SVN Stroke & Vascular Neurology

Dural sinus septum: an underlying cause of cerebral venous sinus stenting failure and complications

Yangang Zhao,[1,2,3] Xiaolan Zhang,[3] Bin Lv,[3] Jun Wang,[3] Xinfeng Liu,[3] Zhihua Du,[3] Fang Cui,[2] Baoming Li,[2] Xing Chen,[1] Xiangyu Cao [3]

To cite: Zhao Y, Zhang X, Lv B, *et al*. Dural sinus septum: an underlying cause of cerebral venous sinus stenting failure and complications. *Stroke & Vascular Neurology* 2024;**9**: e002407. doi:10.1136/svn-2023-002407

YZ and XZ contributed equally.

Received 21 February 2023
Accepted 27 June 2023
Published Online First 11 July 2023

[1]Beijing Advanced Innovation Center for Biomedical Engineering, School of Engineering Medicine, Beihang University, Beijing, China
[2]Department of Neurology, Hainan Hospital of Chinese PLA General Hospital, Sanya, Hainan, China
[3]Department of Neurology, Chinese PLA General Hospital, Beijing, China

Correspondence to
Dr. Xiangyu Cao;
caoxiangyu301@126.com

Pro. Xing Chen;
xingc@buaa.edu.cn

ABSTRACT

Objectives The presence of dural sinus septum has long been identified anatomically but is often neglected for its clinical significance. Our findings revealed the association of dural sinus septum with venous sinus stenting failure and complications supported by clinical evidence.

Methods This retrospective study included 185 consecutive patients treated with cerebral venous sinus stenting from January 2009 to May 2022. We identified the dural sinus septa using digital subtraction angiography (DSA) and classified them into three types based on their location. The septa at the transverse sinus were defined as type I, those at the junction between the transverse sinus and sigmoid sinus were defined as type II and those at the sigmoid sinus were defined as type III. Based on the anatomic features and neuroimaging clues, we investigated the correlation of dural sinus septa with stenting failure and complications.

Results 32 (17.1%) out of 185 patients (121 with idiopathic intracranial hypertension and 64 with venous pulsatile tinnitus) were identified with dural sinus septa by DSA. More than half of the septa were type I (18/32, 56.2%), followed by type II (11/32, 34.4%) and type III (3/32, 9.4%). The dural sinus septa caused three stenting failures and complications, including one case of venous sinus injury with subdural haemorrhage and two cases of incomplete stent expansion. Statistical analysis revealed that the presence of dural sinus septum ($p<0.01$) was associated with complications of cerebral venous sinus stenting.

Discussion The dural sinus septum is a common structure in the cerebral venous sinus. We found that the presence of dural sinus septa introduces uncertainties to cerebral venous sinus stenting and suggested precautions and ingenious skills in imaging and treatment.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ The septum is a unique yet common structure in the cerebral venous sinus, but clinicians almost neglect its existence.

WHAT THIS STUDY ADDS

⇒ This study discloses that the septum is an underlying cause of complications of cerebral venous sinus stenting.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ This study introduces original imaging and treatment techniques to reduce septum-related complications.

INTRODUCTION

Cerebral venous sinus stenting has been increasingly used to treat pulsatile tinnitus or intracranial hypertension caused by venous sinus stenosis.[1–3] However, its efficacy is undermined by a complication rate from 1.9% to 12%, mainly involving subdural haemorrhage, sinus or stent thrombosis, headache and retroperitoneal haemorrhage.[4–6] The latter two complications are associated with mechanical stimulation of the venous sinus wall or puncture site haemorrhage,[7] whereas the causes of the former two are still unclear. Haemorrhage typically occurs when the vasculature is damaged during stenting. Unlike the anatomic structure of arteries, we recognised that the venous sinus is complexed with the fibrous septum, a special structure in the form of bridges, tunnels or pockets.[8] As early as 1975, a septal structure in the straight sinus was first discovered and reported.[9] Since then, few studies have followed this finding until researchers identified that 29.4% of cadavers presented with septa in 2010.[10] In a recent anatomic study, 44.4% of dissected sinuses had septal structures.[8] The presence of dural sinus septa was considered an ignored aetiology of sinus stenosis and the resultant idiopathic intracranial hypertension. More importantly, they speculated that angioplasty of the septal lumen could tear the sinus wall and ultimately cause devastating epidural or subdural haemorrhage. Despite the sound reasoning given the special structure and high incidence of dural sinus septa, no clinical case was used to support their hypothesis.

This study aimed to provide direct clinical evidence that the presence of dural sinus septa can cause stenting failure and complications and to introduce measures that identify the septa and avoid stent placement into the septal lumen for reducing complications.

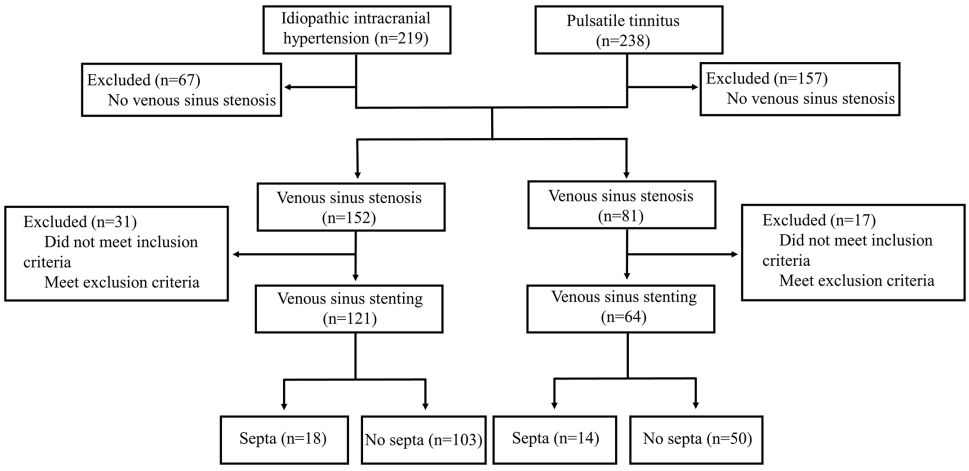

Figure 1 Flow diagram of the study design.

METHODS

Patients

We conducted a retrospective collection of patients who were admitted to our department between January 2009 and May 2022, presenting with intracranial hypertension and pulsatile tinnitus. Out of the total of 457 patients, 233 were diagnosed with venous sinus stenosis. Patients with idiopathic intracranial hypertension were considered to receive venous sinus stenting if medical treatment was ineffective, and venous sinus stenosis with a transstenosis pressure gradient was ≥10 mm Hg.[11] For venous sinus stenotic patients with pulsatile tinnitus, they were considered to receive venous sinus stenting if they experienced persistently intolerable pulsatile tinnitus accompanied by psychological disorders.[12] Patients were excluded from the study if they met any of the following criteria: (1) <18-year old; (2) intolerance to antiplatelet drugs, heparin, contrast agents or anaesthetic drugs; (3) pregnancy; (4) concomitant malignant tumours, severe coagulation dysfunction, severe liver or kidney dysfunction; (5) history of intracranial haemorrhage; (6) presence of intracranial vascular malformations or aneurysms; (7) refusal to receive digital subtraction angiography (DSA) examination and cerebral venous sinus stenting. The study design is illustrated in figure 1.

This retrospective study included a total of 185 consecutive patients with symptomatic sinus stenosis who underwent cerebral venous sinus stenting. Among them, 121 patients had idiopathic intracranial hypertension and 64 had venous pulsatile tinnitus. All the patients were routinely examined for visual acuity, fundus, visual field and lumbar puncture. The degree of venous sinus stenosis was determined through cerebral angiography, while the pressure gradient across the stenosis was measured using venous manometry with a microcatheter.

Perioperative management

Routine antiplatelet therapy, including a daily dose of clopidogrel (75 mg) and aspirin (100 mg), was prescribed at least 5 days before stenting. Thromboelastography was used for response testing of antiplatelet drugs. For all patients, the inhibition rate of arachidonic acid was >50%, and the inhibition rate of ADP was >30%. After stenting, patients were prescribed dual-antiplatelet therapy (75 mg of clopidogrel and 100 mg of aspirin daily) for at least 6 months.[13]

Venous sinus imaging and stenting protocol

Angioplasty and stenting were performed under general anaesthesia by consultant neurointerventionalists. After arterial femoral access was obtained, preprocedural DSA was performed to exclude vascular malformations or fistulas and to identify the location and degree of venous sinus stenosis. Subsequently, an 8F guide catheter was inserted into the lateral sinus with stenosis through venous femoral access. This was followed by the use of a Renegade microcatheter (Boston Scientific, Miami, Florida) to cross the stenosis. The morphological features of the stenosis were further observed by diagnostic venography using a Renegade microcatheter. Meanwhile, the distal and proximal pressures of the stenosis were measured by connecting them to a monometer. A 300 cm Transend microwire (Boston Scientific, Miami, Florida) was first placed in the superior sagittal sinus through the Renegade microcatheter. A 6 mm/20 mm Aviator balloon (Cordis Neurovascular, Miami, Florida) was navigated to the stenosis and inflated slowly at 6–8 atmospheric pressure with a 50% mixture of iodinated contrast (visipaque 320 (GE Healthcare, Princeton, NJ, USA)) and saline. Following balloon dilatation, a Precise stent (Medtronic, Irvine, California) of appropriate size was deployed to open the stenosis. After stenting, both arteriography and venography were performed to evaluate residual stenosis and improve venous drainage at the stenotic sinus. The distal and proximal pressures were also recorded.

DSA features and classification of dural sinus septa

At present, no radiological features are available to define dural sinus septa. As the anatomical morphology of the dural sinus septum is similar to that of a dissection, we defined the double lumen sign on DSA as dural sinus septa. The angiography data were interpreted by two

Table 1 Baseline clinical characteristics

Clinical characteristics	Means±SD (min–max) or n%
Female, n (%)	30 (93.8)
Age (mean±SD), years	20–57 (37.3±9. 5)
Clinical duration (mean±SD), months	0.2–360 (30.0±66.2)
Preoperative symptoms	
Headache, n (%)	10 (31.2)
Dizziness, n (%)	4 (12.5)
Visual dysfunction, n (%)	14 (43.8)
Pulsatile tinnitus, n (%)	14 (43.8)
Temporary amaurosis, n (%)	3 (9.4)

neuroradiologists with a consensus. During endovascular therapy, the location of the dural sinus septum is related to the failure and complication rates. To clearly describe the features of the dural sinus septa and guide the endovascular treatment of venous sinus stenosis, we classified the septa into three types based on their locations. The septa at the transverse sinus were defined as type I, those at the junction between the transverse sinus and sigmoid sinus were defined as type II and those at the sigmoid sinus were defined as type III.

Statistical analysis

Frequencies and percentages were calculated for the categorical variables. Means, SD, medians and ranges were determined for continuous variables. χ^2 test or Fisher's exact test was used to analyse the correlation between categorical variables. Statistical analyses were performed using IBM SPSS Statistics V.22 software.

RESULTS

Patient baseline

In total, 32 (17.3%) of the collected 185 cases were found to have dural sinus septa by cerebral angiogram. As shown in table 1, the average age of the patients was 37.3 (20–57) years, and 30 (93.8%) were women. They presented with typical symptoms, including headache (31.2%), visual dysfunction (43.8%) and pulsatile tinnitus (43.8%).

Stenting procedure and complications

Of the 32 included patients, the majority of stenoses occurred at the transverse–sigmoid junction (96.87%), except for one (3.13%) in the transverse sinus. Among them, 25 stenoses were at the right transverse–sigmoid sinus junction, one at the right transverse sinus, five at the left transverse–sigmoid sinus junction and one patient had bilateral stenoses at the transverse–sigmoid junction. Stenting procedures were performed with a technical success rate of 100%. There were three complications, including one patient with venous sinus occlusion and subdural haemorrhage and two cases of incomplete stent expansion (one with no significant relief of symptoms after treatment). The stenting-related complication rate of the septa group (3/32) was higher than that of the non-septa group (0/153), and the difference was statistically significant (p<0.01). Among the 32 patients in the septa group, 7 (7/32) experienced postoperative headaches, which was not significantly different compared with the non-septa group (31/153). Postoperative headache symptoms were mild to moderate pain that could be tolerated, and the symptoms gradually resolved within a few days.

Follow-up outcomes

In total, 30 (93.75%) patients were followed up. Fourteen patients had improved pulsatile tinnitus (3/14) or had completely resolved (11/14) immediately after treatment. Eight of them experienced relief from their headache 1–4 weeks after stenting. Twelve of the 14 patients with visual dysfunction reported partial or full recovery.

Classification of venous sinus septa

Based on the septa classification defined by their location (figure 2A,B), more than half of the septa were classified as type I (56.2%), followed by type II (34.4%) and type III (9.4%). Three representative classification cases are summarised in figure 2C–E, as evidenced by the double lumen sign.

Representative cases and complications study

Case 1. An adult patient was admitted to our centre in 2009 during the early stages of this study. The patient presented with a 2-month history of progressive headaches, nausea and vomiting, followed by new-onset bilateral blurry vision. Funduscopy revealed bilateral cotton–wool spots and papilledema. The cerebrospinal fluid opening pressure was 400 mm H_2O. The cerebral angiogram in figure 3A shows severe stenosis at the right transverse–sigmoid sinus junction, and the pressure gradient was measured to be 32 mm Hg. After the patient underwent stent implantation, poststenting angiography revealed occlusion of the right transverse sinus (figure 3B), and a subdural haemorrhage was found on cranial CT (figure 3C). The treatment aggravated the patient's headaches, nausea and vomiting. This unexpected complication was experienced by us for the first time and was unexplainable against routine venous sinus stenting. Through in-depth analysis, we judged that the stent was misplaced into the septal lumen, occluded the sinus lumen and further tore up the sinus wall, as shown in figure 3G,H. Guided by our new cognition, we deliberately directed the guidewire through the implanted stent cell to the true lumen of the transverse sinus. After solid confirmation by angiography, we first applied balloon dilatation and then placed another balloon-expandable stent to compress the expanded septum (figure 3D). The treatment partially recanalised the occluded transverse sinus, as shown by DSA in figure 3E,F. The symptoms gradually resolved postoperatively. At the 10-year follow-up, the patient reported no symptoms.

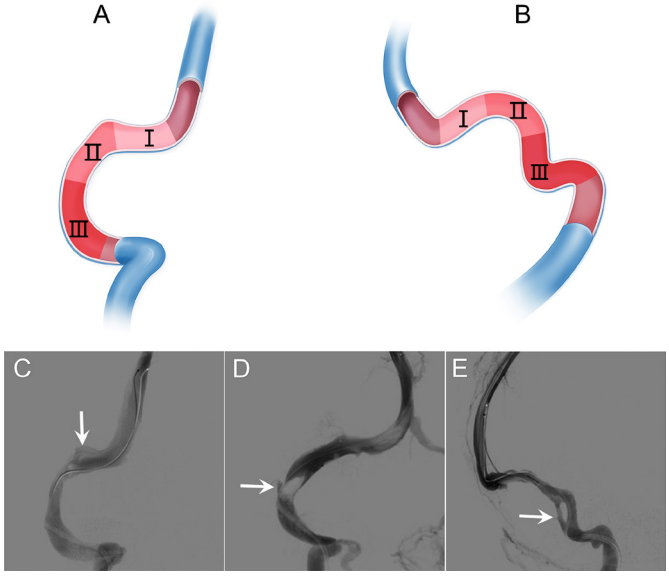

Figure 2 The classification and DSA imaging of venous sinus septa. (A) Frontal and (B) lateral schematics of type I: septa at transverse sinus, type II: septa at transverse–sigmoid sinus junction and type III: septa at sigmoid sinus. (C) Frontal angiogram of type I. (D) Frontalangiogram of type II. (E) Lateral angiogram of type III. DSA, digital subtraction angiography.

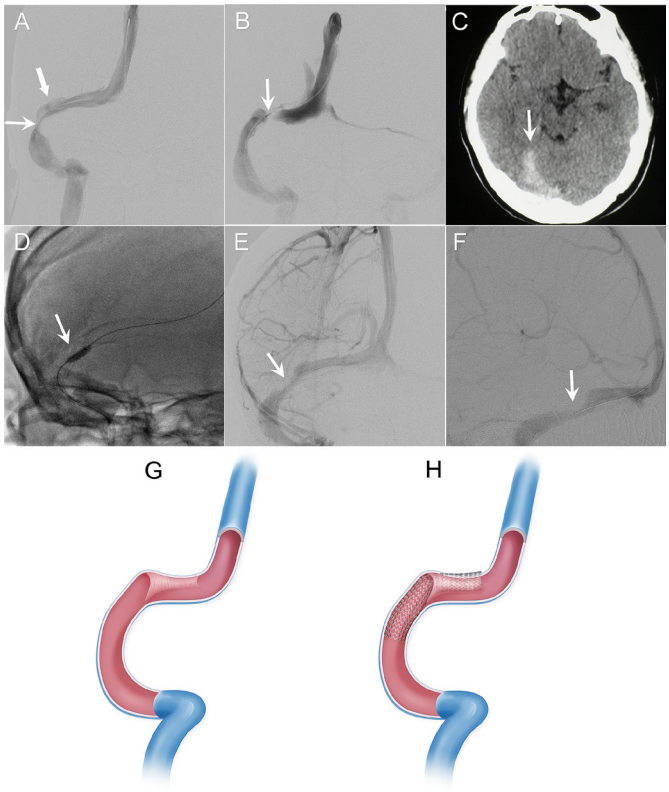

Figure 3 Images of Case 1 and animation of complication reasoning. (A) The sinus stenosis at the right transverse–sigmoid junction (arrow) and the venous sinus septal lumen (type I, thick arrow). (B) Occluded right transverse sinus (arrow). (C) Subdural haemorrhage (arrow). (D) Dilatated balloon (arrow) in the occluded sinus segment. (E, F) Frontal and lateral angiograms of the partially recanalised venous sinus (arrow). (G) The schematic of a septum at the right transverse sinus (type I). (H) The schematic of the occluded and torn transverse sinus after stenting into the septal lumen.

Case 2. An adult patient was admitted to our centre in 2017. This patient presented with 1-year temporary amaurosis and 1-month visual impairment of the left eye. Funduscopy revealed bilateral optic disc oedema. The cerebrospinal fluid opening pressure was 380 mm H_2O. The cerebral angiogram in figure 4A shows stenosis at the right transverse–sigmoid junction, and the pressure gradient was measured to be 15 mm Hg. We performed balloon dilatation to fully open the stenosis (figure 4B). Subsequently, a self-expanding stent was placed in the stenotic lumen. Poststenting fluoroscopy revealed that the central segment of the stent was unexpanded (figure 4C,D). Given the rare morphology of the placed stent, we judged that it was misplaced into the septal lumen, which in turn restricted the full expansion of the stent at that segment (figure 4E,F). The patient's venous return improved after stenting. We were concerned that repeated balloon dilatation could tear the sinus wall; therefore, we terminated the operation. At the 11-month follow-up, the symptoms of bilateral temporary amaurosis were relieved, but the vision of the left eye did not recover.

Case 3. An adult patient was admitted to our centre in 2016. This patient presented with a 2-month history of headaches and blurry vision. Funduscopy revealed bilateral optic disc oedema. The cerebrospinal fluid opening pressure was > 400 mm H_2O. The cerebral angiogram in figure 5A,B shows severe stenosis at the right transverse–sigmoid sinus junction and a septum at the sigmoid sinus. The pressure gradient was 40 mm Hg. Considering the clear presence of the septum, we deliberately directed the microcatheter and the following stent through the main lumen of the sigmoid sinus (figure 5C) to avoid entry into the septal lumen. It is noted that the proximal end of the stent was left at the main lumen of the septum to ensure an adequate radial force. After stenting, poststenting angiography revealed that the stenotic segment was opened, as shown in figure 5D,E. The fluoroscopic image in figure 5F shows that the proximal end of the stent was less fully expanded than the rest of the stent segment.

DISCUSSION

Cerebral venous sinus stenting is a common treatment for patients with symptomatic stenosis, but its higher complication rate than that of cerebral arterial stenting is unexplainable.[4 14] This has motivated our interest to thoroughly investigate the underlying cause of complications through anatomic analysis and clinical studies, with a focus on the venous sinus septum.

The venous sinus is formed by dural folds, and it lacks venous valves and typical vascular wall structures (intima, media and adventitia).[8] This kind of anatomical structure

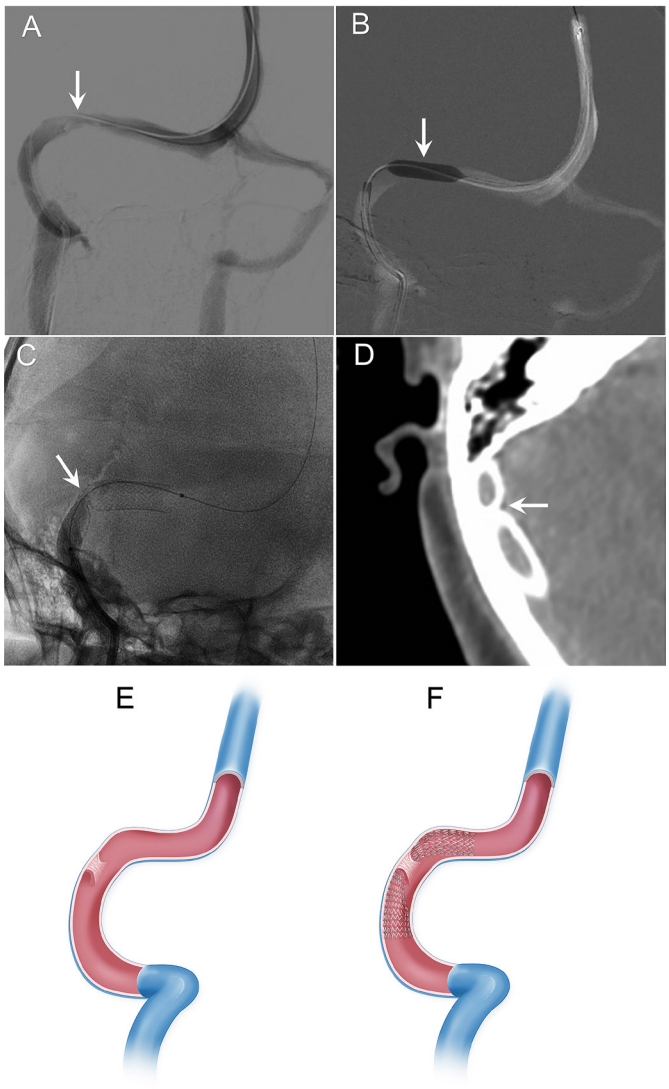

Figure 4 Images of Case 2 and animation of complication reasoning. (A) The sinus stenosis at the right transverse–sigmoid junction (arrow). (B) Fully dilated balloon at the stenotic segment (arrow). (C) Fluoroscopy image of an incompletely expanded stent (arrow). (D) VasoCT scan image of the stent cramped at the central segment (arrow). (E) The schematic of a septum at the transverse–sigmoid sinus junction (type II). (F) The schematic of the incomplete stent expansion at the septal lumen.

is accompanied by a unique structure or anatomical variation, the dural sinus septum. It is assumed that the dural sinus septum is the merger and formation of the embryonic venous plexus in venous sinuses during embryonic development.[15] It was first confirmed through an autopsy study in 1975, in which the incidence of straight sinus septa was reported to be approximately 20%. In addition, the dural sinus septum was found to present with a filling defect or double-lumen sign on postmortem radiography, which should be differentiated from venous sinus thrombosis.[9] In another more recent autopsy study, an incidence rate of up to 44% has disclosed the high proportion of dural sinus septa.[8] Besides anatomical method, high-resolution MRI, three-dimensional CT venography and

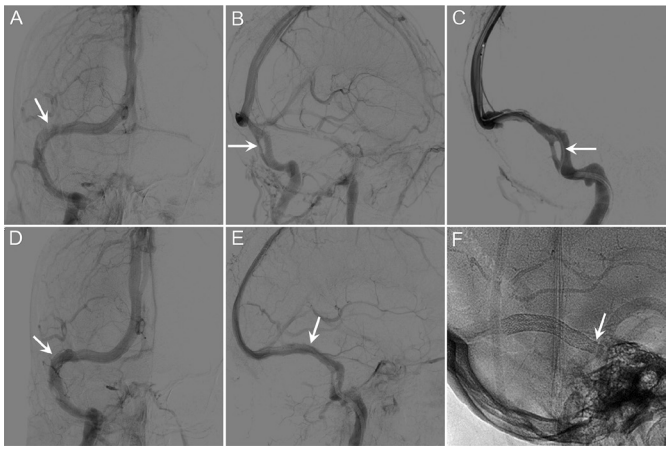

Figure 5 Images of Case 3. The sinus stenosis at the right transverse–sigmoid junction (arrow). (B) The septum at the sigmoid sinus (arrow). (C) The microcatheter through the main lumen of the sigmoid sinus (arrow). (D, E) Frontal and lateral angiograms of the expanded stent in the venous sinus (arrow). (F) Fluoroscopic image of the incompletely expanded proximal end of the stent (arrow).

intravascular ultrasound have also confirmed the presence of dural sinus septa.[16–18] In our study, DSA was used to identify dural sinus septum and defined double-lumen sign as its DSA feature. We reported an occurrence rate of 17.3%, which is lower than that from anatomical research because of the unrecognised small dural sinus septa by DSA, while DSA is advantageous for its easy access to stenting treatment.

Although the existence of dural sinus septa has been confirmed, its pathophysiological effects remain debatable. Some studies have regarded dural sinus septa as a physiological structure. Strydom *et al* reported that dural sinus septa could stabilise and maintain the three-dimensional structure of the venous sinus and prevent dilation or collapse of the venous sinus. Torn septa may form a valvular structure, obstruct normal cerebral venous drainage and eventually raise sinus venous pressure.[10] While the others considered dural sinus septa as a pathological structure. Subramaniam *et al* found that dural sinus septa could change haemodynamics and cause venous sinus stenosis or thrombosis.[15] In another study, 23.1% of patients with idiopathic intracranial hypertension had septa in the transverse sinus,[19] so dural sinus septa was thought to involve in its pathological process. This has raised concerns regarding its clinical significance in cerebral venous sinus stenting.

Through an in-depth analysis of the anatomical structure and DSA features of the septum combined with clinical cases, we found that the dural sinus septum introduces uncertainties to the treatment and could cause various venous sinus stenting failures and complications. Our study also revealed that stent implantation at the lateral sinus is sensitive to septum location; therefore, we divided the dural sinus septa into three types based on location. Type I has the highest proportion and largest footprint and features a strong double-lumen sign on

DSA. In case 1, the stent was misled in the septal lumen of type I because of our limited cognition back then. The head end of the stent injured the venous sinus and caused a subdural haemorrhage, and the inflated septum further occluded the venous sinus. Type II has the second highest proportion and can cause venous sinus stenosis. Type II usually features a small size and weak imaging signal, so the guidewire easily enters the septal lumen during stenting. Case 2 represents the second type of complication, in which the stent was incompletely expanded because it was misplaced into the false lumen of type II. An unexpanded stent increases the risk of stent thrombosis. Further attempts to inflate the unexpanded stent could lead to subdural haemorrhage. The incidence of type III is low; therefore, its anatomical characteristics are indistinct. Type III stents could cause incomplete expansion at the proximal end of the implanted stent. After recognising the presence of the septum, we took measures to avoid possible complications in case 3.

A previous retrospective study reported a case of poor stent apposition. The authors speculated that the stent was placed into the false lumen of septum and suggested recapturing the stent and avoiding stenting.[20] However, this is only a remedial approach, and this procedure will increase the risk of complications as the distal end of stent may damage the venous sinus during the recapturing process. Since we identified the septum as an underlying cause of stenting complications, we developed a special slide-test technique to guide the guidewire and catheter into the correct lumen of the venous sinus during stenting until 2018. The operating technique is shown in figure 6. Initially, we inflated the balloon at 1–2 atm to make it in a partial expansion state and slid the balloon along the guide wire. If the balloon can slide smoothly without jamming, it suggests that the balloon was in the venous sinus lumen, and subsequent stenting can continue (figure 6A). If the balloon was stuck, it was highly possible that it was placed in the septal lumen (figure 6B). If this occurred, the guidewire was withdrawn and tried again with the slide-test method until it was confirmed to enter the true lumen.

This is the first study to prove that the dural sinus septum is an underlying cause of cerebral venous sinus stenting complications, supported by strong clinical evidence. We found that stenting in the unidentified septal lumen posed a risk of subdural haemorrhage or stent thrombosis. The proposed imaging and treatment techniques are helpful for decreasing the complication rate of cerebral venous sinus stenting. This study has some limitations. DSA has a low resolution for identifying small-sized septa, and our developed slide-test technique strongly depends on the operating experience. We recommend employing multimodal imaging techniques, such as high-resolution MRI, intravascular ultrasound or optical coherence tomography, to identify septa and discern their anatomical features prior to cerebral venous sinus stenting in the future.

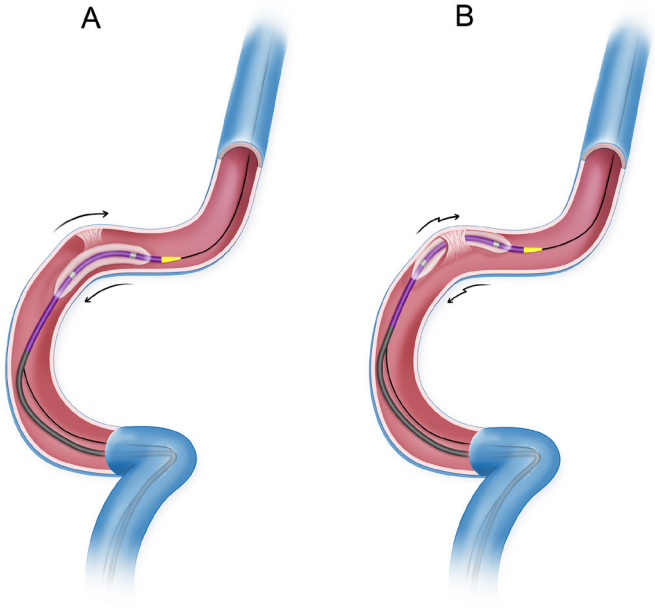

Figure 6 Animation of the slide-test technique. (A) When placed in the venous sinus lumen, the partially dilated balloon can freely slide back and forth. (B) When placed in the septal lumen, the partially dilated balloon gets stuck and should be withdrawn.

Acknowledgements We appreciate the consent of the patients/families and the support from all physicians and nurses involved in the care of this patient.

Contributors YZ designed and conceptualised the study and drafted the manuscript for intellectual content. XZ designed and conceptualised the study. BL, JW, XL and ZD had a major role in angiography and operation. FC and BL performed patient management and follow-up. XC and XC interpreted the data and revised the manuscript for intellectual content and is responsible for the overall content as the guarantors.

Funding This study was supported by the National Natural Science Foundation of China (Number 82101442 and Number 62074013), Fundamental Research Funds of the Central Universities, Beijing Municipal Natural Science Foundation (Number 7232152), the Capital Health Research and Development of Special (2022-2-5024), and Hainan Province Science Foundation for Youths (Number 821QN385).

Competing interests None declared.

Patient consent for publication Consent obtained directly from patient(s).

Ethics approval The study protocol was approved by the Ethics Committee of the First Medical Center of the Chinese PLA General Hospital (S2019-115-01). Participants gave informed consent to participate in the study before taking part.

Provenance and peer review Not commissioned; externally peer reviewed.

Data availability statement Data are available upon reasonable request.

ORCID iD

Xiangyu Cao http://orcid.org/0000-0002-4591-3220

REFERENCES

1 Verillaud B, Bedarida V, Labeyrie M-A. Association of spontaneous cerebrospinal fluid Rhinorrhea with transverse venous sinus stenosis: a retrospective matched case-control study. *Int Forum Allergy Rhinol* 2021;11:207.

2 Quintas-Neves M, Freitas E, Amorim JM, *et al*. Venous sinus stenosis causing isolated pulsatile tinnitus. *Can J Neurol Sci* 2019;46:591–2.
3 Liu X, Di H, Wang J, *et al*. Endovascular stenting for idiopathic intracranial hypertension with venous sinus stenosis. *Brain Behav* 2019;9:e01279.
4 Kalyvas AV, Hughes M, Koutsarnakis C, *et al*. Efficacy, complications and cost of surgical interventions for idiopathic intracranial hypertension: a systematic review of the literature. *Acta Neurochir (Wien)* 2017;159:33–49.
5 Narsinh KH, Hui F, Duvvuri M, *et al*. Management of vascular causes of pulsatile tinnitus. *J Neurointerv Surg* 2022;14:1151–7.
6 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;32:1408–14.
7 Mondie C, Maguire NJ, Rentea RM. *Retroperitoneal hematoma*. Treasure Island (FL): StatPearls, 2022.
8 McCormick MW, Bartels HG, Rodriguez A, *et al*. Anatomical variations of the transverse-sigmoid sinus junction: implications for endovascular treatment of idiopathic intracranial hypertension. *Anat Rec (Hoboken)* 2016;299:1037–42.
9 Bergquist E. Fibrous septa in the straight dural sinus. *Acta Radiol Diagn (Stockh)* 1975;16:331–6.
10 Strydom MA, Briers N, Bosman MC, *et al*. The anatomical basis of Venographic filling defects of the transverse sinus. *Clin Anat* 2010;23:153–9.
11 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;9:867–74.
12 Baomin L, Yongbing S, Xiangyu C. Angioplasty and Stenting for intractable pulsatile tinnitus caused by dural venous sinus stenosis: a case series report. *Otol Neurotol* 2014;35:366–70.
13 Cao X, Li B, Su H, *et al*. Pearls & Oy-Sters: angioplasty and stenting as new treatment method for cough headache with stenotic internal jugular vein: case report with 12-month follow-up. *Neurology* 2021;97:e964–7.
14 Alexander MJ, Zauner A, Chaloupka JC, *et al*. WEAVE trial: final results in 152 on-label patients. *Stroke* 2019;50:889–94.
15 Subramaniam RM, Tress BM, King JO, *et al*. Transverse sinus septum: a new aetiology of idiopathic intracranial hypertension *Australas Radiol* 2004;48:114–6.
16 Liang L, Korogi Y, Sugahara T, *et al*. Normal structures in the intracranial dural sinuses: delineation with 3d contrast-enhanced magnetization prepared rapid acquisition gradient-echo imaging sequence. *AJNR Am J Neuroradiol* 2002;23:1739–46.
17 Fukusumi A, Okudera T, Takahashi S, *et al*. Anatomical evaluation of the dural sinuses in the region of the torcular herophili using three dimensional CT Venography. *Acad Radiol* 2010;17:1103–11.
18 Yan F, Rajah G, Ding Y, *et al*. Safety and efficacy of intravascular ultrasound as an adjunct to Stenting for cerebral venous sinus stenosis-induced idiopathic intracranial hypertension: a pilot study. *J Neurosurg* 2019;132:749–54.
19 Tian Y, Zhang Z, Jing J, *et al*. Anatomic variation of the lateral sinus in patients with idiopathic intracranial hypertension: delineation with black-blood contrast-enhanced MRI. *Front Neurol* 2021;12:715857.
20 Labeyrie M-A, Fantoni M, Vever U, *et al*. Intracranial venous sinus Stenting for the treatment of lateral sinus stenoses: an analysis of 200 patients. *Diagn Interv Imaging* 2021;102:619–27.