NICeR
Journal of Neurointerventional Case Reports

Rapid Resolution of Cerebral Venous Sinus Thrombosis Following Transvenous Embolization of a Cerebrospinal Fluid-Venous Fistula in Spontaneous Intracranial Hypotension

CASE REPORT

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TO CITE THIS ARTICLE:
Risi G, Mourand I, Formoso A, Lonjon N, Costalat V, et al. Rapid Resolution of Cerebral Venous Sinus Thrombosis Following Transvenous Embolization of a Cerebrospinal Fluid-Venous Fistula in Spontaneous Intracranial Hypotension. Journal of Neurointerventional Case Reports. 2025;2(1):6, 1-4. DOI: https://doi.org/10.70355/NICeR.11

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Received: 2 December 2024; Accepted: 6 January 2025; Published Online: 3 March 2025

ABSTRACT

Cerebral venous thrombosis (CVT) is a rare but potentially life-threatening cerebrovascular condition. Spontaneous intracranial hypotension (SIH), typically caused by cerebrospinal fluid (CSF) leaks, is a well-documented predisposing factor for CVT, occurring in approximately 1%–2% of SIH cases. The optimal approach for managing SIH-related CVT remains unclear. We report the case of a 63-year-old female who presented with extensive CVT secondary to SIH. Initial treatment of CVT included low-molecular-weight heparin anticoagulation. Diagnostic lateral decubitus digital subtraction myelography identified a right-sided CSF-venous fistula (CSFVF) at the T7–T8 spinal level as the source of SIH. The CSFVF was successfully treated with transvenous endovascular embolization using Onyx. This intervention led to the rapid resolution of both SIH and CVT, as confirmed by a follow-up brain MRI performed 5 days post-embolization.

KEYWORDS

cerebral venous thrombosis (CVT), spontaneous intracranial hypotension (SIH), CSF-venous fistula (CSFVF), endovascular embolization

INTRODUCTION

Cerebral venous thrombosis (CVT) is a rare cerebrovascular disorder, with an estimated prevalence of approximately five cases per million individuals (0.0005%) in the general population, with a higher incidence in females.[1] In Western countries, the annual incidence of spontaneous intracranial hypotension (SIH) is estimated at four to five cases per 100 000 people, predominantly affecting women aged 35–55 years [2]. CVT can occur as a complication of SIH in 1%–2% of cases, a prevalence markedly higher than in the general population, suggesting that SIH is a predisposing factor for CVT.

SIH is caused by cerebrospinal fluid (CSF) leakage through dural tears, nerve root diverticula, or direct fistulas into the periradicular veins (CSF-venous fistulas [CSFVFs]). Currently, there is no consensus on the optimal treatment or management of SIH-related CVT.

This report presents the case of a patient with extensive thrombosis of the superior sagittal sinus (SSS), the right transverse sigmoid venous sinus (TVS), and the right internal jugular vein, secondary to SIH associated with a CSFVF. Targeted treatment of the fistula led to rapid regression of the CVT.[3,4] The case highlights diagnostic challenges, treatment approaches, and radiological and clinical outcomes associated with SIH-related CVT.

CASE PRESENTATION

A 63-year-old female with a medical history of breast cancer presented to the emergency department with aphasia, psychomotor retardation, right-sided motor deficit, sixth cranial nerve palsy with diplopia, and partial epileptic seizures (NIHSS score: 13). Notably, the patient reported no history of headache either prior to admission or at any point in her life.

She was a non-smoker with no history of hypertension, cerebrovascular events, trauma, or known clotting disorders. On admission, a brain MRI with MR venography demonstrated extensive CVT involving the SSS, right TVS, right sigmoid sinus, and right jugular vein. This was complicated by a right frontal intraparenchymal venous infarction and hematoma (Figure 1). Additional findings included bilateral pachymeningeal enhancement, cerebral venous engorgement, and effacement of the suprasellar cistern (<4 mm), with a Bern score of 6, suggestive of underlying SIH.[5]

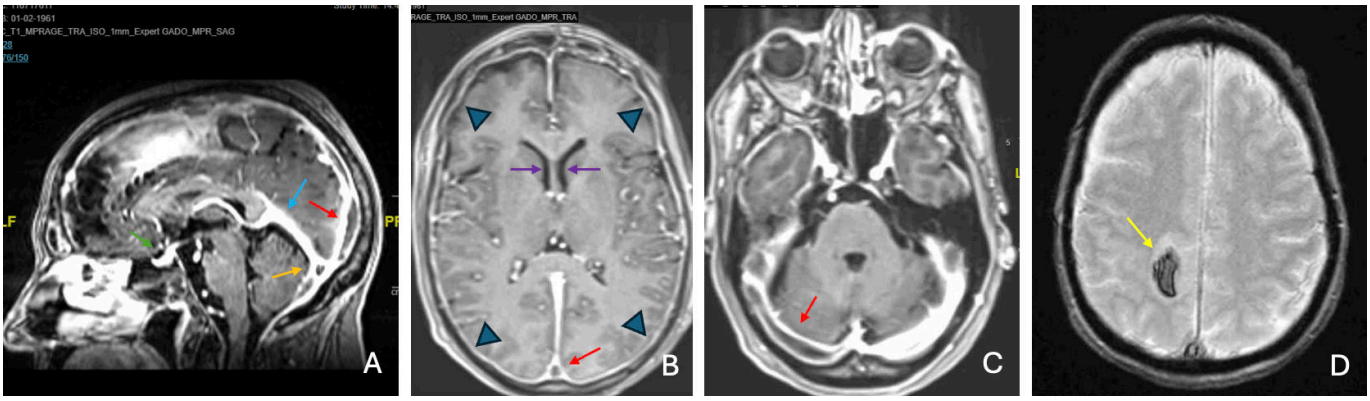

FIGURE 1. (A) Sagittal gadolinium-enhanced brain T1-weighted MRI showing superior sagittal sinus thrombosis (SSS) (red arrow) and torcular thrombosis (orange arrow), associated with signs of spontaneous intracranial hypotension, including engorgement of the straight sinus (blue arrow) and effacement of the suprasellar cistern (green arrow). (B) Axial gadolinium-enhanced brain T1-weighted MRI showing bilateral pachymeningeal enhancement (arrowheads), SSS thrombosis (red arrow), and reduced diameter of the lateral ventricles (purple arrows). (C) Right transverse venous sinus thrombosis (red arrow). (D) Hemorrhagic transformation of a right frontal parenchymal infarction due to cerebral venous sinus thrombosis.

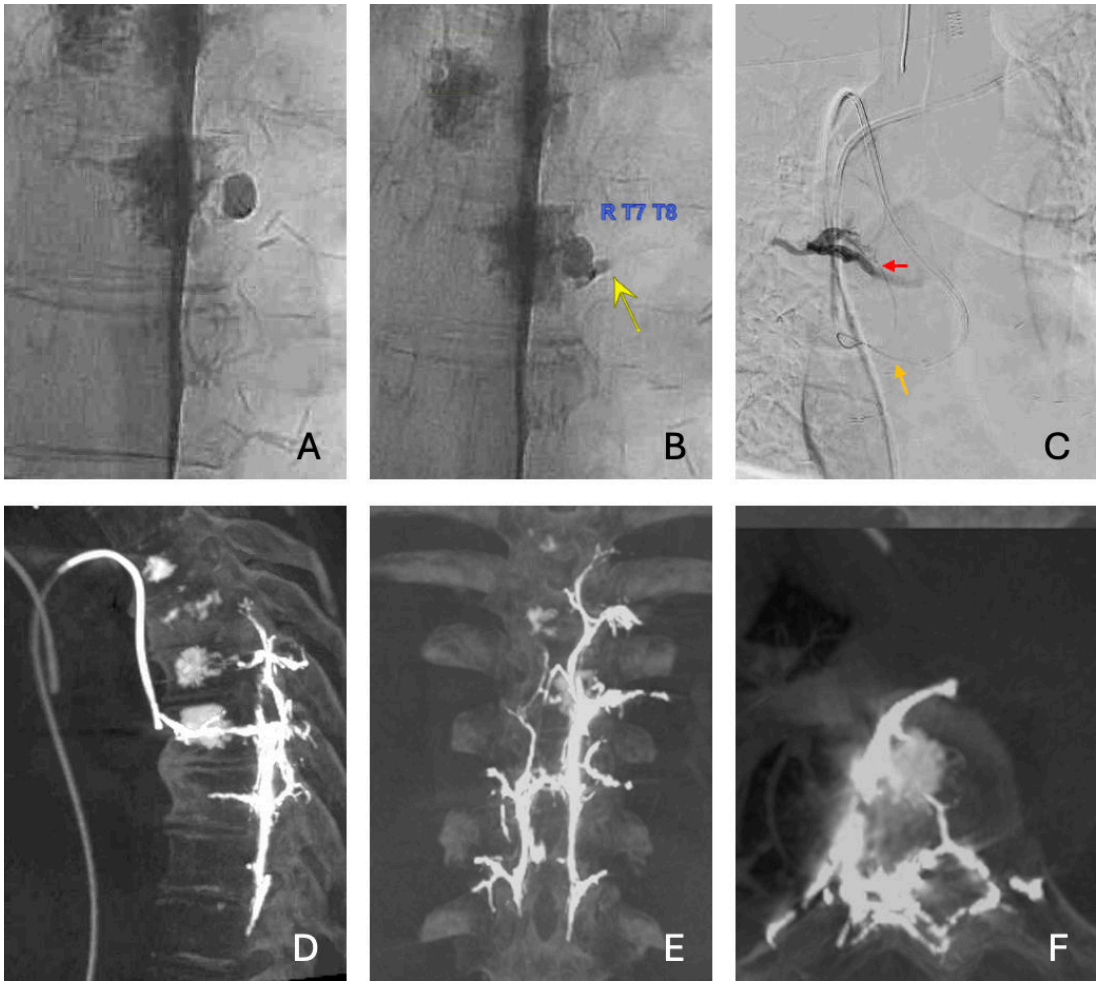

FIGURE 2. (A) Lateral decubitus right-sided digital subtraction dynamic myelography demonstrating a T7 for aminal dural cyst. (B) A cerebrospinal fluid-venous fistula originating from the cyst (yellow arrow). (C) Transvenous embolization of the fistula via catheterization of the azygos vein. The right T7 segmental vein (red arrow) was accessed using a 156 cm Headaway Duo catheter navigated through the epidural plexus (orange arrow) from the T8 segmental vein. (D)–(F) Post-embolization cone-beam CT images in sagittal (D), coronal (E), and axial (F) reconstructions showing successful embolization with Onyx.

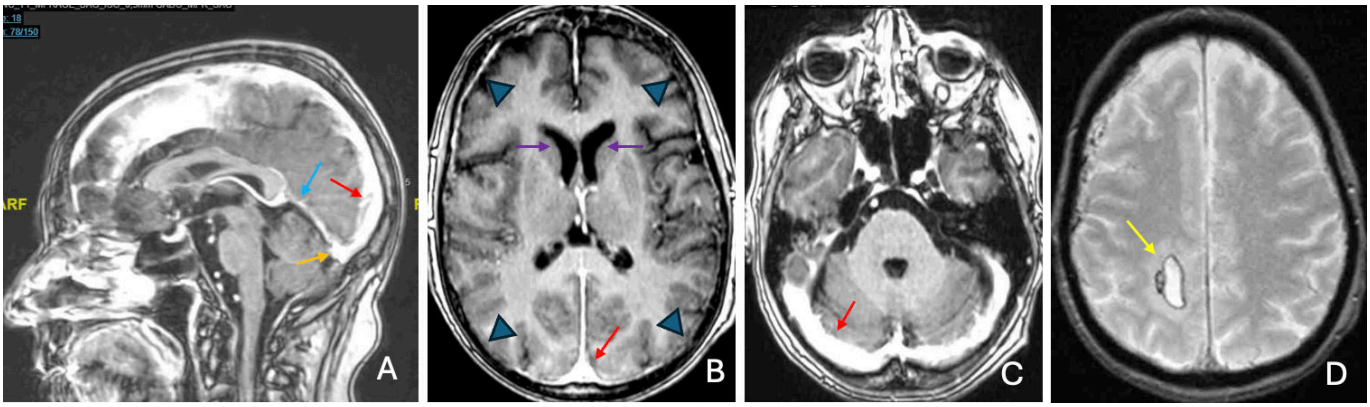

FIGURE 3. (A) Sagittal gadolinium-enhanced brain T1-weighted MRI 5 days after cerebrospinal fluid-venous fistula embolization, demonstrating resolution of superior sagittal (red arrow) and torcular (orange arrow) sinus thrombosis, as well as resolution of straight sinus engorgement (blue arrow). (B) Axial gadolinium-enhanced brain T1-weighted MRI showing resolution of bilateral pachymeningeal enhancement (arrowheads), recanalization of the superior sagittal sinus (red arrow), and increased lateral ventricle diameter (purple arrows). (C) Patent right transverse venous sinus (red arrow). (D) Stability of the hemorrhagic transformation of the frontal parenchymal infarction.

The patient was admitted to the neurology intermediate care unit with a diagnosis of SIH-related CVT complicated by a right frontoparietal hematoma. Treatment with low-molecular-weight heparin (LMWH; enoxaparin) for anticoagulation and valproate for seizure management was initiated. Neurological symptoms progressively improved, with an NIHSS score of 4 within the first 48 hours of treatment.

During hospitalization, the patient tested positive for COVID-19 after developing respiratory symptoms. A follow-up brain MRI on day 5 showed no significant improvement in the CVT, with stable radiological signs of SIH. High-resolution spine MRI with 3D T2 SPACE and fat suppression sequences ruled out the presence of a spinal longitudinal extradural CSF collection.

Given the diagnostic complexity, a multidisciplinary team comprising neurologists, neuroradiologists, and neuroanesthesiologists decided to perform lateral decubitus digital subtraction myelography (DSM) to identify a CSFVF as the underlying cause of SIH. Anticoagulation with LMWH was withheld 24 hours prior to the procedure.[6]

DSM confirmed the presence of a CSFVF at the T7–T8 level (Figure 2). Under general anesthesia, a transvenous endovascular embolization with Onyx was performed. Through a right femoral vein approach, a 6 F Benchmark catheter was advanced into the azygos vein, and a 156 cm Headway Duo microcatheter was navigated through the epidural plexus to the targeted segmental vein at T7. Embolization with Onyx was successfully completed without procedural complications.

Five days after the embolization, a follow-up brain MRI demonstrated complete recanalization of the cerebral sinuses and a significant reduction in SIH-related findings (Figure 3). Anticoagulation was resumed post-procedure with LMWH for 2 weeks and subsequently transitioned to direct oral anticoagulants.

The patient was transferred to another facility 2 weeks post-procedure. At discharge, her neurological condition had substantially improved with an NIHSS score of 2.

DISCUSSION

CVT is a recognized complication of SIH and is attributed to several interrelated mechanisms. First, a reduction in CSF volume leads to increased cerebral blood volume, resulting in venous sinus dilation, venous stasis, and thrombosis.[7,8] Second, brain sagging due to CSF depletion can stretch and damage the venous endothelium, distorting vessel walls and creating a prothrombotic state.[9] Third, reduced CSF volume elevates blood viscosity, impairing CSF absorption into the venous sinuses. These morphological and hemodynamic changes collectively heighten the risk of CVT in patients with SIH.

In more than 50% of reported cases of SIH-related CVT, the underlying CSF leak was either not investigated or not identified. However, among the cases where a leak was detected, the majority (80%) were classified as type I leaks, while only a minority (5%) were type II.[10] A single recent report described a CSFVF as the cause of CVT in a patient presenting with coma, successfully treated with transvenous Onyx embolization.[11]

Currently, no standardized treatment guidelines exist for managing SIH-related CVT. A recent systematic review[10] emphasized that complete resolution of CVT was primarily achieved when anticoagulation therapy was combined with treatment of the underlying cause—SIH—using an epidural blood patch.

In the present case, a right-sided T7 CSFVF was identified and successfully treated using a transvenous approach. MRI findings indicative of SIH resolved within a few days after embolization, accompanied by complete resolution of the CVT. This outcome highlights the critical importance of accurately identifying and selectively treating the CSF leak in cases of SIH-related CVT, as addressing the underlying pathophysiological mechanism of SIH is essential for effective CVT management.

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Patient consent

Patient consent was obtained for the publication of patient images and associated medical information using the institutional form.

Contributors

Each author made substantial contributions to the conception, design, acquisition, analysis, or interpretation of data for the study.

Competing interests

The authors declare that they have no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Acknowledgements

None.

REFERENCES

1. Ferro JM, Aguiar de Sousa D. Cerebral venous thrombosis: An update. Current Neurology and Neuroscience Reports. 23 Aug 2019; 19(10): 74 DOI: 10.1007/s11910-019-0988-x.
2. Schievink WI. Spontaneous intracranial hypotension. The New England Journal of Medicine. 2 Dec 2021; 385(23): 2173–2178. DOI: 10.1056/NEJMra2101561.
3. Cagnazzo F, Ducros A, Risi G, et al. Safety and efficacy of transvenous embolization of cerebrospinal fluid-venous fistula in patients with spontaneous intracranial hypotension. Interventional Neuroradiology. 23 Apr 2024: 15910199241247698 DOI: 10.1177/15910199241247698.
4. Brinjikji W, Madhavan A, Garza I, et al. Clinical and imaging outcomes of 100 patients with cerebrospinal fluid-venous fistulas treated by transvenous embolization. Journal of NeuroInterventional Surgery. Dec 2024; 16(12): 1256–1263. DOI: 10.1136/jnis-2023-021012.
5. Dobrocky T, Grunder L, Breiding PS, et al. Assessing spinal cerebrospinal fluid leaks in spontaneous intracranial hypotension with a scoring system based on brain magnetic resonance imaging findings. JAMA Neurology. 1 May 2019; 76(5): 580–587. DOI: 10.1001/jamaneurol.2018.4921.
6. Schievink WI, Maya MM, Moser FG, et al. Lateral decubitus digital subtraction myelography to identify spinal CSF-venous fistulas in spontaneous intracranial hypotension. Journal of Neurosurgery Spine. 1 Dec 2019; 31(6): 902–905. DOI: 10.3171/2019.6.SPINE19487.
7. Mokri B. The Monro-Kellie hypothesis: applications in CSF volume depletion. Neurology. 26 Jun 2001; 56(12): 1746–1748. DOI: 10.1212/wnl.56.12.1746.
8. Yoon KW, Cho MK, Kim YJ, Lee SK. Sinus thrombosis in a patient with intracranial hypotension: a suggested hypothesis of venous stasis. a case report. Interventional Neuroradiology. Jun 2011; 17(2): 248–251. DOI: 10.1177/159101991101700218.
9. Haritanti A, Karacostas D, Drevelengas A, et al. Spontaneous intracranial hypotension: clinical and neuroimaging findings in six cases with literature review. European Journal of Radiology. Feb 2009; 69(2): 253–259. DOI: 10.1016/j.ejrad.2007.10.013.
10. Risi G, Ducros A, van Dokkum L, et al. Prevalence, clinical presentation, and treatment-management of cerebral venous thrombosis associated with spontaneous intracranial hypotension: A combined case-series and systematic literature review approach. Interventional Neuroradiology. 21 Jul 2024: 15910199241263139 DOI: 10.1177/15910199241263139.
11. White TG, Turpin J, Shao M, et al. Cerebrospinal fluid venous fistula causing spontaneous intracranial hypotension resulting in venous sinus thrombosis and coma. Interventional Neuroradiology. 23 Aug 2024: 15910199241272582 DOI: 10.1177/15910199241272582.