISCUSSION

The syndrome of spontaneous intracranial hypotension has been recognised for more than eight decades. The introduction of MRI has greatly facilitated diagnosis.(3) Nevertheless, SIH continues to be frequently underdiagnosed and misdiagnosed. Schievink described patients with SIH who underwent unnecessary invasive procedures such as catheter angiography, dural biopsy, and even craniotomy due to misdiagnosis.(3)

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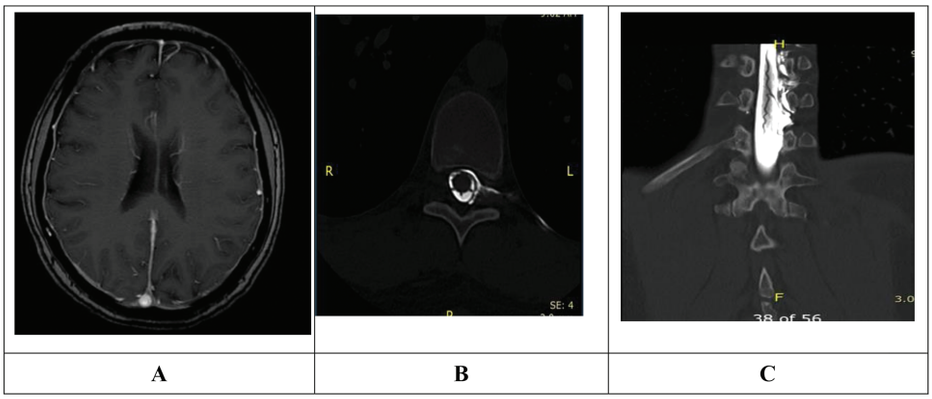

Figure 1: A: Axial MRI showing pachymeningeal enhancement. **B:** Axial CT myelogram with CSF leak tracking along the intercostal nerve. **C:** Coronal CT myelogram with CSF tracking along a spinal nerve.

CLINICAL FEATURES

The exact epidemiology of SIH remains unknown and is frequently mischaracterised as rare. An orthostatic headache typically raises clinical suspicion; however, with chronicity, the headache may lose its postural component or even be absent. Some patients present instead with a constellation of non-specific neurological features, including blurred vision, dizziness, and cognitive dysfunction.(1,2)

Diagnosis can be challenging, as brain and spinal imaging may initially be normal, as seen in our patient. A low CSF opening pressure on lumbar puncture may support the diagnosis, but it is neither sensitive nor consistently present. Although the site of CSF loss in this case was identified using static CT myelography, dynamic myelography may offer a higher diagnostic yield, though it is not universally available.(1,2)

Epidural blood patches remain the first-line treatment for SIH. When unsuccessful, alternative interventions, including surgical repair, may be considered.(1,2)

PATHOPHYSIOLOGY

Spontaneous intracranial hypotension is invariably caused by spinal CSF leak(s), which are frequently cryptic.(1,2) CSF loss via the cribriform plate may occur, but is usually clinically apparent and rarely causes the SIH syndrome.

Headache associated with intracranial hypotension is thought to result from traction on pain-sensitive intracranial structures and compensatory cerebral vasodilation. Venous sinus engorgement has also been implicated in symptom generation.(2,4)

Although termed intracranial "hypotension," the primary pathological mechanism is CSF volume depletion, most commonly originating from the spinal thecal sac.(1,2,4)

In contrast, intracranial hypertension is well described by the Monro–Kellie doctrine, which states that the intracranial volume is fixed and composed of brain tissue, blood, and CSF.(5) An increase in one component necessitates a compensatory decrease in another. Conversely, a reduction in CSF volume leads to an increase in intracranial blood volume, particularly venous blood, explaining many of the imaging features seen in SIH.(2,3,5)

These radiological findings include pachymeningeal enhancement, venous sinus congestion, and pituitary engorgement. The pachymeninges lack a blood–brain barrier, accounting for their characteristic enhancement on contrast-enhanced MRI.(3)

Spontaneous intracranial hypotension may be as clinically significant as idiopathic intracranial hypertension, with severe complications, including stroke and death, having been reported.(6) Normal neurological function requires a delicately balanced intracranial environment, and disturbances in either direction may have profound consequences.

CONCLUSION

Spontaneous intracranial hypotension is frequently overlooked, particularly in resource-limited public-sector settings where access to advanced imaging may be restricted. Clinicians should maintain a high index of suspicion in patients presenting with vague or atypical neurological symptoms, especially when headaches demonstrate any postural component. Contrast-enhanced MRI and CT myelography significantly improve diagnostic accuracy. Increased clinical awareness may improve case detection and facilitate the development of local epidemiological data.

REFERENCES

1. Schievink WI. Spontaneous spinal cerebrospinal fluid leaks and intracranial hypotension. JAMA. 2006; 295(19):2286–2296. doi:10.1001/jama.295.19.2286
2. Mokri B. Spontaneous intracranial hypotension. Continuum (Minneap Minn). 2015; 21(4):1086–1108. doi:10.1212/CON.0000000000000198
3. Schievink WI. Misdiagnosis of spontaneous intracranial hypotension. Arch Neurol. 2003; 60(12):1713–1718. doi:10.1001/archneur.60.12.1713

4. Fishman RA, Dillon WP. Dural enhancement and cerebral displacement secondary to intracranial hypotension. Neurology. 1993; 43(3):609–616. doi:10.1212/WNL.43.3_Part_1.609
5. Wilson MH. Monro-Kellie 2.0: the dynamic vascular and venous pathophysiological components of intracranial pressure. J Cereb Blood Flow Metab. 2016; 36(8):1338–1350. doi:10.1177/0271678X16648711
6. Schievink WI. Stroke and death due to spontaneous intracranial hypotension. Neurocrit Care. 2013; 18(2):248–251. doi:10.1007/s12028-012-9797-5

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