JNeViS
Journal of Neurointervention and Stroke

Journal of Neurointervention and Stroke 2025;1(2):83-87
doi: 10.63937/jnevis-2025.12.20

Case Report

Endovascular Stenting for Chronic Headache Secondary to Cerebral Venous Sinus Stenosis: A Case Report

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Received: November 15, 2025
Revised: November 27, 2025
Accepted: November 30, 2025
Published: December 2, 2025

Introduction: Chronic headache poses a significant diagnostic and therapeutic challenge. Cerebral venous sinus stenosis (CVSS) is an underrecognized yet important etiology in which stenosis impairs venous outflow and elevates intracranial pressure, leading to headache. Endovascular stenting offers a therapeutic approach by mechanically restoring venous flow. **Case:** A 43-year-old female presented with a chronic headache for 3 months, refractory to multimodal medical therapy. Digital subtraction angiography (DSA) confirmed stenosis in the bilateral transverse sinuses. The patient subsequently underwent successful endovascular venous stenting. **Conclusion:** This case underscores that CVSS is a potentially treatable cause of chronic headache. Endovascular intervention can provide rapid symptomatic relief for patients with medically refractory headache secondary to venous sinus stenosis. Long-term follow-up remains essential to confirm sustained benefit.

Keywords: Cerebral venous sinus stenosis, Chronic headache, Endovascular stenting, Venous sinus stenting

Highlights
- CVSS is an underdiagnosed but reversible cause of chronic headache.
- Venous stenting effectively restores venous outflow and reduces ICP.
- Early recognition and intervention lead to rapid and sustained symptoms resolution.

Introduction

Chronic headaches are among the most prevalent and disabling conditions worldwide and significantly impact quality of life.[1] Management is often challenging due to their diverse etiologies and limitations in diagnostic approaches, which make accurate and effective treatment difficult.[2]

Among the various causes of chronic headache, cerebral venous sinus stenosis (CVSS) a form of cerebral venous vascular disease should also be considered.[3] CVSS affects headache symptoms by altering cerebral autoregulation. Cerebral circulation consists of both arterial and venous systems. CVSS is a condition in which obstruction or slowing of the venous system impairs venous outflow. Several studies have shown that this condition can lead to elevated intracranial pressure, disrupted cerebral blood flow, impaired blood–brain barrier function, and ultimately worsened intracranial hypertension.[4,5]

CVSS is increasingly recognized as a significant factor in the pathophysiology of idiopathic intracranial hypertension (IIH). However, several critical questions regarding its precise role remain unanswered. The most fundamental unresolved issue concerns the causal relationship: whether CVSS is a primary etiology of IIH or a secondary consequence of elevated intracranial pressure.[6]

This uncertainty is further compounded by a scarcity of high-quality clinical evidence. There is a notable lack of detailed case reports and cohort studies focusing on specific demographic populations. This evidence gap hinders the development of clear guidelines for optimal

patient selection, precise procedural indications, and a comprehensive understanding of long-term therapeutic outcomes.[7]

The standard management of IIH typically follows a stepwise approach. Initial first-line therapy consists of conservative medical treatment, including lifestyle interventions such as weight loss, pharmacotherapy with carbonic anhydrase inhibitors (e.g., acetazolamide) and therapeutic lumbar punctures for transient symptom relief.[8] For patients who experience progressive visual loss refractory to medical management, surgical interventions remain the mainstay. These include cerebrospinal fluid (CSF) diversion procedures and optic nerve sheath fenestration (ONSF).[9]

More recently, venous sinus stenting (VSS) has emerged as a promising therapeutic modality for a specific patient cohort. It is not considered a first-line or universal therapy. Instead, VSS is generally reserved for patients with medically refractory symptoms and radiologically confirmed venous sinus stenosis accompanied by a significant trans-stenotic pressure gradient.[10]

In this report, we describe a successful endovascular intervention in a patient with transverse sinus stenosis presenting with symptoms of idiopathic intracranial hypertension.

Case

A 43-year-old right-handed woman with a normal BMI (20.8 kg/m²) presented with a 3-month history of continuous chronic headache that did not improve even with rest. She had taken several analgesic medications and had undergone a CT scan, which showed no remarkable findings, yet her symptoms persisted with a numeric pain rating scale of 6–7. During the anamnesis, the patient also reported diplopia and pulsatile tinnitus.

Physical examination revealed no limb weakness and no cranial nerve deficits. However, bilateral optic disc swelling and retinal hemorrhages were noted, suggesting elevated intracranial pressure. A lumbar puncture was performed to confirm this suspicion, revealing an opening pressure >400 mmH_2O, while cerebrospinal fluid analysis was within normal limits. These findings confirmed markedly elevated intracranial pressure.

Magnetic resonance venography demonstrated bilateral transverse sinus stenosis (Figure 1). Based on these findings, diagnostic angiography was performed. A cerebral digital subtraction angiography (DSA) confirmed stenosis in both transverse sinuses (Figure 2).

DSA combined with venous manometry was performed under conscious sedation. Vascular access was established percutaneously via the right common femoral artery for arterial angiography and the left common femoral vein for cerebral venous intervention. A 5F sheath was placed in the arterial access, while a 6F sheath was used for the venous side.

For the arterial phase, a guiding catheter was navigated to the relevant cerebral arteries following femoral puncture. For venous access, the guiding catheter was advanced from the femoral vein into the internal jugular vein. Selective cannulation of the superior sagittal sinus was then accomplished by navigating a 0.027-inch microcatheter over a 0.014-inch microwire under fluoroscopic guidance.

Following catheter positioning, contrast venography was performed to visualize the cerebral venous anatomy, including the superior sagittal sinus, transverse sinuses, sigmoid sinuses, and jugular bulbs. Subsequently, a comprehensive venous manometry was conducted. This was achieved by connecting a certified pressure transducer to the microcatheter, allowing for sequential pressure measurements at key venous locations to hemodynamically map the venous system.

Intravenous pressure measurements were as follows: superior sagittal sinus 45 cm H_2O, right transverse sinus 40 cm H_2O, and left transverse sinus 40 cm H_2O (Table 1). These results indicated that the venous stenosis significantly impeded venous outflow.

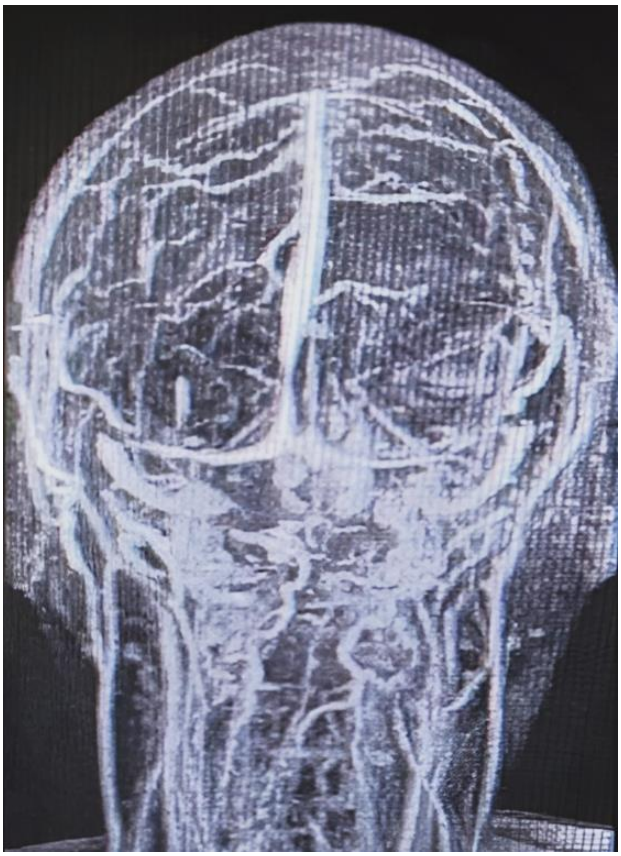

Figure 1. Magnetic resonance venography demonstrated stenosis in the bilateral transverse sinuses.

Table 1. Venous pressure gradient prior the treatment

Venous Location	Venous Pressure (cm H_2O)
Superior sagittal sinus	45
Left transverse sinus	40
Right transverse sinus	40

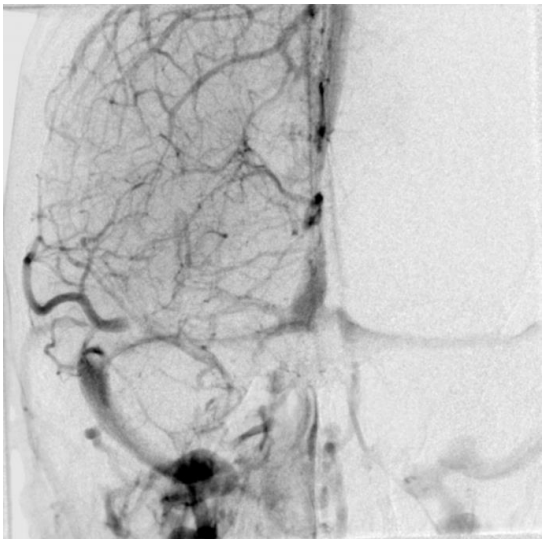

Figure 2. Cerebral DSA revealed stenosis in both transverse sinus.

The patient underwent therapeutic endovascular venous stenting under general anesthesia via right femoral venous access. Self-expanding stent were deployed across the stenosis via a transfemoral venous approach (Figure 3). Post-stenting pressure measurements confirmed resolution of the gradient, venous pressure decreased after endovascular venous stenting (Table 2). Sequential balloon dilations were performed using 4×20 mm, 5×20 mm, and 6×30 mm balloons. Subsequently, two self-expanding stents (7×30 mm and 8×40 mm) were deployed in an overlapping configuration within the right transverse sinus. No complications occurred during the procedure.

A follow-up evaluation 72 hours post-procedure showed that the CSF pressure had decreased to 250 mmH_2O. Post-stenting venous pressure measurements demonstrated a marked reduction in the venous pressure gradient, as shown in Table 2, confirming effective restoration of venous outflow. The patient experienced complete resolution of headache, tinnitus, and diplopia, consistent with reversal of IIH.

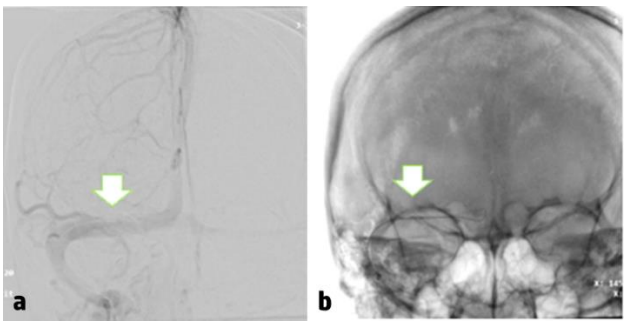

Figure 3. Cerebral DSA demonstrating post–venous stent placement with marked improvement of the stenosis (white arrow), shown on (a) subtraction images and (b) bone-window images.

Table 2. Venous pressure gradient after the treatment

Venous Location	Venous Pressure (cm H_2O)
Superior sagittal sinus	33
Left transverse sinus	22
Right transverse sinus	22

Discussion

CVSS is a type of cerebral venous vascular disease. Several studies have reported that venous sinus stenting is a feasible treatment for refractory IIH with continuous chronic headache,[11–13] and that most patients experience symptomatic improvement following stenting.[7,14] This case highlights the classic IIH triad: headache, papilledema, and pulsatile tinnitus. In addition, the presence of a markedly elevated opening pressure with normal cerebrospinal fluid composition supports the diagnosis[15,16]

Previous studies have shown that transverse sinus narrowing occurs in up to 83% of patients with IIH. Proposed mechanisms underlying IIH include increased CSF production, impaired CSF resorption, dysregulation of cerebral blood flow, and disturbances in overall fluid homeostasis. Another hypothesis suggests that venous outflow obstruction contributes to the development of IIH. However, the reversibility of these venous findings following effective medical therapy and reduction of intracranial pressure suggests that venous occlusion may be secondary to elevated intracranial pressure rather than the primary cause.[17]

A growing body of synthesized evidence consolidates the therapeutic position of VSS for IIH refractory to medical management.[18] Meta-analysis data substantiate its efficacy, with Raper et al., (2018) documenting pronounced reductions in the trans-stenotic pressure gradient, which correlated with significant clinical improvement.[19]

These findings are corroborated by Nicholson et al., (2019), who observed resolution of papilledema in 93.7% of cases and alleviation of headache in 79.6% of their cohort.[20] The consistent benefit of VSS is further affirmed by a recent review by Lim et al. (2023), which highlighted its effectiveness in ameliorating core symptoms of IIH, including visual disturbances, headache, and tinnitus, in patients who had failed conservative therapy.[18]

Despite the compelling efficacy profile, the potential for restenosis remains a consideration, with pooled analyses indicating an occurrence rate of approximately 17.7%. Nevertheless, when contextualized against its significant clinical benefits, the current evidence base robustly supports VSS as a viable and effective intervention for carefully selected patients who do not respond to conventional medical treatment.[18]

In our case, stent deployment at the site of sinus obstruction resulted in a significant reduction in the venous pressure gradient before and after treatment, as well as a marked decrease in CSF opening pressure. Follow-up evaluations confirmed that the patient experienced complete resolution of symptoms.

This case also demonstrates the use of self-expanding stents in venous sinus stenting. Currently, there are no dedicated stent devices specifically designed for use in the cerebral venous sinuses available on the clinical market.

Conclusion

CVSS should be considered in patients with continuous, treatment-resistant chronic headache accompanied by signs of elevated intracranial pressure. Endovascular stenting offers a promising therapeutic option, providing immediate hemodynamic improvement and symptom relief in selected cases.

Acknowledgement
None.

Conflict of Interest
All authors have no conflict of interest.

Patient consent for publication
Written consent was obtained from the patient and/or family for publication of this case.

Funding
None.

Author contribution
All authors contributed equally to all stages of the preparation of this case report.

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