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The impact of stent length on stent-adjacent stenosis following transverse sinus stenting in idiopathic intracranial hypertension

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OBJECTIVE Cerebral venous sinus stenting (VSS) is an established treatment for transverse sinus (TS) stenosis associated with idiopathic intracranial hypertension (IIH). The authors' early practice favored shorter stents that only spanned the focal narrowing to reduce the metal burden. However, stent-adjacent stenosis (SAS) emerged as a significant failure mode prompting the use of longer constructs. In this study, the authors sought to compare the incidence of SAS, clinical outcomes, and angiographic findings between shorter and full-length stent constructs.

METHODS The authors conducted a retrospective analysis of patients with IIH who underwent TS stenting at Tufts Medical Center in the period from 2013 to 2023. Patients with medically refractory IIH symptoms and a significant cerebral venous pressure gradient were included. Symptom presentation, neuroimaging, venous manometry, preoperative and follow-up angiography, and clinical examination findings were recorded and analyzed across two categories of stent length: shorter (40 or 60 mm) and full length (80 mm). SAS was defined as de novo narrowing of the TS contiguous with the stent edge.

RESULTS Endovascular treatment was successful in all 60 patients included in the study (48 females, mean patient age of 39 years). Three patients were treated with 40-mm stents, 13 with 60-mm stents, and 44 with full-length 80-mm stents. SAS was documented in 8 patients (13%) and was significantly more frequent ($p < 0.01$) among those with shorter stents (6/16), with 3 patients requiring reintervention using a second overlapping stent. Among the 44 patients with full-length constructs, 2 demonstrated SAS at the residual unstented TS lateral to the torcular Herophili. After a mean follow-up of 40 months, 29 patients (48%) reported persistent symptoms, although there was no significant difference in this rate between the stent groups. Full-length constructs demonstrated a more favorable postoperative reduction in TS pressure gradients ($p < 0.01$) and lower gradients on follow-up ($p < 0.05$).

CONCLUSIONS SAS is a clinically relevant complication following VSS in patients with IIH and may contribute to persistent symptoms and the need for reintervention. Full-length stent constructs extending from the sigmoid sinus to the torcula significantly reduce the incidence of SAS and are associated with better hemodynamic outcomes following VSS.

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KEYWORDS idiopathic intracranial hypertension; transverse sinus stenting; stent length; stent-adjacent stenosis

Cerebral venous outflow obstruction is significantly implicated in the pathophysiology of idiopathic intracranial hypertension (IIH). Unilateral or bilateral transverse sinus (TS) stenosis has been found in 30%–90% of patients with IIH.[1] The specific role of TS stenosis as a cause or effect of IIH remains unclear, however.[2–4] Nonetheless, cerebral venous sinus stenting (VSS) is an endovascular therapy that has been adopted to relieve focal stenosis and alleviate elevated intracranial pressure (ICP).[5] Surgical interventions are reserved for patients with persistent symptoms despite maximal medical therapy including weight loss, carbonic anhydrase inhibitors, and repeated lumbar punctures.[6] Other surgical interventions, such as optic nerve sheath fenestration and ventriculoperitoneal or lumboperitoneal CSF shunting, have greater risks, are less effective, and have higher treatment failure rates than VSS.[1,7]

Initially, given our center's experience with VSS, we preferred shorter stents (40–60 mm) with the goal of treating only the most visibly stenotic portion of the TS (Fig.

ABBREVIATIONS CBCT = cone-beam CT; DAPT = dual antiplatelet therapy; ICP = intracranial pressure; IIH = idiopathic intracranial hypertension; IJV = internal jugular vein; SAS = stent-adjacent stenosis; TS = transverse sinus; VSS = venous sinus stenting.

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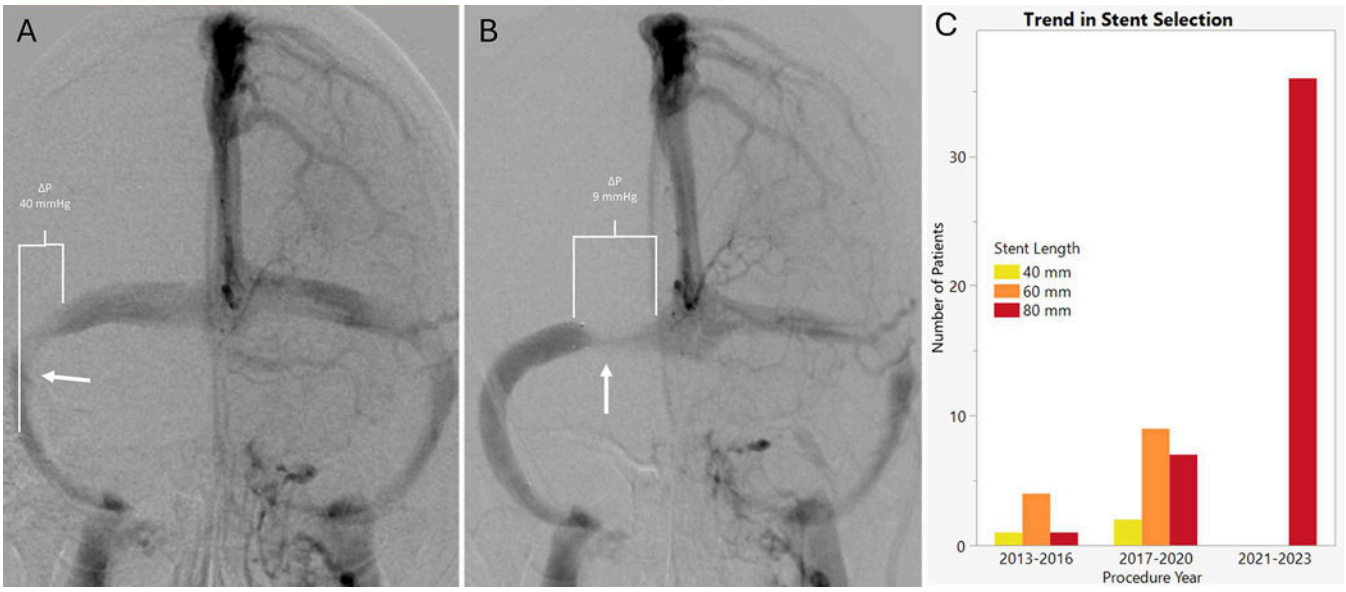


FIG. 1. A: DSA study from a patient with IIH depicting right TS stenosis (*arrow*) with an associated transstenotic pressure gradient. This patient reported headaches, visual obscurations, pulsatile tinnitus, and papilledema. **B:** DSA study obtained after TS stenting with a 60-mm Cook Medical Zilver stent, showing wide patency of the stented sinus, with marked narrowing (*arrow*) adjacent to the distal end of the stent and a significant pressure gradient. **C:** Trends in stent selection for VSS over a 10-year period at our center. Currently, only full-length 80-mm stents are used for VSS at our center.

1A and B) and to minimize the metal burden.[8] However, as we accumulated follow-up data, stent-adjacent stenosis (SAS) became apparent as a failure mode of VSS. This narrowing of the TS, seen past the distal edges of stents (Fig. 1B), often coincides with symptom recurrence and elevated venous pressure gradients, requiring additional management and possible surgical reintervention.[9–11] The etiology of such restenosis is incompletely understood and likely multifactorial. These observations at our center prompted a shift toward deploying longer (80 mm) stent constructs that extend well beyond the focal lesion to mitigate restenosis, as they encompass the entire at-risk segment of the TS, bridging the torcular Herophili to the sigmoid sinus (Fig. 1C).

In this study, we describe our single-center experience with SAS through a series of patients who underwent VSS for medically refractory IIH. We investigated and compared the incidence of SAS, symptom recurrence, and angiographic outcomes between patients with partial coverage via 40- or 60-mm stents and those with full-length 80-mm stents spanning the entire transverse–sigmoid sinus. Furthermore, we discuss possible mechanisms of restenosis and risk factors that arise from VSS.

Methods

Patients

This retrospective study included patients with IIH who underwent transverse–sigmoid sinus stenting at Tufts Medical Center in the period from 2013 to 2023. The diagnosis of IIH in our patient population was largely based on the modified Dandy criteria.[12] IIH clinical signs and symptoms included papilledema, headache, transient visual obscurations, diplopia due to sixth cranial nerve palsy, and pulse synchronous tinnitus. Patients with an identifiable cause of intracranial hypertension such as mass lesions, cerebral edema, or CSF abnormalities such as hydrocephalus were excluded from this series. Patients without available follow-up angiograms were also excluded.

Indications for endovascular treatment included 1) persistent or progressive symptoms despite maximally tolerated medical therapy or intolerance to the side effects of carbonic anhydrase inhibitors, 2) a dural venous pressure gradient ≥ 7 mm Hg, and 3) the presence of stenotic pathology in the dominant sinus confirmed on imaging or angiographic studies.

Cerebral VSS

All patients suspected to have IIH associated with venous sinus stenosis were first evaluated with DSA, including venous phase cone-beam CT (CBCT) of the bilateral carotid circulation, as well as microcatheter-mediated venous pressure manometry. Those with findings confirming angioplasty candidacy returned for cerebral VSS electively. Patients were pretreated with dual antiplatelet therapy (DAPT) using aspirin 81 mg and either clopidogrel 75 mg or prasugrel 10 mg, daily for 1 week prior to stent placement.

Routine VSS practice at our center involves performing balloon angioplasty across the stenotic region of the TS before stent deployment across that segment. All procedures were performed under general anesthesia, and heparin was administered to achieve an activated clotting time goal of ≥ 220 seconds. The dominant sinus was chosen for stent placement to restore the primary venous outflow pathway. Stent sizing varied on a case-by-case basis. As mentioned above, our center's practice evolved from using

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shorter stents that only spanned the focal stenosis. Currently, all patients undergoing VSS receive full-length 80-mm stents (Fig. 1C). The stent diameter (6, 8, or 10 mm) is tailored to minimize incomplete stent apposition to the sinus walls based on the luminal diameter near the torcula and sigmoid sinus, as measured on CBCT venography.

A 4-Fr Berenstein catheter was used initially to access the target internal jugular vein (IJV), and a stiff exchange guidewire (Glidewire, Terumo Neuro) was used to replace the Berenstein with a stiff guide catheter. A support catheter was then positioned in the superior IJV, and a microcatheter and microwire were extended to the mid–superior sagittal sinus. The microwire was then replaced with a V18 stiff exchange wire (Boston Scientific Corp.) to facilitate angioplasty and stent placement. Balloon angioplasty of the focal venous stenosis was performed with a rapid exchange 6 × 40–mm Sterling balloon (Boston Scientific Corp.), followed by stenting with a Zilver 518 self-expanding stent (Cook Medical) or, in a single case, with a Precise Pro stent (Cordis).[13] Poststenting imaging and venous manometry were performed intraoperatively to confirm good apposition of the stent to the sinus walls, resolution of TS stenosis, and a decrease in the associated pressure gradient.

Postprocedure

All patients underwent contrast-enhanced MRI on postoperative day 1 to establish a baseline and rule out ischemic complications. Regular follow-up for VSS at our center includes contrast MRI and MR venography (MRV), followed by DSA with venous manometry at approximately 3 months postprocedure and subsequent contrast MRI and MRV at 6 and 12 months. Patients were weaned off DAPT after the first satisfactory DSA evaluation and continued to take aspirin 81 mg. Patients with persistent symptoms on follow-up such as headaches, visual obscurations, and pulsatile tinnitus were assessed with DSA and manometry to rule out stent-related complications. Those with recurrent stenosis and/or an elevated TS pressure gradient were considered for reintervention with a contralateral stent or extension of the original construct.

Data Analysis

Patient characteristics and data including clinical presentation and pertinent history were collected, along with neuroimaging and angiographic studies before, during, and after VSS. This was done in accordance with the Tufts Medical Center Institutional Review Board guidelines. Statistical analysis was performed using JMP Pro 17 software (SAS Institute Inc.). Two-sided p values < 0.05 were deemed statistically significant.

Results

Sixty patients were included in the study, 48 of whom (80%) were female. The mean age was 39 years (range 12–65 years). Forty-one patients had a BMI > 30. Table 1 includes a summary of all patient characteristics.

Clinical manifestations of IIH included headaches in all 60 patients, papilledema in 46 (77%), pulsatile tinnitus in 42 (70%), and visual deficits in 50 (83%). Lumbar punctures were performed in 45 patients, 44 of whom had opening pressures ≥ 24 cm H_2O (range 14–55 cm H_2O). TS stenosis was found on neuroimaging in all patients, as were unabated symptoms with conventional medical therapy. Such findings were followed by DSA with venous pressure manometry, which demonstrated a transstenosis pressure gradient ≥ 7 mm Hg in 59 patients. One patient had previously undergone bilateral optic nerve sheath fenestration; 1 patient, coil embolization for a dural arteriovenous fistula; and another patient, occipital decompression for a Chiari malformation type I.

Stenting of the TS was successfully performed in all 60 patients. Fifty-one patients underwent right-sided TS treatment and 9 had left-sided treatment. Three patients were treated with 40-mm stents, 13 with 60-mm stents, and 44 with 80-mm stents spanning the full length of the TS (Fig. 1C). At a median of 4 months, follow-up examinations were performed, and subsequent or earlier follow-up DSA studies were tailored to patient symptomatology. Angiographic follow-up with venous manometry was performed in 54 patients (90%). Extension of the construct with a second overlapping stent was required in 3 patients due to SAS and unresolved symptoms. Furthermore, another 3 patients subsequently underwent stenting of the contralateral sinus due to an inadequate pressure reduction or persistent symptoms. The mean follow-up among all patients was 40 months postprocedure.

Stent-Adjacent Stenosis

SAS distal to the implanted stent was observed in 8 patients, 6 of whom had shorter stent constructs (60 or 40 mm) and 2 with full-length stents (80 mm). This corresponded to a significant reduction ($p = 0.0032$) in the occurrence of SAS in full-length versus shorter constructs (Fig. 2A). Among the patients treated with 80-mm stents, SAS was observed only in those with wider stent constructs (10-mm diameter), and this observation indicated a significant difference ($p = 0.015$) in SAS incidence in full-length stents with larger diameters. However, this association was not observed in the patients with shorter stents, as 4 of 7 patients with narrow stents (≤ 8-mm diameter) exhibited SAS compared to 2 of 9 patients with wider stents (10-mm diameter; Fig. 2B).

SAS in the cohort with shorter stents appeared to be a result of extrinsic compression (Fig. 3A and B) in all except 1 patient, who demonstrated intrinsic SAS due to a herniated arachnoid granulation (Fig. 4A and B). As mentioned previously, SAS was treated in 3 of these patients through reintervention to extend the stent construct with a second overlapping stent that extended to the torcula (Figs. 3C and 4C). In the cohort with full-length stents, SAS was observed at the unstented region of the TS between the distal end of the stent and the torcula (Fig. 5). These patients were not considered for stent construct extension because of the manageable nature of their symptoms.

Clinical and Angiographic Outcomes

Clinical follow-up examinations were performed in all patients, during which 29 patients (48%) reported no improvement or a recurrence of symptoms. Headaches re-

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TABLE 1. Summary of patient characteristics categorized by stent length

Parameter	Stent Length			Total
	40 mm	60 mm	80 mm	
No. of pts	3	13	44	60
Treatment yrs	2013–2019	2013–2019	2016–2023	2013–2023
FU in mos	71 ± 47 (29–122)	67 ± 43.4 (6–140)	30 ± 15.4 (2–61)	40 ± 30.4 (2–140)
Age in yrs	47 ± 15 (32–62)	34 ± 11.6 (12–57)	39 ± 12.5 (17–65)	39 ± 12.5 (12–65)
Sex				
M	1	4	7	12
F	2	9	37	48
Race				
White, non-Hispanic	3	10	31	44
Black or African American	0	1	9	10
Hispanic or Spanish origin	0	2	4	6
BMI	32 ± 2.9 (28–34)	34 ± 5.4 (22–41)	34 ± 7.6 (18–47)	34 ± 7 (18–47)
Underweight	0	0	1	1
Normal	0	1	4	5
Overweight	1	2	10	13
Obese, WHO class 1	2	5	11	18
Obese, WHO class 2	0	3	7	10
Obese, WHO class 3	0	2	11	13
TSPG before stenting in mm Hg	23 ± 13.5 (9–36)	17 ± 9.1 (8–40)	18 ± 10.4 (2–60)	18 ± 10.1 (2–60)
LP opening pressure in cm H_2O	33 ± 6 (27–39)	35 ± 8.5 (27–55)	34 ± 9 (14–55)	34 ± 8.6 (14–55)
Stenting side				
Rt	2	11	38	51
Lt	1	2	6	9
Sxs on presentation				
Papilledema	2	10	34	46
Headache	3	13	44	60
Tinnitus	2	7	33	42
Visual deficits	3	11	36	50
Sx recurrence after VSS	3	7	19	29
Stent construct extended	1	2	0	3

FU = follow-up; LP = lumbar puncture; pt = patient; Sx = symptom; TSPG = transstenosis pressure gradient; WHO = World Health Organization.
Values are expressed as number of patients or mean ± standard deviation (range), unless indicated otherwise.

mained or recurred in 21 patients (35%), pulsatile tinnitus in 17 (28%), and visual obscurations in 14 (23%). Papilledema, however, was improved or resolved in all but 1 patient, who reported papilledema 10 years after initial stenting. In all patients with SAS, papilledema was noted as resolved or stable optic atrophy after initial stenting. Overall symptom persistence did not vary significantly across the different stent lengths (Fig. 6A).

Elevated TS pressure gradients were largely relieved by VSS, as confirmed intraoperatively by poststenting manometry; the length of the initial stent construct did not impact this measurement (Fig. 6B). However, on follow-up DSA and manometry, the TS pressure gradient was significantly higher ($p = 0.025$) in the cohort with shorter stents. This represented a significant difference ($p = 0.009$) in the TS pressure gradient reduction postoperatively between patients with full-length and those with shorter stents. The preprocedural TS pressure gradient was found to be higher, though not statistically significant, in patients who developed SAS ($p = 0.07$).

Discussion

SAS is defined as dural venous sinus stenosis developing adjacent to the stent construct following endovascular placement for the treatment of IIH. This stenosis occurs exclusively distal to the stent construct, usually in the segment of the TS just lateral to the torcula. This phenomenon has been described in 11% of cases in one review, of which 52% underwent further stenting;[11] another review found the incidence of SAS to be 14%.[14] Nonetheless, no previous study has investigated the role of stent construct length in the development of SAS. In our cohort of 60 patients, 8 (13%) developed SAS, which aligns with previous observations. Three of these patients with SAS (38%) underwent reintervention for stent construct extension; the

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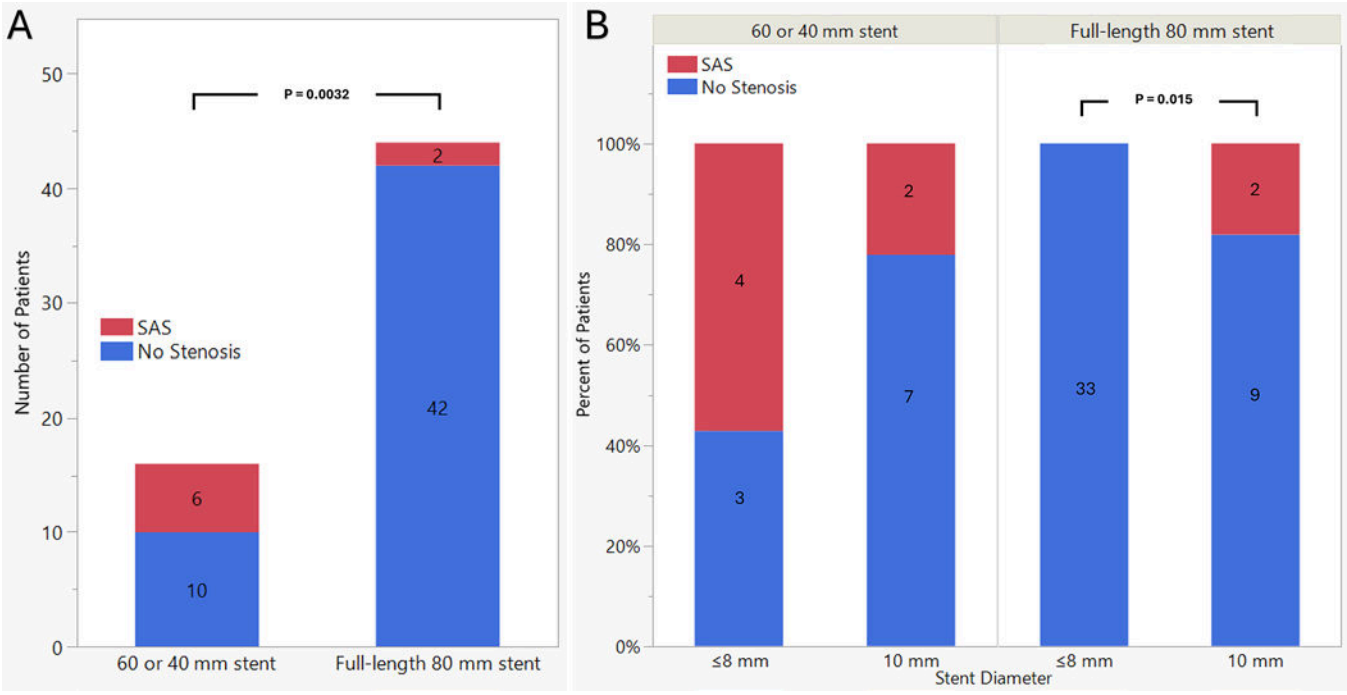


FIG. 2. A: Graph showing that full-length stents (80 mm) had a significantly lower rate of SAS development than shorter stent constructs ($p < 0.01$). **B:** Graph revealing that among patients with full-length stents, SAS occurred only in those with a wider (10 mm) stent diameter ($p < 0.05$). There was no significant difference in the incidence of SAS associated with stent diameter in the patients with shorter stents.

other patients with SAS had manageable symptomatology or minor TS pressure gradients. Along with the 3 patients who underwent contralateral TS stenting, the reintervention rate following cerebral VSS in our cohort was 10%, which is similar to previous studies.[15,16] Although VSS has been shown to be a superior endovascular treatment option for IIH,[1,7] phenomena such as SAS may contribute to a high rate of symptom persistence and recurrence, which was noted in up to 22% of patients in one review.[6] Our study had a higher rate, with 48% (29 patients) having recurrent or persistent symptoms on follow-up.

The mechanism of SAS is not fully understood and is likely multifactorial. Raper et al. suggested that the pattern of pressure gradient alterations after VSS for IIH might be predictive of SAS.[17] They proposed a classification mechanism based on how distal and proximal mean venous pres-

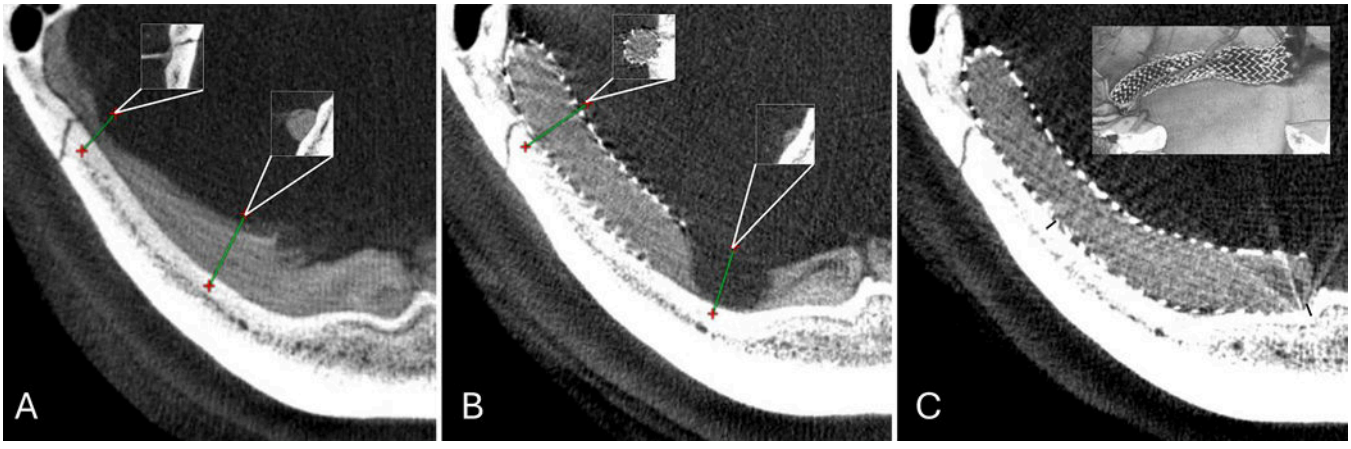


FIG. 3. A: Axial CBCT reconstruction of the right TS in a patient with TS stenosis. The cross-sectional area of the stenosed lateral TS (**left inset**) is 86% narrower than the patent medial sinus (**right inset**). **B:** This was treated with a 60-mm Cook Zilver stent, which resolved the focal stenosis (**left inset**). However, follow-up CBCT demonstrated marked SAS past the distal edge of the stent construct (**right inset**). **C:** In this case, placement of a second overlapping construct (ends denoted by *black dashes* and seen on 3D CBCT reconstruction [**inset**]) that extended to the torcula was required. Reintervention led to resolution of the pressure gradient and an improvement of symptoms.

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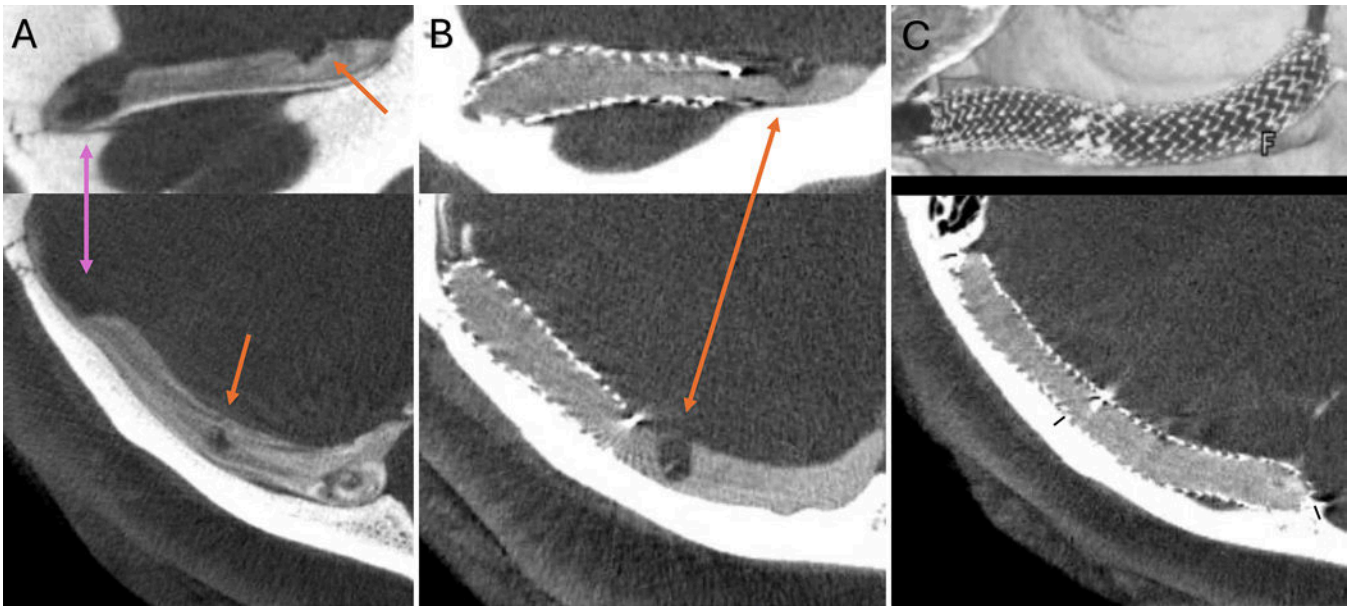


FIG. 4. A: A large herniated arachnoid granulation (*double-sided pink arrow*) causing severe narrowing of the right proximal TS and a 36–mm Hg gradient on coronal (*upper*) and axial (*lower*) CBCT images. Note a minor arachnoid granulation in the distal TS (*orange arrows*). **B:** Coronal (*upper*) and axial (*lower*) CBCT images obtained after stenting with a 40-mm construct, depicting intrinsic SAS. Outpouching of the previously noted arachnoid granulation (*double-sided orange arrow*) resulted in a 30–mm Hg gradient. **C:** This was managed with an overlapping stent (ends denoted by *black dashes*) extending to the torcula, as seen on an axial CBCT (*lower*) and 3D reconstruction (*upper*). This patient continues to experience symptoms.

sures in the TS resolved after VSS and noted a significant correlation with SAS in the groups in which proximal (sigmoid sinus) pressures increased after stenting. Our study, however, did not reflect this pattern, with no significant difference in the incidence of SAS across the resolution types described in Raper et al. (Fig. 7A). Furthermore, the patients who developed SAS in our study had higher preprocedural TS pressure gradients, although the difference between these patients and those who did not develop SAS was not statistically significant ($p = 0.07$). A previous study found that lumbar puncture opening pressures were not only correlated with venous sinus pressures and TS pressure gradients, but also predicted VSS failure, of which SAS was one criterion.[15] TS pressure gradients did not independently predict treatment failure, but this brings into question the combined role of ICP and venous sinus

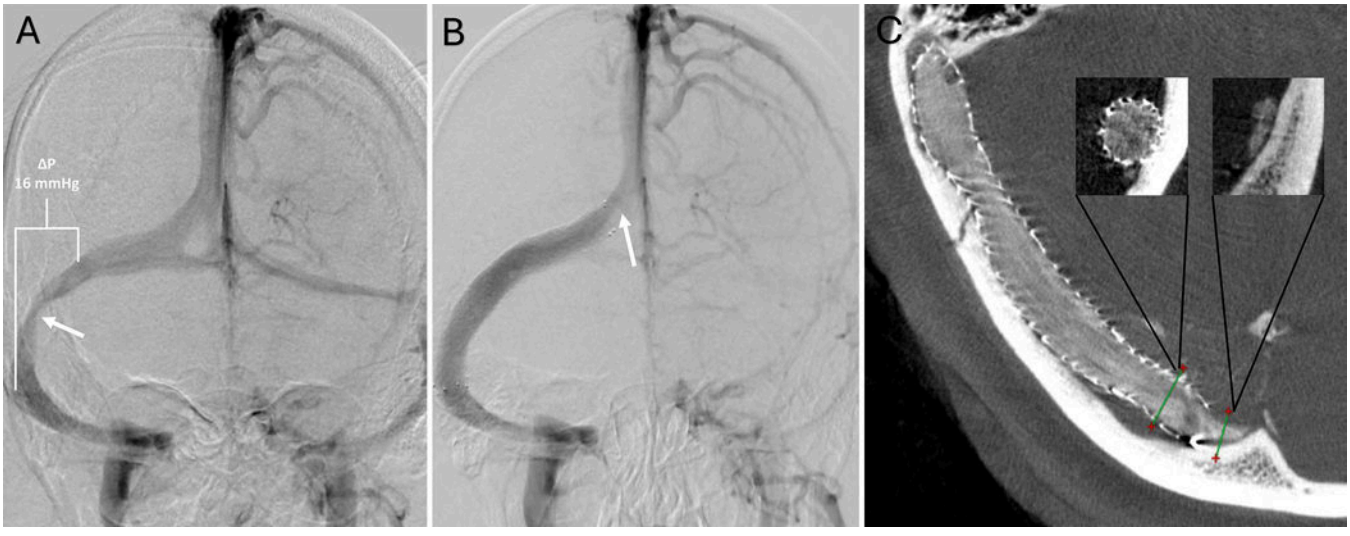


FIG. 5. A: DSA study in an patient with IIH and right TS stenosis (*arrow*) associated with a 16–mm Hg pressure gradient. **B:** DSA study obtained poststenting with an 80 × 10–mm construct, depicting SAS distal to the stent edge (*arrow*). Note that the stent is terminated lateral to the torcula, exposing a susceptible region of the TS. **C:** Axial CBCT study in the same patient, demonstrating a widely patent stent (**right inset**) with immediate cinching at the unstented segment of the medial TS (**left inset**).

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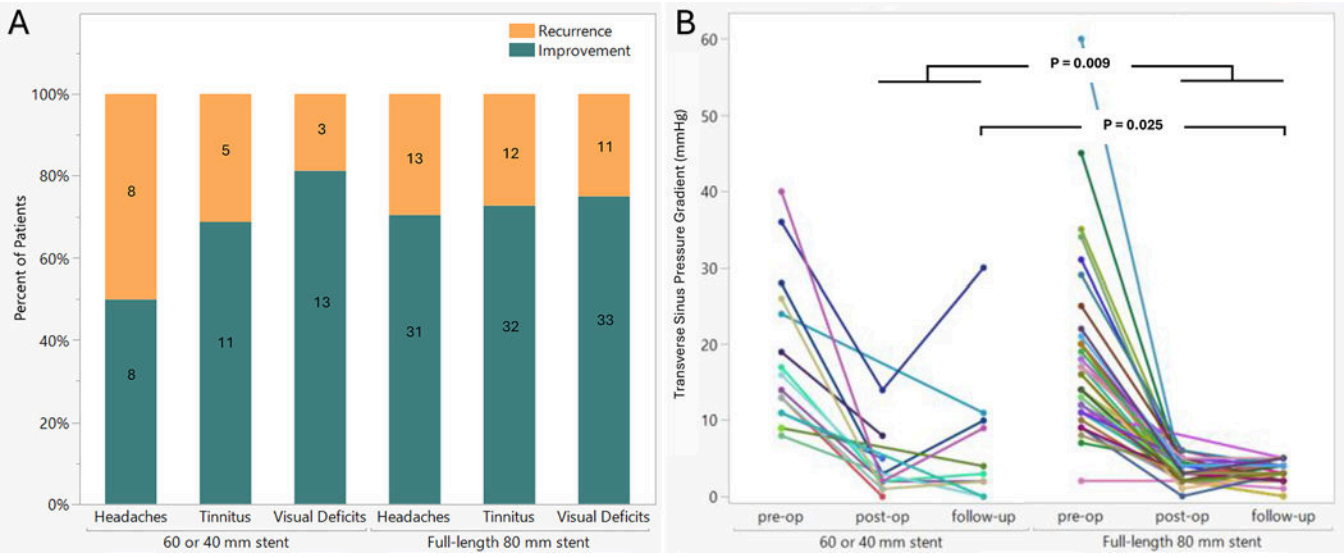


FIG. 6. A: Graph showing that on clinical follow-up, no significant difference in recurrence rates of headache, pulsatile tinnitus, or visual deficits was found between patients with full-length stents and those with shorter stents. **B:** Graph showing resolution of the pressure gradient in all patients after TS stenting, without a significant difference between stent lengths. However, shorter stents were less effective in maintaining the pressure gradient reduction postoperatively ($p < 0.01$) and demonstrated higher gradients on follow-up ($p < 0.05$).

pressures in restenosis of the TS. To understand this, we can refer to the Starling-like resistor mechanism described in several previous studies on IIH.[18] This model posits the existence of a stenotic equilibrium where the increase in venous pressures distal to stenosis maintains sinus patency through a reduction of the transluminal pressure across the sinus wall (Fig. 7B).[4,19] The introduction of a stent reduces distal venous pressure, inducing a new positive feedback loop that results in both upstream stenosis at an unstented, compressible region of the TS and recurrent ICP elevation.[3,20]

It has been suggested that the lateral TS is the most susceptible to extramural compression (up to 60% of patients with IIH exhibit stenosis at this location), followed by the medial TS. The torcula, at the confluence of sinuses, and the sigmoid sinus, encased by bone, seem to be rigid and incompressible.[21,22] Thus, studies have counseled against terminating the stent construct in a susceptible region of the TS to avoid SAS.[21,23] These assumptions regarding the mechanical characteristics of the dural sinuses, along with other previous assumptions, are largely theoretical and therefore must be verified through cadaveric or modeling

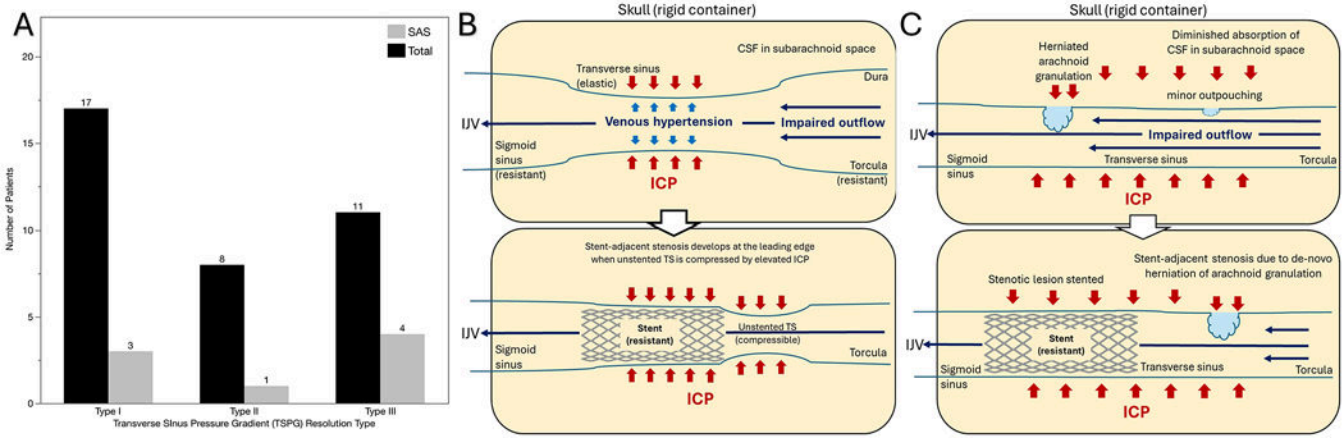


FIG. 7. A: The incidence of SAS did not differ significantly across TS pressure gradient resolution types described by Raper et al.[17] Type I denotes a decrease in distal (near the torcula) mean venous pressure (MVP) after VSS; type II, an increase in proximal (sigmoid sinus) MVP after VSS; and type III, a decrease in distal MVP combined with an increase in proximal MVP. **B:** Schematic of the Starling-like resistor mechanism for TS stenosis (*upper*). An unstented part of the TS is susceptible to compression by heightened transluminal pressure, resulting in SAS (*lower*). **C:** Schematic of an intrinsic mechanism for TS stenosis due to a herniated arachnoid granulation (*upper*). Stenting of the lesion causes an outpouching of a previously undetected arachnoid granulation, which results in SAS (*lower*). Panels B and C adapted from BioRender. V, D. (2025) https://BioRender.com/h4k0xj1.

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studies. Nonetheless, our findings in this retrospective single-center study are, to our knowledge, the first to reveal the benefits of longer stent constructs in terms of having a statistically lower chance of developing SAS (Fig. 2A). Longer stents that span the entire length from the sigmoid sinus to the torcula ensure that no segment of the TS is susceptible to extramural compression. Moreover, SAS in patients with 80-mm stents occurred in the distal TS, as the stent construct was terminated lateral to the torcula (Fig. 5B and C). The decreased incidence of SAS using full-length stents corresponded to lower pressure gradients on follow-up (Fig. 6B), a reduced need for reintervention, and better symptom relief, the latter not significantly so (Fig. 6A). These benefits greatly outweigh the drawback of a higher metal burden for the patient, and our center now exclusively uses the full-length 80-mm stents in VSS for patients with IIH.

Additionally, an oversized cylindrical stent can lead to sinus stretching, recoil cinching, and SAS. Dural venous sinuses are noncylindrical fibrous vessels with variable luminal caliber inherent to their collapsible nature.[24] The reported mean diameter of the TS is around 6 mm and is significantly narrower in patients with IIH.[25] A study has found that wider stents (> 6 mm in diameter) predicted de novo stenosis with an OR > 7.[26] This aligns with our experience, albeit only with the full-length stents, as SAS only occurred in patients with the larger 10-mm stent diameter (Fig. 2B). However, this consideration must be balanced with the dangers of a narrow construct that inadequately apposes the sinus wall, which can lead to turbulent flow in the stent, intimal hyperplasia, and in-stent thrombosis.[27,28] Finally, VSS can only serve to alleviate the exacerbation of IIH due to the pressure gradient from TS stenosis; it fails to address the underlying cause of initial ICP elevation. However, this is not the case with intrinsic stenosis from an enlarged arachnoid granulation (Fig. 4A), which causes venous outflow obstruction, resulting in increased ICP. Although stenting of this lesion should obviate any further cause for ICP elevation, we encountered a case in which stenting of the initial lesion caused the outpouching of another arachnoid granulation distal to the stent (Fig. 4B). This finding indicates a possible intrinsic mechanism to SAS as well (Fig. 7C), which would also be mitigated by a longer stent construct.

Given our single-center experience, we recommend that patients with IIH-associated TS stenosis undergo the placement of a long stent construct extending from the sigmoid sinus to the torcula with appropriate sizing of the stent diameter to mitigate the risk of SAS.

Conclusions

Cerebral VSS is an established endovascular treatment in patients with IIH-associated TS stenosis that fails conventional medical therapy. However, the notable rates of symptom recurrence and SAS remain concerning. The etiology of SAS is multifactorial, and the preemptive use of full-length constructs terminating at the torcula is crucial for its prevention. However, further investigation is necessary to determine the specific mechanisms underlying SAS and describe their correlation with patient symptoms.

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OpenAI ChatGPT was used to offer grammatical and syntax-related suggestions as well as for brevity.

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Disclosures

A. M. Malek is a cofounder and shareholder of and consultant for CereVasc Inc. outside the submitted work.

Author Contributions

Conception and design: Malek. Acquisition of data: Malek, Vajipayajula, Schreiner, Yu, Burke. Analysis and interpretation of data: Malek, Vajipayajula, Schreiner, Snyder. Drafting the article: Vajipayajula, Schreiner, Snyder. Critically revising the article: Malek, Vajipayajula, Snyder, Burke. Reviewed submitted version of manuscript: Vajipayajula, Snyder, Burke. Approved the final version of the manuscript on behalf of all authors: Malek. Statistical analysis: Malek, Vajipayajula. Administrative/technical/material support: Burke. Patient care: Ferraro.

Supplemental Information

Previous Presentations

Posters with preliminary data and portions of the study were presented at the Society of NeuroInterventional Surgery Annual Meeting held in San Diego, CA, on July 31–August 4, 2023; the AANS Annual Scientific Meeting held in Chicago, IL, on May 3–6, 2024; and the Society of Vascular and Interventional Neurology Annual Meeting held in Orlando, FL, on November 19–22, 2025.

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