Radiology Case Reports 20 (2025) 1338–1341

Available online at www.sciencedirect.com

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journal homepage: www.elsevier.com/locate/radcr

Case Report

Iatrogenic intracranial hypotension complicated to dural venous sinus thrombosis and lobar hemorrhage: A case report ☆

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ARTICLE INFO

Article history:
Received 1 November 2024
Accepted 13 November 2024

Keywords:
Iatrogenic intracranial hypotension
Intracranial hypotension, Cerebral venous sinus thrombosis
Lobar parenchymal hemorrhage
Lumbar puncture
Case report

ABSTRACT

Iatrogenic intracranial hypotension is a known complication of spinal anesthesia that can lead to more severe conditions, such as dural or cerebral venous sinus thrombosis (CVST).

This report presents a case of intracranial hypotension in a young woman after lumbar anesthesia for a cesarean section that was complicated by CVST and subsequently by lobar hemorrhage, clinically presenting with severe headache and seizures. The diagnosis was made via cerebral magnetic resonance (MR) imaging, and the patient was treated medically.

This case study aimed to assist clinicians in considering the possible complications of intracranial hypotension when evaluating patients with a recent history of spinal procedures, leading to early diagnosis and treatment to prevent devastating consequences.

Introduction

Intracranial complications induced by spinal procedures are rare and mostly result from intracranial hypotension due to lumbar puncture and cerebrospinal fluid (CSF) leakage [1]. Cerebral venous sinus thrombosis (CVST) is a rare intracranial condition in the normal population, but its incidence increases with the presence of intracranial hypotension, reaching 2% [2].

The most common clinical presentation of intracranial hypotension is postural/orthostatic headache; other clinical manifestations include nausea, vomiting, neck pain, dizziness, and visual and hearing disturbances including diplopia, tinnitus, and vertigo [1–3]. The progression of CVST complicates the clinical presentation and masks the symptoms of intracranial hypotension; orthostatic headache changes to a continuous headache, and the patient progresses to seizures and even death [5]. The most common radiological feature of intracranial hypotension is pachymeningeal enhancement

Abbreviations: CE-MRI, contrast-enhanced magnetic resonance imaging; CSF, cerebrospinal fluid; CVST, cerebral venous sinus thrombosis; EDB, epidural blood patch; IIH, iatrogenic intracranial hypotension; MR, magnetic resonance; MRV, magnetic resonance venography.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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https://doi.org/10.1016/j.radcr.2024.11.035

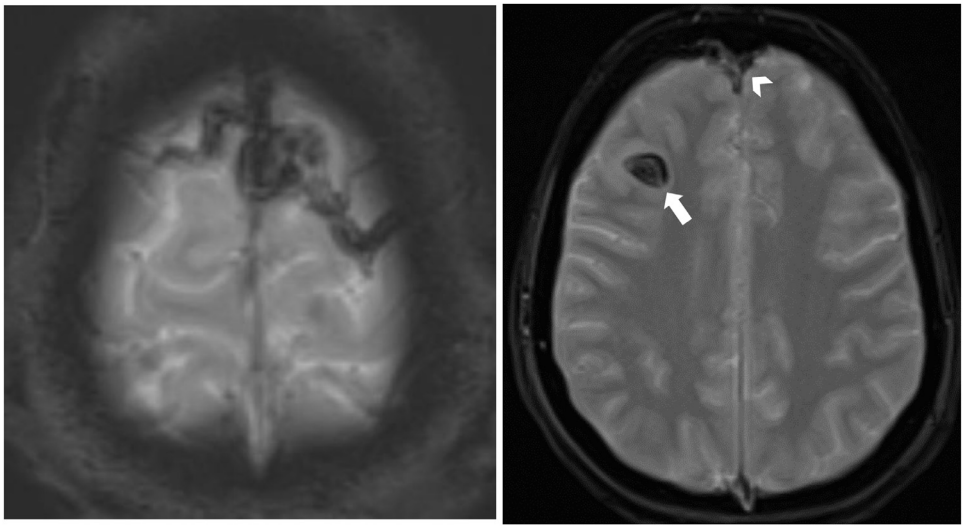

Fig. 1 – (A) T2* gradient echo axial image from high frontal regions: blooming artifact of the dilated superficial cortical veins. (B) Focal area of blooming artifact (white arrow) in the periphery of right frontal lobe representing lobar hemorrhage. There is also blooming artifact in the anterior aspect of the superior sagittal sinus (white arrow head) representing thrombosis.

on contrast-enhanced magnetic resonance imaging (CE-MRI). Other MR imaging features include meningeal thickening, descent of the cerebellar tonsils and brainstem, pituitary hyperemia, and, rarely, subdural fluid collections (hygromas and hematoma) [1]. Imaging can also be used to diagnose CVSTs and complications, such as venous hemorrhage [4]. The authors present a case of iatrogenic intracranial hypotension complicated by cerebral venous sinus thrombosis with lobar hemorrhage in the right frontal lobe.

Case presentation

A 19-year-old female developed orthostatic headache 5-6 days after C-sectioning and spinal anesthesia. Subsequently, in the next few days, the headache pattern changed to a continuous severe headache and diplopia. Later, on day 14, she developed dizziness and 3 seizures within 1 day. She was first brought to the obstetrician and then to a neurologist who advised her to undergo brain MR venography.

MR imaging revealed extensive thrombosis of the cerebral superficial cortical veins (Fig. 1A), the anterior half of the superior sagittal sinus (Fig. 2A, Fig. 3), and parts of the left sigmoid sinus (Fig. 2B) complicated by a small lobar hemorrhage in the right frontal lobe (Figure1b). There was also diffuse smooth meningeal enhancement along the brain (Fig. 3), suggesting intracranial hypotension. The patient received conservative treatment for intracranial hypotension while receiving anticoagulants (warfarin and enoxaparin) for CVST. Patient follow-up was conducted via phone. Most of her symptoms resolved following medical therapy, but she still had dizziness. After 2 months of follow-up, all her complaints were resolved.

Discussion

The progression of cerebral venous sinus thrombosis (CVST) in a patient with iatrogenic CSF leakage was first reported in the 1980s [5]. Several cases of intracranial hypotension (spontaneous or iatrogenic) complicated by CVST have since been published, but cases of iatrogenic intracranial hypotension (IIH) complicated with CVST and lobar hemorrhage are rare [1].

According to pathophysiology, 3 mechanisms predispose the progression of CVST in patients with intracranial hypotension. First, according to the Monro-Kellie doctrine, as the sum of the brain, CSF, and intracranial blood is constant; therefore, a decrease in one of these 3 results in an increase in one or both of the other. In the case of CSF volume loss, compensatory increases in blood volume occur, mainly in the dural sinuses. This process leads to venous engorgement, reduces blood flow velocity, and increases the risk of thrombosis. Second, brain sagging because of CSF hypovolemia causes vein and sinus traction, which leads to mechanical stretching of the blood vessel endothelium and increases the chance of thrombosis. Third, the absorption of CSF into the dural venous sinuses decreases due to low CSF volume, resulting in a further increase in blood viscosity. These 3 mechanisms contribute to CVST formation [5,6].

Other risk factors for CVST include genetic factors, the cesarean delivery/postpartum period, pregnancy, oral contraceptives, inflammatory bowel disease, infection, drugs/substance abuse, and head trauma [2,4,5,7]. There should be clinical suspicion for CVST in patients with intracranial hypotension when there is a change in headache characteristics from orthostatic to continuous (as in the present patient). Moreover, clinical symptoms of CVST include continuous headache, dizziness, and seizures [8].

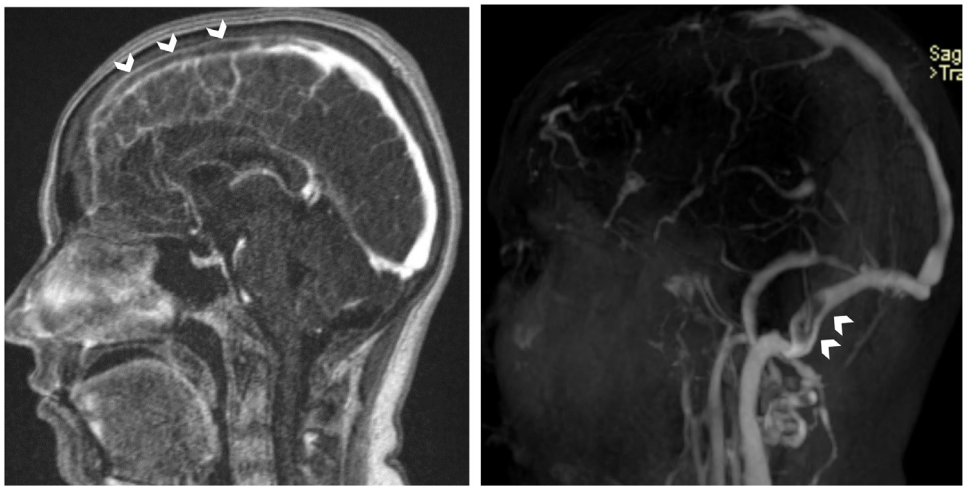

Fig. 2 – (A) Sagittal MRV image show long segment central filling defect in the anterior half of the superior sagittal sinuses (white arrow heads) with engorgement of the deeper veins. (B) Reconstructed MRV image sagittal view: nonopacification of the anterior half of the superior sagittal sinuses and large filling defect in the left sigmoid sinus (white arrow heads).

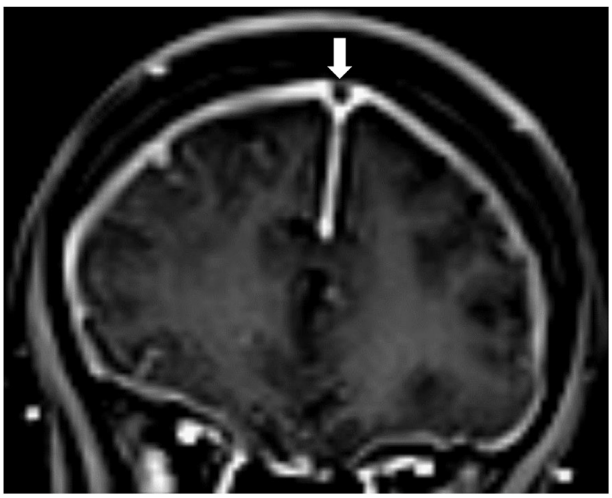

Fig. 3 – Contrast enhanced T1 fat saturated Coronary image: shows diffuse smooth meningeal enhancement along the brain coverage. Filling defect is noted in the superior sagittal sinus (white arrow) representing thrombosis of the superior sagittal sinus.

According to our patient's clinical presentations, she initially had an orthostatic headache for the first 5-6 days. The headache pattern subsequently changed to continuous and worsened, suggesting the development of intracranial hypotension in patients with CVST syndrome. On the 14th day, the patient developed seizures. Keeping with this clinical presentation, the patient initially developed intracranial hypotension and subsequently developed CVST, as well as being in postcaesarean delivery status, which is another risk factor for CVST.

Radiographic features of intracranial hypotension on MR images range from pachymeningeal enhancement and venous sinus engorgement first to subdural effusion, which appears last. Other findings are downward displacement of the brain, hematoma formation, enlargement of the pituitary gland, descent of the cerebellar tonsils and brainstem, and crowding of the posterior fossa in severe cases of intracranial hypotension [3,8]. MR venography is the modality of choice for identifying CVSTs as filling defects within venous sinuses and lacking flow, as well as its sequelae. One-third of patients with CVST develop lobar parenchymal hemorrhage.

The management of intracranial hypotension in the presence of CVST is still uncertain. However, the literature suggests the initial management of intracranial hypotension with conservative treatments and an epidural blood patch (EDB), followed by the use of anticoagulants for CVST. However, the use of anticoagulants is controversial because they increase the risk of bleeding [1,3,8]. In our patient, intracranial hypotension was treated conservatively, and CVST was treated with anticoagulants (warfarin and enoxaparin).

Conclusions

This case study aims to assist clinicians in considering the possible complications of intracranial hypotension when evaluating patients with a recent history of spinal procedures, leading to early diagnosis and treatment to prevent devastating consequences.

Patient consent

Written informed consent was obtained from the patient for publication. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

REFERENCES

[1] Saffarini S, Kamil S, Suradi Y. Superior sagittal sinus thrombosis in a patient with iatrogenic intracranial hypotension: a case report. Cureus 2023;15(8):1–8.

[2] MC Garcia-Carreira, Vergé DC, Branera J, Zauner M, Herrero JE, Tió E, et al. Cerebral venous thrombosis in two patients with spontaneous intracranial hypotension. Case Rep Neurol Med 2014;2014(1):528268.

[3] Honig A, Eliahou R, Pikkel YY, Leker RR. Iatrogenic intracranial hypotension and cerebral venous thrombosis. J Neurol Sci 2016;366:191–4.

[4] Sinnaeve L, Vanopdenbosch L, Paemeleire K. Association of cerebral venous thrombosis and intracranial hypotension: review of 3 cases. J Stroke Cerebrovasc Dis 2017;26(8):e165–9.

[5] Ferrante E, Trimboli M, Petrecca G, Allegrini F. Cerebral venous thrombosis in spontaneous intracranial hypotension: a report of 8 cases and review of the literature. J Neurol Sci 2021;425:117467.

[6] Oh J, Han SW, SY Chung. Cerebral venous thrombosis combined with spontaneous intracranial hypotension: a case report and review of the literature. J Neurosonol Neuroimag 2023;15(1):57–9.

[7] Huang P. Rare cause of cerebral venous sinus thrombosis: spontaneous intracranial hypotension syndrome: a case report. World J Clin Cases 2023;11(19):4677.

[8] Lee CY. Treatment for spontaneous intracranial hypotension associated with cerebral venous thrombosis resulting in severe neurologic deterioration. Interdisciplin Neurosurg 2023;34:101815.