JOURNAL OF NEUROSURGERY:
Case Lessons

J Neurosurg Case Lessons 9(10): CASE24781, 2025
DOI: 10.3171/CASE24781

Iatrogenic intracranial venous hypertension treated with intracranial venous stenting: illustrative case

M. Harrison Snyder, MD, Carl B. Heilman, MD, and Adel M. Malek, MD, PhD

Department of Neurosurgery, Tufts Medical Center, Boston, Massachusetts

BACKGROUND Venous sinus injury resulting in thrombosis is a possible complication of skull base surgery and neck dissection. Although usually asymptomatic, sinus thrombosis can obstruct the dominant cranial venous outflow pathways, leading to a cycle of increased intracranial pressure secondary to venous congestion, which further compresses the remaining sinuses in a positive feedback loop. This can present with symptoms resembling idiopathic intracranial hypertension.

OBSERVATIONS A patient underwent a left mastoidectomy for chronic mastoiditis, complicated by injury of the ipsilateral dominant sigmoid sinus, with subsequent thrombosis. The patient developed chronic severe headaches, papilledema, visual blurring, and tinnitus. Angiography revealed a contralateral arachnoid granulation causing stenosis of the remaining venous outflow tract with a pressure gradient of 18 mm Hg. Transverse sinus stenting led to normalization of the gradient and resolution of symptoms.

LESSONS Injury of a dominant cranial venous outflow pathway led to a feedback loop that caused increased intracranial pressures and worsening contralateral transverse sinus stenosis, which was successfully treated using transverse sinus stenting. Venous congestive physiology leading to intracranial hypertension is underrecognized as a complication of venous injury in skull base surgery and responds to stenting if the patient's symptoms are refractory to conservative management or anticoagulation.

https://thejns.org/doi/abs/10.3171/CASE24781

KEYWORDS idiopathic intracranial hypertension; transverse sinus stenting; endovascular treatment; intracranial pressure

Idiopathic intracranial hypertension (IIH) symptoms as a result of iatrogenic injury to the cranial venous sinuses have been described as an extremely rare entity, occurring in < 0.2% of cases in large series.[1] Iatrogenic venous sinus injury presents in a similar fashion to IIH with recurrent headaches (which can be holocranial, pulsatile, and refractory), papilledema, blurred and double vision, and pulsatile tinnitus. Prior reports have noted variable management of this complication, including ventriculoperitoneal or lumboperitoneal shunts, anticoagulation if the injury was associated with thrombosis, and medical management of intracranial pressure (ICP).[1,2]

The physiology of IIH has gained increased attention over the past decade, with venous hypertension discussed as an underlying impetus in many of these cases.[3] Transverse sinus stenosis (TSS) is a condition associated with IIH symptoms, as it can cause outflow reduction of blood from the cranial compartment causing venous hypertension and resultant increased ICP.[4–11] It is theorized that a positive feedback loop exists in patients with this condition, wherein elevations in ICP cause extrinsic compression of the venous sinuses leading to progressive stenosis; this stenosis then causes increased intramural venous pressures and venous congestion, further increasing ICP.[3,4] Stenting of the transverse sinus has emerged as a useful treatment option in patients with this physiology who demonstrate pressure gradients attributable to TSS between the proximal cerebral venous sinuses and the central venous system.[4–7,9,10] Stents in this area allow blood to flow uninterrupted out of the intracranial compartment, reducing venous congestion and ICPs. It is less invasive than other options such as cerebrospinal fluid (CSF) shunting and can help reduce complications seen with CSF shunt procedures.[4,10]

We present the unique case of a patient with iatrogenic venous congestive intracranial hypertension that developed after a surgical injury during mastoidectomy involving the patient's dominant venous system progressing to occlusive thrombosis. This initiated a positive feedback loop of increasing ICP and worsening contralateral stenosis of the nondominant venous system. The patient was successfully treated using

ABBREVIATIONS AVM = arteriovenous malformation; CBCT = cone beam computed tomography; CSF = cerebrospinal fluid; CVST = cerebral venous sinus thrombosis; ICP = intracranial pressure; IIH = idiopathic intracranial hypertension; IJV = internal jugular vein; MRI = magnetic resonance imaging; TSS = transverse sinus stenosis.

INCLUDE WHEN CITING Published March 10, 2025; DOI: 10.3171/CASE24781.
SUBMITTED November 13, 2024. **ACCEPTED** December 16, 2024.

venous sinus stent angioplasty. We also include a technical note on the use of large intermediate catheters to improve catheter access and navigation to the intracranial venous system.

Illustrative Case

A 64-year-old man had undergone a repeat left mastoidectomy for recurrent mastoiditis at another hospital years before his initial evaluation at our center. He had a history of chronic otological problems, with recurrent otitis media and mastoiditis. He had undergone several rounds of antibiotic treatment and a prior mastoid operation. This second mastoidectomy was complicated by a laceration of the left sigmoid sinus, causing significant blood loss requiring resuscitation with transfusion. The laceration caused a left sigmoid sinus thrombus.

The patient developed difficulty with vision in his right eye, with a scotoma in the superior visual field in his right eye, which he first noticed when awakening after the complicated mastoid procedure. He described difficulty focusing his vision and also complained of horizontal diplopia when looking at a distance. Finally, he also noted left-sided pulsatile tinnitus. He was evaluated by the neuro-ophthalmology team, and bilateral papilledema was noted on examination. He underwent magnetic resonance imaging (MRI), which revealed severe right TSS as well as left transverse-sigmoid junction and superior internal jugular vein (IJV) signal dropout, indicating thrombosis (Fig. 1). He was provisionally diagnosed with intracranial hypertension secondary to thrombosis of the left transverse and sigmoid sinus and was treated with acetazolamide for 6 months and warfarin for 1 year, which improved his symptoms. He was eventually tapered off anticoagulation due to improvement of the initial thrombus and symptom control with acetazolamide.

After being off anticoagulation for a year, the patient's symptoms recurred. He underwent repeat MRI, which demonstrated recurrent thrombosis of the left transverse-sigmoid junction, and his warfarin was restarted. He was referred to our center for further evaluation of possible cerebral venous hypertension and candidacy for venous stent intervention, approximately 2 years after the mastoidectomy. He underwent diagnostic cerebral angiography with venous pressure manometry. This demonstrated right TSS due to arachnoid granulations herniating into the right mid-transverse sinus best seen on cone beam computed tomography (CBCT) angiography (Fig. 2), as well as the occluded dominant left transverse-sigmoid junction (Fig. 3). He underwent venous pressure manometry, which demonstrated an elevated superior sagittal sinus and torcular pressure of 36 mm Hg, with measurements of 34 mm Hg in the right medial transverse sinus, 31 mm Hg mid-sinus, and 18 mm Hg at the transverse-sigmoid junction, down to 13 mm Hg and 12 mm Hg in the distal and proximal sigmoid sinus, respectively (Fig. 3A); there was a gradient of 18 mm Hg between the superior sagittal sinus and the transverse-sigmoid junction. It was determined that the patient would qualify for stent angioplasty of the right transverse sinus.

The patient returned for the stent procedure the following month. At the junction between the jugular bulb and the sigmoid sinus, a tight curve was encountered and caused significant resistance to the angioplasty balloon's advancement, although this was able to pass successfully and angioplasty of the right transverse sinus was performed. However, when the 8 × 80–mm Zilver stent (Cook Medical Inc.) was to be advanced through the same area, the system would not advance despite multiple attempts and repositioning of the Shuttle (Cook Medical Inc.) sheaths and guide catheter. Stent placement was aborted, and pressure transduction revealed an improved gradient to 10 mm Hg after the balloon angioplasty. At this time, the procedure was concluded to minimize the risk of iatrogenic injury. On follow-up, the patient reported stable symptoms of headaches, tinnitus, and left-sided neck pain without improvement.

In the subsequent months, our institution adopted a large-bore, 0.088-inch intermediate guide catheter (TracStar catheter, Imperative Care Inc.) for use in venous sinus cases due to its navigability, and it was believed that this would be of use in the acute-angle stenosis located at the patient's sigmoid–jugular bulb junction. The patient returned for a repeat stent placement attempt. This time, a 95-cm TracStar 0.088-inch catheter was primed with Zoom 71 and Zoom 35 catheters (Imperative Care Inc.) along with a Synchro Support microwire (Stryker Corp.). This was advanced via the right transverse sinus into the superior sagittal sinus, with the new catheter successfully advancing over the smaller catheters through the acute angle of the stenotic area into the right mid-transverse sinus. Repeat angioplasty was performed, and an 8 × 80–mm Zilver stent was successfully advanced through the intermediate catheter and then deployed, with the stent spanning from the torcula to the proximal sigmoid sinus

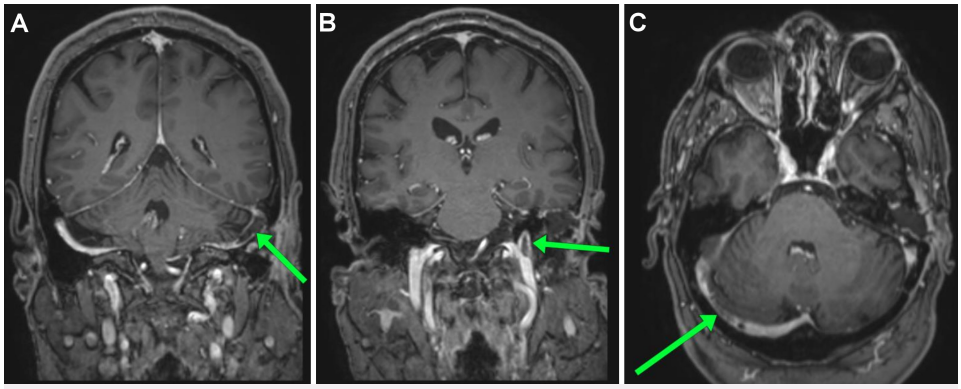

FIG. 1. Brain MRI with and without contrast demonstrating right TSS and left sigmoid and superior internal jugular thrombosis. **A:** Coronal postcontrast T1-weighted MRI scan demonstrating a filling defect in the left sigmoid sinus consistent with a thrombus (*green arrow*). **B:** Coronal postcontrast T1-weighted MRI scan demonstrating a filling defect in the left superior IJV, also consistent with a thrombus (*green arrow*). **C:** Axial postcontrast T1-weighted MRI scan demonstrating a stenotic right transverse sinus (*green arrow*).

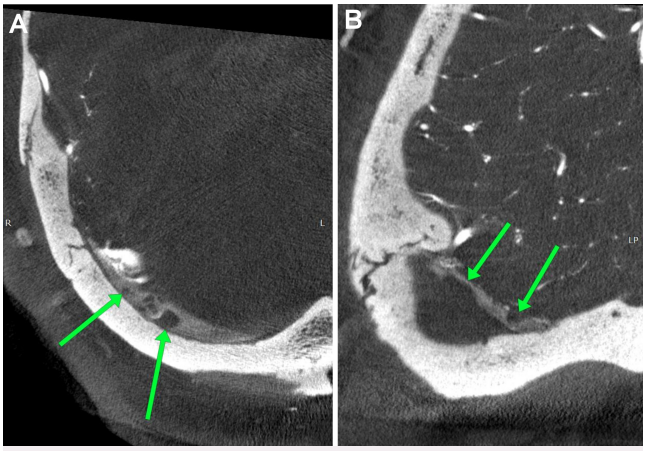

FIG. 2. Axial (**A**) and oblique sagittal (**B**) CBCT angiograms in the venous phase demonstrating severe right TSSs secondary to arachnoid granulations (*arrows*).

(Fig. 4A). The pressure gradient at the end of the procedure was less than 5 mm Hg, with the distal stent pressure measuring 28 mm Hg and the proximal stent measuring 23 mm Hg (there were elevated central pressures relative to the preoperative measurements due to the effect of general anesthesia). The patient recovered without complications.

Two months after stent placement, the patient returned for repeat angiography, and CBCT angiography demonstrated wide patency of the stent without any appreciable stenosis or residual compression from the prior arachnoid granulations (Fig. 4B). Venous pressure measurements were repeated, confirming a persistently improved gradient of 4 mm Hg (22 mm Hg in the distal stent and 18 mm Hg in the proximal stent; see Fig. 4C). On follow-up, the patient reported resolution of left-sided pulsatile tinnitus and improvement of his headaches.

Informed Consent

The necessary informed consent was obtained in this study.

Discussion

The patient's case of cerebral venous hypertension due to an iatrogenic venous outflow tract injury is illustrative of the possible complications of postoperative venous thrombosis, particularly in relation to surgery of the skull base close to the venous sinuses. Thrombosis of the IJV or transverse–sigmoid sinus system can occur secondary to surgeries for cerebellopontine angle approaches (including translabyrinthine, middle fossa, and retrosigmoid approaches) as well as surgical neck dissection, trauma, infection, and procedures such as central venous catheter insertion.[1,2,12–20] More specifically, iatrogenic venous sinus thrombosis can occur after posterior fossa surgery or neck dissection at rates ranging from 0% to almost 35%.[1,2,13–20] It can lead to problems such as dural arteriovenous fistulas (some of which cause pulsatile tinnitus or intracranial hemorrhage), acute or chronic increases in ICP, increased postoperative CSF leakage rate, and an overall increase in the postoperative complication rate. However, the majority of cases are asymptomatic, ranging from 78% to 100% in previous series.[2,16–20] Prior studies on cranial surgery, particularly posterior fossa surgery, have found that postoperative cerebral venous sinus thrombosis (CVST) tends to occur more frequently after translabyrinthine approaches, after surgeries for meningiomas and vestibular schwannomas over other tumor types, in patients with larger tumors and longer operative times, and in cases done on the patient's nondominant venous drainage side.[1,15–21] The latter is hypothesized to occur because of lower flow rates through the nondominant sinuses, increasing the probability of thrombosis. It is debated whether to treat these cases with anticoagulation; given the propensity of anticoagulation to increase complications in cranial surgery as well as the majority of cases of CVST remaining asymptomatic, it is generally not routinely recommended.[16–22]

Postoperative IIH symptomology due to sinus injuries has only been described in a handful of cases and has generally occurred after cranial surgery. Keiper et al. described a series of 107 patients who underwent suboccipital or translabyrinthine approaches for tumors; 5 of these patients (4.6%) developed venous congestive symptoms (4 due to thrombosis and 1 due to postoperative stenosis).[2] All patients were treated with shunting (either ventriculo- or lumboperitoneal

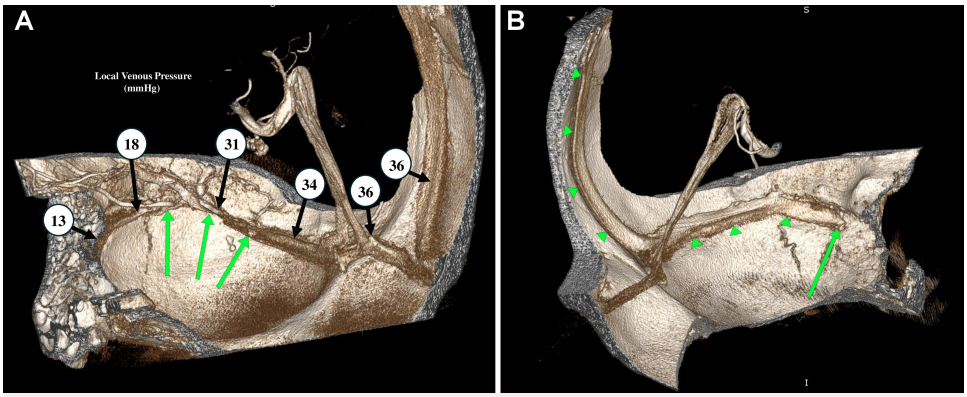

FIG. 3. A: Three-dimensional (3D) reconstruction of the venous CBCT view of the right posterior fossa demonstrating the stenotic areas (*arrows*) of the nondominant right transverse sinus. *Numbers* indicate pressure (in mm Hg) at the indicated locations along the venous sinus system. **B:** 3D reconstruction of the venous CBCT view of the left posterior fossa demonstrating the occluded left transverse-sigmoid junction (*arrow*) with a dominant venous drainage pathway via the superior sagittal sinus and left transverse sinus (*arrowheads*).

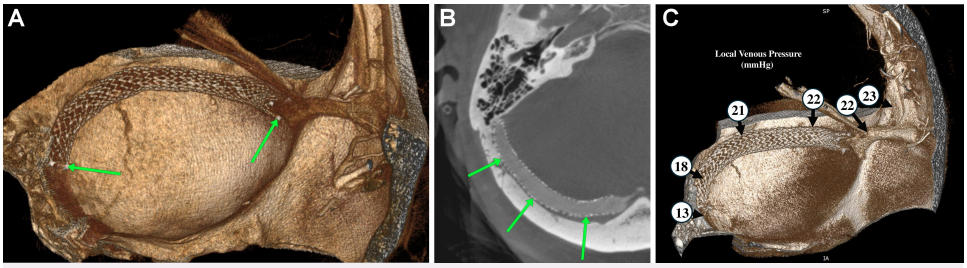

FIG. 4. A: 3D reconstruction of intraprocedural poststenting CBCT angiography in the venous phase showing final Zilver stent placement, spanning from the torcula to the right proximal sigmoid sinus (*arrows*). **B:** Two-month follow-up axial CBCT scan in the venous phase demonstrating wide stent patency without recurrence of arachnoid granulations in area of previous stenoses (*arrows*). **C:** 3D reconstruction of 2-month follow-up axial CBCT angiography in the venous phase showing the stent in position, with follow-up venous pressure measurements after stent placement indicated by the *numbers* (in mm Hg).

shunting) except 1, who responded to medical therapy. Another study by Roberson et al. analyzed more than 3500 cases and identified 6 cases (< 0.17%) of venous insufficiency due to venous sinus sacrifice or thrombosis after neurotological surgeries, with 4 patients developing chronic IIH symptoms and 2 developing acute postoperative ICP issues.[1] The 4 patients with chronic IIH symptoms were treated variably with lumboperitoneal shunting, serial lumbar puncture, or anticoagulation and medical ICP control, while the 2 patients with acute postoperative ICP were treated respectively with emergency venous embolectomy and medical ICP management.

Observations

To our knowledge, this is the first case of iatrogenic injury with subsequent sinus thrombosis and resulting refractory cerebral venous congestive physiology being treated with venous sinus stenting in a similar fashion to de novo cases of venous intracranial hypertension. An important aspect of our patient's case was the use of CBCT and venous pressure manometry to evaluate the anatomy and physiology of the sinus system. CBCT delineated the sinus contralateral to the injury, which demonstrated a specific area of stenotic anatomy that both explained the patient's venous hypertension and may have complicated endovascular intervention. CBCT has been demonstrated to be effective in evaluating detailed anatomy in a number of cerebral vascular disorders, including the identification of arteriovenous malformations (AVMs), particularly for subcentimeter nidi, as well as identification of features that portend high rupture risk or embologenic ulceration in aneurysms and atherosclerotic plaques, and in high-resolution AVM targeting for stereotactic radiosurgery.[23–27] CBCT has been utilized at our institution in venous cases to better evaluate suspected structural causes of cerebral venous hypertension; in this case, it confirmed a suspected area of stenosis on MRI, strengthening the case alongside venous pressure manometry to intervene with stenting. The prior study by Keiper et al. noted that despite seemingly patent venous sinuses contralateral to the relevant surgical thrombosis or injury seen on MRI, some patients still developed signs of cerebral venous hypertension;[2] it is possible that if such cases were to be evaluated by CBCT and pressure manometry, underlying structural changes and a venous pressure gradient might have been revealed. The transition from invasive CSF shunting to minimally invasive venous stenting has been a significant practice shift in patients with IIH secondary to venous intracranial congestion at many centers and will likely also benefit future patients experiencing refractory IIH symptoms from iatrogenic CVST.

More typical cases of cerebral venous hypertension likely originate from a positive feedback loop whereby ICP elevations (often thought to be incited by factors such as obesity and obstructive sleep apnea as well as nocturnal recumbency) cause extramural compression of the venous sinuses, which in certain individuals are more prone to compression and narrowing, particularly in the lateral portions of the transverse sinuses. This narrowing of the sinuses causes increased intramural venous pressures leading to venous congestion, which again increases ICP. This increased ICP can further compress the sinuses, and the cycle repeats until the extramural compression from increased ICP reaches a state of equilibrium with the intramural venous pressures, thus resisting further compression from intracranial compartment pressure.[3,28]

Our patient's case lends interesting evidence to the positive feedback theory of cerebral venous hypertension because it originated at a different point in the feedback loop from those in de novo cases, namely that the inciting event for the feedback loop was the closure of a dominant sinus with thrombosis rather than transient increases in ICP leading to sinus compression. While most cases of cerebral venous hypertension are thought to result first from increases in ICP causing subsequent extramural narrowing of the sinuses, the loss of the patient's dominant transverse sinus combined with a contralateral hypoplastic sinus first caused increased intramural venous pressures and congestion, followed by increases in ICP, which further compressed the sinuses (likely via herniation of an arachnoid granulation) and worsened the existing barriers to venous outflow. The feedback theory of recurrent stenosis further explains the failure of the patient's condition to respond to balloon angioplasty alone, because the existing ICP elevations caused continued extramural pressure on the angioplasty-treated areas, leading to rapid recurrence of the right TSS. This feedback cycle was broken when the stent was successfully placed in the second procedure, allowing venous outflow despite continued extrinsic compression from increased ICP (which is now resisted by the high radial force of the Zilver stent) and reversing venous congestion.[3] Overall, this supports the idea that our patient had been dependent on his dominant left transverse sinus for cranial venous drainage prior to his iatrogenic injury to the sinus, which provoked the positive feedback loop typical of other cranial venous hypertension patients despite his lack of typical risk factors for de novo cerebral venous hypertension.

An important technical aspect of this case was the use of the TracStar large distal platform intermediate catheter and Zoom system,

which has helped to alleviate difficulty with tortuous venous anatomy and improve patient access to the transverse sinus stenting procedure. The catheter has previously been proven useful for recanalization in stroke thrombectomy, providing a large inner diameter and higher catheter-to-vessel ratio during these procedures.[29–32] The 0.088-inch bore catheter is characterized by a soft, flexible distal segment with a beveled tip, paired with a stiff proximal segment, providing greater support for the catheter and devices passed through it. Its distal segment demonstrates improved trackability along the supporting Zoom catheters, allowing the large-bore catheter to make tighter turns and extend further distally compared to similar-sized catheters. It allows passage of the stiff, large-caliber stents we utilize to stent almost the entirety of the transverse sinus from the torcula to the transverse sigmoid junction, which improves rates of stent-adjacent stenosis by locating stent tines in areas known to be less susceptible to compression.[3,7,8,10] Zyck et al. conducted a retrospective multicenter series using this catheter system to deliver various stents to the transverse sinus in 58 patients and were successful in a striking 100% of cases.[5]

Lessons

This case demonstrates a less recognized etiology of intracranial venous hypertension-associated IIH symptoms, which was provoked by iatrogenic injury during skull base surgery and subsequent thrombosis of a patient's dominant cranial venous outflow pathway. This incited a positive feedback loop wherein reduced venous outflow led to venous congestion and subsequent increases in ICP, which caused further extramural compression on the patient's nondominant transverse sinus as the only remaining outflow pathway. This stenosis significantly reduced the navigability of the sinus for intervention, but this problem was addressed with the use of a large intermediate catheter, which was able to traverse the necessary distance by virtue of its flexible distal end with a beveled tip. With a long stent construct successfully in place, the patient's symptoms improved significantly. Cranial venous hypertension should be recognized as a potential complication after sinus thrombosis, particularly after skull base and neck procedures that increase the risk of thrombosis to these structures.

References

1. Roberson JB, Brackmann DE, Fayad JN. Complications of venous insufficiency after neurotologic-skull base surgery. *Am J Otol*. 2000; 21(5):701-705.
2. Keiper GL, Sherman JD, Tomsick TA, Tew JM. Dural sinus thrombosis and pseudotumor cerebri: unexpected complications of suboccipital craniotomy and translabyrinthine craniectomy. *J Neurosurg*. 1999;91(2):192-197.
3. Fargen KM. A unifying theory explaining venous sinus stenosis and recurrent stenosis following venous sinus stenting in patients with idiopathic intracranial hypertension. *J NeuroIntervent Surg*. 2021; 13(7):587-592.
4. Abdalkader M, Hui F, Amans MR, et al. Cerebral venous disorders: diagnosis and endovascular management. *J Neuroradiol*. 2023; 50(6):581-592.
5. Zyck S, Malik M, Webb M, et al. Technical and clinical success after venous sinus stenting for treatment of idiopathic intracranial hypertension using a novel guide catheter for access: case series and initial multi-center experience. *Interv Neuroradiol*. 2024;30(4): 524-528.
6. Kulhari A, He M, Fourcand F, et al. Safety and clinical outcomes after transverse venous sinus stenting for treatment of refractory idiopathic intracranial hypertension: single center experience. *J Vasc Interv Neurol*. 2020;11(1):6-12.
7. Saber H, Lewis W, Sadeghi M, Rajah G, Narayanan S. Stent survival and stent-adjacent stenosis rates following venous sinus stenting for idiopathic intracranial hypertension: a systematic review and meta-analysis. *Interv Neurol*. 2018;7(6):490-500.
8. Kumpe DA, Seinfeld J, Huang X, et al. Dural sinus stenting for idiopathic intracranial hypertension: factors associated with hemodynamic failure and management with extended stenting. *J Neurointerv Surg*. 2017;9(9):867-874.
9. Winters HS, Parker G, Halmagyi GM, Mehta A, Atkins T. Delayed relapse in pseudotumor cerebri due to new stenosis after transverse sinus stenting. *BMJ Case Rep*. 2015;2015:bcr2015011896.
10. Ahmed RM, Wilkinson M, Parker GD, et al. Transverse sinus stenting for idiopathic intracranial hypertension: a review of 52 patients and of model predictions. *AJNR Am J Neuroradiol*. 2011;32(8): 1408-1414.
11. Starke RM, Wang T, Ding D, et al. Endovascular treatment of venous sinus stenosis in idiopathic intracranial hypertension: complications, neurological outcomes, and radiographic results. *Sci World J*. 2015;2015:1-8.
12. Kitano D, Yonezawa K, Iwae S, Sakakibara S. Internal jugular vein thrombosis and pulmonary thromboembolism after head and neck reconstructive surgery. *J Plast Reconstr Aesthet Surg*. 2021;74(6): 1239-1245.
13. Motiee-Langroudi M, Amali A, Saedi B, et al. Internal jugular vein thrombosis following different types of neck dissection. *Iran J Otorhinolaryngol*. 2020;32(113):343-347.
14. Lozouet M, Gilard V, Nassihi A, Marie JP, Derrey S. Ruptured dural arteriovenous fistula and sinus venous thrombosis following surgical resection of a vestibular schwannoma: case report and review of the literature. *Neurochirurgie*. 2022;68(6):688-692.
15. Li PMMC, Fischbein NJ, Do HM, Blevins NH. Dural arteriovenous fistula following translabyrinthine resection of cerebellopontine angle tumors: report of two cases. *Skull Base Rep*. 2011;1(1): 51-58.
16. Jean WC, Felbaum DR, Stemer AB, Hoa M, Kim HJ. Venous sinus compromise after pre-sigmoid, transpetrosal approach for skull base tumors: a study on the asymptomatic incidence and report of a rare dural arteriovenous fistula as symptomatic manifestation. *J Clin Neurosci*. 2017;39:114-117.
17. Orlev A, Jackson CM, Luksik A, et al. Natural history of untreated transverse/sigmoid sinus thrombosis following posterior fossa surgery: case series and literature review. *Oper Neurosurg (Hagerstown)*. 2020;19(2):109-116.
18. Benjamin CG, Sen RD, Golfinos JG, et al. Postoperative cerebral venous sinus thrombosis in the setting of surgery adjacent to the major dural venous sinuses. *J Neurosurg*. 2019;131(4):1317-1323.
19. Sturiale CL, Auricchio AM, Valente I, et al. Post-operative segmental cerebral venous sinus thrombosis: risk factors, clinical implications, and therapeutic considerations. *Neurosurg Rev*. 2023;46(1): 161.
20. Brahimaj BC, Beer-Furlan A, Crawford F, et al. Dural venous sinus thrombosis after vestibular schwannoma surgery: the anticoagulation Dilemma. *J Neurol Surg B Skull Base*. 2021;82(suppl 3):e3-e8.
21. Ohata K, Haque M, Morino M, et al. Occlusion of the sigmoid sinus after surgery via the presigmoidal-transpetrosal approach. *J Neurosurg*. 1998;89(4):575-584.
22. Matsushima K, Kohno M, Tanaka Y, Nakajima N, Ichimasu N. Management of sigmoid sinus injury: retrospective study of 450 consecutive surgeries in the cerebellopontine angle and intrapetrous region. *Oper Neurosurg (Hagerstown)*. 2020;19(6):721-729.
23. Heller RS, Malek AM. Successful detection of embologenic ulceration in a symptomatic non-hemodynamic intracranial stenosis using C-arm cone beam CT. *J NeuroInterv Surg*. 2013;5(2):e3.
24. Lauric A, Heller RS, Schimansky S, Malek AM. Benefit of cone-beam CT angiography in visualizing aneurysm shape and identification of exact rupture site. *J Neuroimaging*. 2015;25(1):56-61.

25. Rahal JP, Malek AM. Benefit of cone-beam computed tomography angiography in acute management of angiographically undetectable ruptured arteriovenous malformations. *J Neurosurg*. 2013; 119(4):1015-1020.
26. Safain MG, Rahal JP, Raval A, et al. Use of cone-beam computed tomography angiography in planning for gamma knife radiosurgery for arteriovenous malformations: a case series and early report. *Neurosurgery*. 2014;74(6):682-696.
27. Safain MG, Rahal JP, Patel S, Lauric A, Feldmann E, Malek AM. Superior performance of cone-beam CT angiography in characterization of intracranial atherosclerosis. *J Neurosurg*. 2014;121(2): 441-449.
28. Coffman SA, Singh J, Wolfe S, Fargen KM. Unexpected occlusion of the contralateral transverse sinus after stenting for idiopathic intracranial hypertension. *Interv Neuroradiol*. 2018;24(6):718-721.
29. Zakeri A, Schreiber C, Shah V, et al. Utility of the novel guide catheter in mechanical thrombectomy for emergent large vessel occlusion stroke. *Interv Neuroradiol*. 2024;30(3):336-341.
30. Bageac DV, Gershon BS, Shigematsu T, Majidi S, De Leacy RA. Early institutional experience using the TracStar Large Distal Platform in endovascular flow diversion. *Neuroradiol J*. 2022;35(3): 313-318.
31. Bageac DV, Gershon BS, Vargas J, et al. Comparative study of intracranial access in thrombectomy using next generation 0.088 inch guide catheter technology. *J Neurointerv Surg*. 2022;14(4): 390-396.
32. Nogueira RG, Mohammaden MH, Al-Bayati AR, Frankel MR, Haussen DC. Preliminary experience with 088 large bore intracranial catheters during stroke thrombectomy. *Interv Neuroradiol*. 2021; 27(3):427-433.

Disclosures

Dr. Malek reported being a founder, shareholder, investor, and consultant to CereVasc Inc.

Author Contributions

Conception and design: Malek, Snyder. Acquisition of data: Malek, Snyder. Analysis and interpretation of data: Malek, Snyder. Drafting the article: Snyder. Critically revising the article: all authors. Reviewed submitted version of manuscript: Malek, Snyder. Approved the final version of the manuscript on behalf of all authors: Malek. Administrative/technical/material support: Heilman.

Correspondence

Adel M. Malek: Tufts Medical Center, Boston, MA. adel.malek@tuftsmedicine.org.