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Physiological Relapse after Venous Sinus Stenting in Idiopathic Intracranial Hypertension and the Case for Pressure-Based Surveillance

Mohamed Hassanin, Ahmed Samy Sheta

Department of Neurosurgery, Saudi German Hospital, Alexandria, Egypt
Email: dr.m.hassanin@gmail.com

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Abstract

Background: Dural venous sinus stenting (VSS) is an accepted treatment for idiopathic intracranial hypertension (IIH) in patients with confirmed transverse sinus stenosis and a haemodynamically significant trans-stenotic pressure gradient on catheter manometry. The procedure interrupts a venous hypertension feedback loop and produces reliable short-term symptomatic benefit. Current post-stenting follow-up protocols focus on imaging surveillance for stent-adjacent stenosis (SAS) and clinical review. They do not routinely incorporate lumbar puncture (LP) manometry to verify that intracranial pressure (ICP) remains controlled after the procedure. The consequence is a structural inability to detect the specific failure mode we describe here: physiological relapse—sustained ICP re-elevation behind a radiographically patent stent, in the absence of structural stent failure. **Objective:** To present three patients with medically refractory IIH who underwent technically successful VSS with documented initial improvement and subsequently relapsed with confirmed stent patency and elevated LP opening pressures—and to argue that routine post-stenting LP manometry is necessary to detect this pattern. In all three cases, stenosis was confirmed on pre-procedural imaging and a trans-stenotic gradient meeting the published threshold for intervention was documented on catheter manometry. Specific gradient values were not available in the data set; this is acknowledged as a principal limitation. **Cases:** A 33-year-old woman with systemic lupus erythematosus (SLE) and acetazolamide intolerance (Case 1), a 27-year-old woman with three priors failed lumboperitoneal shunts and IIH without papilloedema (Case 2), and a 39-year-old man with morbid obesity (BMI 57.1 kg/m^2, pre-stenting LP opening pressure 480 mmH_2O) (Case 3). All three experienced documented initial improvement following VSS. All

three subsequently relapsed. Post-relapse imaging confirmed bilateral stent patency without thrombosis or SAS in every case. LP opening pressures at relapse, where measured, were above the diagnostic threshold despite patent venous outflow. **Conclusion:** These patients had the right diagnosis, the right imaging, the right gradients, and the right procedure. The stents are open. The intracranial pressure was not controlled. This is not a failure of patient selection or surgical technique—it is a failure of the post-stenting surveillance framework to ask the correct question. A patent stent does not guarantee controlled ICP. Serial LP opening pressure measurement at defined intervals after VSS is the minimum required to detect physiological relapse before it causes irreversible harm.

Keywords

Idiopathic Intracranial Hypertension, Venous Sinus Stenting, Intracranial Pressure, Physiological Relapse, Lumbar Puncture Opening Pressure, Post-Stenting Surveillance, CSF Dynamics, Trans-Stenotic Pressure Gradient, Stent Patency

1. Introduction

Idiopathic intracranial hypertension sits at the intersection of neurology and metabolic disease. Its strong epidemiological association with obesity in women of reproductive age, together with accumulating evidence for androgen excess, 11β-hydroxysteroid dehydrogenase type 1 (11β-HSD1) dysregulation, insulin resistance, and choroid plexus CSF hypersecretion as active pathogenic mechanisms [1] [2] has repositioned IIH from a diagnosis of exclusion to a metabolic disorder with neurological expression. This distinction matters clinically: it means that the biological processes generating raised ICP operate upstream of any mechanical intervention, and that addressing the mechanical consequence of that raised pressure—however successfully—does not extinguish the source.

Venous sinus stenosis, found in the majority of IIH patients on venography, is now understood to be predominantly extrinsic in origin—the compliant sinus wall compressed by surrounding elevated ICP rather than intrinsically narrowed by primary venous pathology [3] [4]. This distinction has direct implications for stenting. When focal stenosis generates a haemodynamically significant trans-stenotic pressure gradient, it establishes a self-reinforcing feedback loop: impaired venous outflow raises cerebral venous pressure, reduces the driving gradient for CSF reabsorption across the arachnoid granulations, further elevates ICP, and further collapses the sinus wall [3]. Venous sinus stenting interrupts this cycle by mechanically preventing sinus collapse, restoring outflow, and acutely lowering ICP. In patients where the feedback loop is the dominant mechanism, stenting can be durably effective. In patients where a strong upstream metabolic or inflammatory driver continues generating raised ICP independently of sinus haemody-

namics, the loop will reform behind the stent over time—even with the stent fully patent.

Patient selection for VSS is grounded in two requirements: confirmation of stenosis on cross-sectional venography, and documentation of a haemodynamically significant trans-stenotic pressure gradient on catheter manometry—typically defined as greater than 8 mmHg [5] [6]. These criteria establish that the stenosis is physiologically consequential and that stenting is mechanistically justified. What they do not predict is whether the upstream ICP-generating mechanism will remain adequately suppressed by venous decompression over the medium and long term.

Post-stenting follow-up protocols in the published literature are structured to detect the most recognized form of VSS failure: stent-adjacent stenosis (SAS), occurring in 10% - 18% of cases, in which compression develops at sinus segments immediately flanking the stented region [7] [8]. Published protocols include a repeat angiogram at three months, fundoscopy, and clinical review at intervals [9]. What none of these protocols include is a systematic measure of LP opening pressure after stenting. The implicit and untested assumption embedded in this framework is that a patent stent equates to controlled ICP. We present three cases that test that assumption and find it wanting.

Each patient had stenosis confirmed on pre-procedural CT venography and a trans-stenotic gradient meeting the threshold for intervention documented on catheter manometry. Each underwent technically successful stenting with documented initial improvement. Each subsequently relapsed. Post-relapse imaging confirmed patent stents in every case. In the patients where LP manometry was performed at relapse, ICP was elevated above the diagnostic threshold. Although the stents were working the pressure was not controlled.

2. Materials and Methods

2.1. Study Design and Ethics

Single-center retrospective case series from a dedicated IIH multidisciplinary service incorporating neurology, neuro-ophthalmology, and interventional neuroradiology. Conducted as a service evaluation; formal ethics committee approval was not required per institutional policy. Written informed consent for anonymized publication was obtained from all three patients.

2.2. Diagnostic Criteria

All diagnoses met the revised modified Dandy criteria [10]. Required elements: papilloedema on dilated fundoscopy; LP opening pressure ≥ 250 mmH_2O in the lateral decubitus position with normal CSF composition; and exclusion of structural or vascular causes on MRI with venography. Under the revised criteria, diagnosis without papilloedema is valid when LP OP is elevated alongside compatible neuroimaging and an appropriate clinical syndrome—the situation in Case 2,

whose IIH without papilloedema phenotype is a baseline characteristic, not a post-stenting finding.

2.3. Criteria for Stenting

All three patients were selected for VSS on the basis of: 1) transverse sinus stenosis confirmed on pre-procedural CT venography or MR venography; 2) a haemodynamically significant trans-stenotic pressure gradient documented on catheter venography with manometry, meeting the published threshold for intervention; and 3) failure of or contraindication to adequate medical management. These criteria were fulfilled in each case before any stent was deployed. Specific gradient values were not systematically recorded in the available data set; however, in all three cases the operating clinician documented a significant gradient as the procedural indication. The absence of precise gradient values is acknowledged as a principal limitation and is explicitly reported in both the Abstract and the Limitations section.

2.4. Stenting Procedure

All procedures were performed via femoral venous access under local anaesthesia and conscious sedation. Catheter cerebral venography with manometry was performed to confirm the stenosis and document the trans-stenotic pressure gradient immediately prior to stent deployment. Self-expanding nitinol stents were placed to span the stenotic segments under fluoroscopic guidance. Post-deployment venography confirmed sinus patency in all cases. All patients received dual antiplatelet therapy (aspirin and clopidogrel) according to institutional protocol, with loading doses administered 48 - 72 hours before the procedure.

2.5. Follow-up, LP Technique, and Relapse Definition

Patients were reviewed at 4 - 6 weeks post-procedure and at symptomatic recurrence, with fundoscopy and visual field assessment at each visit. Post-stenting imaging—MR venography (MRV) or CT venography (CTV)—was obtained to assess stent patency. Relapse was defined as recurrence of IIH-attributed symptoms following a documented period of improvement after VSS, confirmed by papilloedema on fundoscopy or LP opening pressure above 250 mmH_2O at the time of symptomatic return.

LP opening pressures were measured with patients in the lateral decubitus position with hips and head flexed, in accordance with standard practice. In Cases 1 and 2, all LPs were performed without pharmacological sedation. In Case 3, the LP performed at relapse was carried out under conscious sedation at the operating clinician's discretion, given patient factors including body habitus. Sedation may moderately reduce recorded opening pressures relative to the fully awake state; this methodological caveat is noted in the Case 3 narrative and in the Limitations section. LP technique was not standardized prospectively across the series, which is acknowledged as a limitation of the retrospective design.

3. Case Reports

The results of our cases involving initial clinical presentation, investigations, indication for stenting and relapse of symptoms are summarized in the table below (Table 1).

Table 1. Summary of patient characteristics, stenting indication, and relapse findings.

	Case 1—33F	Case 2—27F	Case 3—39M
Key comorbidities	SLE; renal calculi; acetazolamide intolerance	Three failed lumboperitoneal shunts; IIH without papilloedema (baseline phenotype)	Morbid obesity ~175 kg; BMI 57.1; male sex; obstructive sleep apnoea
Pre-stent LP OP (mmH_2O)	260	280	480
Pre-stent stenosis	Left transverse sinus stenosis confirmed on MRV	Bilateral TS stenosis with irregular lateral walls confirmed on MRV	Bilateral TS and sigmoid sinus stenosis confirmed on MRV
Trans-stenotic gradient	Significant gradient on catheter manometry; met intervention threshold	Significant bilateral gradients on catheter manometry; met intervention threshold	Significant bilateral gradients on catheter manometry; met intervention threshold
Stenting procedure	February 2025—unilateral left transverse sinus stenting	August 2025—bilateral transverse sinus stenting	January 2026—bilateral transverse and sigmoid sinus stenting
Clinical timeline	Stenting 27 Feb 2025; improvement confirmed 20 Apr 2025 (~7 weeks); relapse admission 24 Apr 2025 (~7.5 weeks); relapse LP 290	Initial headache and dizziness regression post-stenting; relapse with LP OP 370 mmH_2O	Pre-stenting assessment Jan 2026; LP under sedation 20 Jan 2026 (OP 480 mmH_2O documented 27 Jan 2026); bilateral stenting; relapse with LP OP 300 mmH_2O under sedation
Initial response	Headache and visual symptoms resolved; papilloedema cleared within weeks	Headache and dizziness regressed; meaningful subjective improvement	Headache and dizziness improved; papilloedema resolved on fundoscopy
Papilloedema at relapse	Present—recurrence of disc swelling	Not applicable—never had papilloedema (baseline IIH phenotype)	Present—papilloedema returned with symptomatic recurrence
Visual acuity	Comparative automated perimetry: visual field deterioration documented; formal acuity not recorded in available data	Not formally recorded in available data	UCVA 1.0/1.0 bilaterally at Jan 2026 ophthalmology review; bilateral papilloedema on fundoscopy
LP technique at relapse	Standard LP; lateral decubitus; no sedation	Standard LP; lateral decubitus; no sedation	Performed under conscious sedation
Post-stenting management	Acetazolamide not restarted (renal calculi); no pharmacological ICP suppression post-stenting	No pharmacological ICP suppression; patient declined thecoperitoneal shunt revision; expressed preference for VP shunt	Weight-reduction programme; dual antiplatelet therapy; no pharmacological ICP suppression
Alternative causes excluded	No SAS or thrombosis on MRV; no medication change; no SLE flare; no venous pathology outside stented segment	Bilateral stent patency on MRV/CTV; no active shunt in situ; no medication change	Bilateral stent patency on MRV; no SAS or re-stenosis; no medication change; persistent metabolic upstream driver (obesity, OSA)

Continued

LP OP at relapse (mmH_2O)	290 (above pre-stenting baseline 260)	370 (above pre-stenting baseline of 280)	300 (above diagnostic threshold; below pre-stenting value of 480)
Stent status at relapse	Patent on MRV; no thrombosis; no SAS; irregular posterior SSS walls (non-obstructive)	Patent on MRV and CTV; no thrombosis; no SAS	Patent bilaterally on MRV and MRI; no re-stenosis; no thrombosis

Abbreviations: LP OP, lumbar puncture opening pressure; MRV, MR venography; CTV, CT venography; SLE, systemic lupus erythematosus; IJV, internal jugular vein; SAS, stent-adjacent stenosis; SSS, superior sagittal sinus; TS, transverse sinus; UCVA, uncorrected visual acuity; OSA, obstructive sleep apnoea. Trans-stenotic gradient: specific values not recorded in data set; significant gradient meeting published intervention threshold was documented by the operating clinician in all three cases before stent deployment.

3.1. Case 1—33-Year-Old Woman with SLE and Acetazolamide Intolerance

A 33-year-old woman with established IIH and comorbid systemic lupus erythematosus (SLE) presented in early 2025 with progressive headache, deteriorating vision, and bilateral peripheral scotomas on automated perimetry. MRI brain demonstrated an empty sella and tortuous intraorbital optic nerves, both recognized markers of chronically elevated ICP. Fundoscopy confirmed bilateral papilloedema. LP opening pressure was 260 mmH_2O, measured in the lateral decubitus position without sedation.

Acetazolamide was commenced and then withdrawn on nephrological advice after the development of renal calculi, leaving no active pharmacological ICP suppression in the post-stenting period. Serial therapeutic LPs gave only transient relief. Serial automated perimetry documented progressive visual field deterioration beyond the pre-LP baseline. Pre-procedural CT venography and MRV confirmed stenosis of the left transverse sinus. Catheter venography with manometry documented a haemodynamically significant trans-stenotic pressure gradient meeting the threshold for intervention. Unilateral left transverse sinus stenting was performed on **27 February 2025**.

Post-procedurally, headache resolved and papilloedema cleared within weeks. At ophthalmological and neurological review on **20 April 2025**—approximately seven weeks following the procedure—fundoscopy confirmed clearance of papilloedema and the patient reported resolution of headache and visual blurring. Acetazolamide (cidamex) was not restarted given sustained clinical improvement and the ongoing history of renal calculi. On **24 April 2025**—approximately seven and a half weeks post-stenting—the patient was admitted with recurrent headache and visual symptoms identical in character to her pre-stenting complaints. Fundoscopy at this visit confirmed recurrent bilateral papilloedema. Post-relapse MRV confirmed stent patency with no in-stent thrombosis and no SAS; irregular walls and attenuated calibre of the posterior superior sagittal sinus were noted as a non-obstructive finding. A post-relapse LP opening pressure was 290 mmH_2O.

With regard to alternative explanations for recurrence: acetazolamide had been withdrawn before stenting and was not restarted, so no medication change oc-

curred in the peri-relapse period. SLE disease activity was not acutely altered and no change in immunosuppressive therapy had been made between the stenting date and relapse. Post-relapse MRV excluded structural stent failure, in-stent thrombosis, and stent-adjacent stenosis. No venous pathology outside the stented segment was identified. The most plausible mechanism of relapse is incomplete upstream ICP suppression in the complete absence of pharmacological adjunctive therapy, compounded by potential SLE-related neuroinflammatory disruption of CSF reabsorption pathways—a mechanism entirely independent of venous haemodynamics.

Post-stenting disease management for this patient did not include pharmacological ICP suppression: acetazolamide was contraindicated by her renal calculus history, and no alternative agent was commenced. No weight-loss intervention was documented in the post-stenting period. The selection criteria for this patient were met rigorously. The stent is open. The disease came back. The autoimmune dimension of SLE—with potential cytokine-mediated disruption of CSF reabsorption pathways—adds a further upstream mechanism entirely independent of venous haemodynamics. (See **Figure 1**)

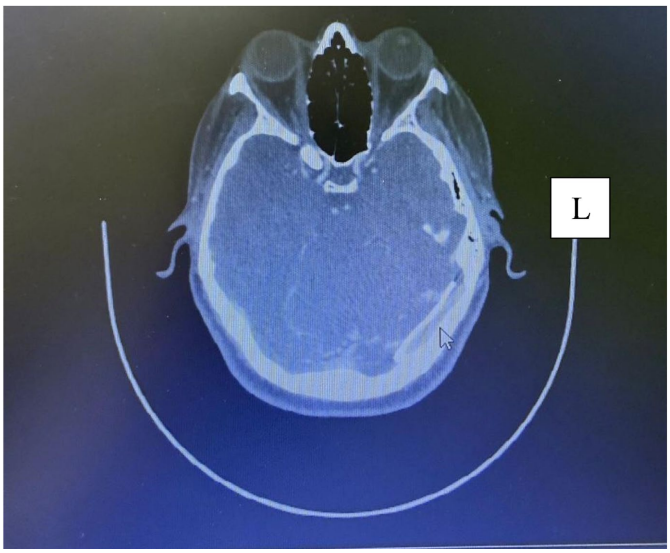

Figure 1. Axial CT obtained at the time of symptomatic relapse in case 1. Left stent hyperdensity within the transverse sinus confirm stent placement and patent luminal flow. No in-stent thrombosis or occlusion is identified.

3.2. Case 2—27-Year-Old Woman with Three Failed Lumboperitoneal Shunts

A 27-year-old woman presented with failed three lumboperitoneal shunts, the most recent extracted with marsupialization of omental and lumbar pseudocysts. Her baseline IIH phenotype was without papilloedema—she had never had disc swelling, and this is a diagnostically recognised presentation under the revised modified Dandy criteria [10], requiring elevated LP OP with compatible neuroimaging in the appropriate clinical context. LP opening pressure was 280 mmH_2O.

Pre-procedural MRV confirmed bilateral transverse sinus stenosis with irregular and stenotic lateral sinus walls. Catheter venography with manometry documented significant trans-stenotic pressure gradients bilaterally, meeting the threshold for intervention. Bilateral transverse and sigmoid sinus stenting was performed in August 2025. Post-operatively, headache and dizziness regressed and the patient reported meaningful improvement.

Post-stenting CT venography confirmed patency of all deployed stents (See Figure 2).

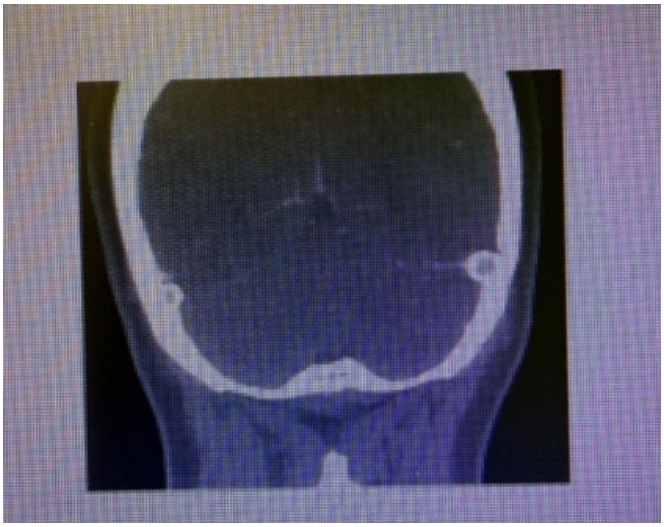

Figure 2. Coronal CT venography in Case 2, obtained at the time of symptomatic relapse. Bilateral stent hyperdensities within the transverse sinuses confirm stent placement and patent luminal flow. No in-stent thrombosis or occlusion is identified. This image demonstrates the central observation of this series: a structurally intact and patent stent in a symptomatic patient with confirmed ICP re-elevation on LP manometry.

The patient subsequently represented with headache, dizziness, and visual symptoms identical to her pre-stenting complaints. LP opening pressure at relapse was 370 mmH_2O—above both the diagnostic threshold and her pre-stenting baseline of 280 mmH_2O—despite bilateral stent patency. The relapse LP was performed in the lateral decubitus position without sedation. She was counselled regarding further management options and declined new implantation of a thecoperitoneal shunt, expressing a preference for a programmable ventriculoperitoneal shunt.

With regard to alternative causes of recurrence: structural stent failure was excluded by CTV and MRV confirming bilateral stent patency without thrombosis or SAS. The most recently placed lumboperitoneal shunt had been extracted prior to stenting; no active shunt was in situ at the time of relapse, and shunt-related factors can therefore be excluded. No new medications were commenced in the interval between stenting and relapse. No secondary cause of intracranial hypertension—including venous pathology outside the stented segment, comorbidity-related secondary ICH, or medication change—was identified.

Post-stenting disease management did not include pharmacological ICP suppression. The persistence of ICP elevation in this patient—having overcome three prior surgical drainage procedures before stenting—is consistent with a robust upstream CSF-secretory drive that venous decompression alone was insufficient to control. This case is instructive: three prior CSF diversion procedures had failed. Then bilateral stenting—with confirmed stenosis and confirmed gradients—also failed to maintain ICP control. The upstream ICP-secretory drive in this patient had already overcome three surgical drainage procedures before stenting was attempted. It would be mechanistically surprising if venous decompression, which does not address CSF secretion at the choroid plexus, produced durable control where three shunts had not.

3.3. Case 3—39-Year-Old Man with Morbid Obesity and Pre-Stenting LP OP of 480 mmH_2O

A 39-year-old man presented with persistent headache, dizziness, and episodic visual blurring in the setting of known benign intracranial hypertension. He weighed approximately 175 kg (BMI 57.1 kg/m^2) and was enrolled in a weight-reduction programme at the time of assessment. IIH in males represents approximately 10% - 13% of the IIH population and is associated with higher rates of severe visual loss and a generally more aggressive disease course [11]. At ophthalmological review on **15th of January 2026**, fundoscopy revealed bilateral disc changes consistent with papilloedema—slightly elevated discs with ill-defined nasal margins bilaterally and partial obliteration of the left optic cup; uncorrected visual acuity (UCVA) was 1.0/1.0 bilaterally. To confirm the intracranial pressure, a further LP was performed under conscious sedation during admission on **20 January 2026**, with an opening pressure of 480 mmH_2O documented on **27 January 2026**; headache improved after this LP. The LP under sedation was performed in the lateral decubitus position at the clinician's discretion given patient body habitus.

Pre-procedural MRV confirmed bilateral transverse and sigmoid sinus stenosis. Catheter venography with manometry documented haemodynamically significant trans-stenotic pressure gradients bilaterally, meeting the published threshold for intervention. Bilateral transverse and sigmoid sinus stenting were performed. Post-stenting MRV confirmed bilateral stent placement and patency (See **Figure 3**). Headache and dizziness improved and papilloedema resolved on follow-up fundoscopy. The patient was discharged on dual antiplatelet therapy and continued enrolment in the weight-reduction programme; no pharmacological ICP suppression was commenced.

He subsequently represented with recurrent dizziness and episodic visual blurring. Fundoscopy at this visit demonstrated recurrent bilateral papilloedema. Post-relapse MRI with MRV confirmed bilateral stent patency with no radiological features of re-stenosis. LP opening pressure at relapse was 300 mmH_2O, measured under conscious sedation—above the diagnostic threshold, confirming ICP re-elevation despite intact bilateral venous reconstruction, though substantially

lower than the pre-stenting value of 480 mmH_2O. The partial pressure reduction from 480 to 300 mmH_2O is evidence that bilateral stenting delivered real haemodynamic work. The persistence of ICP above threshold at relapse is evidence that the metabolic upstream driver was not extinguished by that work.

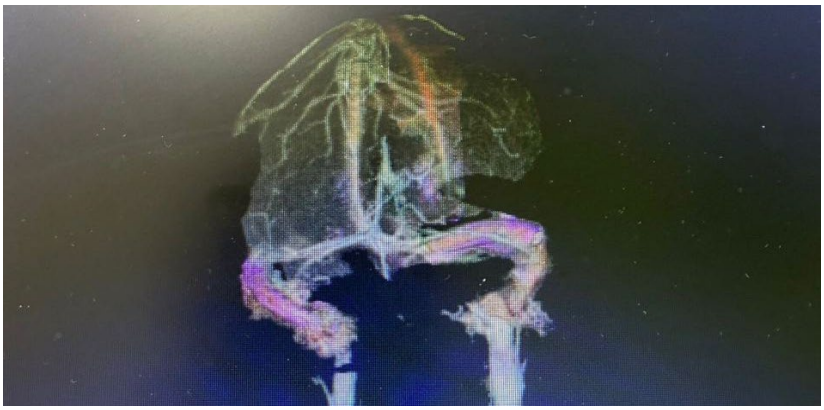

Figure 3. Three-dimensional volume-rendered CT venography, posterior view, in Case 3 obtained at the time of symptomatic relapse. Bilateral transverse and sigmoid sinus stents are patent with no evidence of thrombosis or haemodynamically significant stent-adjacent stenosis. This reconstruction confirms that venous outflow was anatomically restored and remained so at the time the patient was symptomatic with elevated LP opening pressure. No stent displacement or kinking is identified.

With regard to alternative causes of recurrence: bilateral stent patency was confirmed on post-relapse MRI with MRV, demonstrating no in-stent thrombosis, stent displacement, or stent-adjacent stenosis. No venous pathology outside the stented segments was identified. No new medications were commenced and no change in relevant comorbidity—including obstructive sleep apnoea management—occurred in the interval between stenting and relapse. Despite enrolment in a weight-reduction programme, no clinically significant weight reduction had been achieved at the time of relapse; the persistent metabolic upstream driver of extreme morbid obesity—driving choroid plexus hypersecretion through androgen and glucocorticoid dysregulation—was not meaningfully altered in the post-stenting period. The LP at which the relapse pressure of 300 mmH_2O was recorded was performed under conscious sedation; while sedation may moderately reduce the recorded opening pressure relative to the awake state, the value obtained substantially exceeded the diagnostic threshold of 250 mmH_2O and confirmed clinically significant ICP re-elevation. This sedation-related methodological caveat should be considered when interpreting comparability between baseline and relapse LP measurements in this patient.

4. Discussion

4.1. The Foundation: Confirmed Stenosis, Confirmed Gradients, Patent Stents

The argument in this paper rests on a foundation that must be stated clearly. These

were not patients stented on equivocal imaging or without haemodynamic justification. In every case, stenosis was confirmed on pre-procedural CT venography or MRV, and a trans-stenotic pressure gradient meeting the published threshold for intervention was documented on catheter manometry before any stent was deployed. Selection was rigorous. The procedures were technically successful. The stents remain open on post-relapse imaging.

This matters because it closes the most obvious alternative explanation for the relapse: that the patients were not appropriate candidates, or that the stenting did not achieve what it was intended to achieve anatomically. Neither is true. The venous outflow was obstructed, the obstruction was corrected, and the correction is durable. What is not controlled is the ICP. These two facts coexist—and that coexistence is the finding.

4.2. Why Stenting Helps and Why It May Not Be Enough

To understand what these cases show, it is worth being precise about the mechanism by which VSS produces its benefit. When a haemodynamically significant stenosis generates upstream venous hypertension, the pressure gradient driving CSF reabsorption across the arachnoid granulations is reduced, CSF accumulates, ICP rises, and the rising ICP further compresses the sinus—a positive feedback cycle [3] [4]. Stenting prevents sinus collapse, increases wall rigidity, restores outflow, and breaks the cycle. The result is a rapid and often substantial reduction in ICP—which is precisely what all three patients experienced after their procedures.

The critical question is what determines durability. If the feedback loop was the dominant mechanism sustaining raised ICP, then interrupting it may achieve lasting equilibrium. But if an independent upstream driver continues generating raised ICP at a rate that rebuilds the pressure gradient over time, the sinus will eventually be compressed again, the feedback loop will re-engage, and the patient will relapse—with the stent fully patent throughout.

Published data confirm that VSS produces favourable short-term outcomes: headache improvement in approximately 70% of patients, papilloedema resolution or improvement in over 84%, and stent patency maintained in the large majority at 18 months [7] [12]. The most recognized structural failure mode is SAS, occurring in 10% - 18% of cases [7] [8]. What the literature does not systematically document is the proportion of symptomatic recurrence attributable to physiological relapse behind a patent stent—because follow-up protocols are not designed to measure this.

4.3. The Upstream Drivers: What Stenting Cannot Address

IIH is increasingly understood as a metabolic disorder in which raised ICP is the neurological consequence of dysfunctional adipose biology. [1] [2] Androgen excess and 11β-HSD1 upregulation drive increased Na^+/K^+ ATPase and NKCC1 transporter activity at the choroid plexus, directly promoting CSF hypersecretion.[13] Impaired glymphatic CSF clearance, with structural changes at peri-

vascular astrocytic endfeet, [14] represents a further mechanism of CSF accumulation entirely independent of sinus haemodynamics. None of these processes are modified by venous decompression.

Each case in this series illustrates a specific persistent upstream driver. In Case 1, the withdrawal of acetazolamide left the post-stenting period entirely without pharmacological ICP suppression, and the autoimmune inflammatory mechanisms of SLE may independently disrupt CSF reabsorption pathways. In Case 2, a CSF-secretory drive sufficient to overcome three lumboperitoneal shunts was always unlikely to be durably controlled by venous decompression alone, however well executed. In Case 3, the metabolic substrate of extreme morbid obesity—driving choroid plexus hypersecretion through androgen and glucocorticoid dysregulation at maximal biological intensity—was not altered by bilateral stenting; the stent interrupted the venous feedback loop, but the upstream driver gradually rebuilt the pressure until the loop re-engaged.

4.4. The Surveillance Gap

The clinical problem these cases expose is not that stenting fails—it is that current follow-up cannot tell when it has. Published post-stenting protocols focus on structural surveillance: repeat angiography to detect SAS, fundoscopy, clinical symptom review [9]. Contrast-enhanced MRV has been reported with high sensitivity for post-stenting patency assessment [15]. But a patent stent and a controlled ICP are different biological states, and the current protocol measures one while assuming the other.

In a patient who returns after stenting with headache and a normal fundus, the current framework will show an open stent and conclude the stent is working. That conclusion is correct. What it does not address is whether the ICP is normal. Only LP manometry answers that question. In our cases the LP was the investigation that revealed the relapse for what it was—not a stent problem but a pressure problem—and directed management accordingly. Without it, patients would have been discharged with a normal imaging report and an unexplained clinical picture.

We propose that LP opening pressure measurement at defined post-stenting intervals—at minimum at 3 - 6 months and at any symptomatic recurrence—should be incorporated into standard follow-up for IIH patients after VSS. This is the same tool used to diagnose the disease. Its systematic absence from post-stenting surveillance constitutes a gap that this series makes concrete. The cases here establish proof-of-concept that physiological relapse with a patent stent is a real and clinically significant pattern; prospective studies with serial LP manometry at standardised intervals are now needed to quantify its frequency, predictors, and optimal management [16] [17].

4.5. What These Cases Do Not Argue

These cases do not argue against venous sinus stenting. All three patients benefited from their procedures. Case 3's ICP fell from 480 to 300 mmH_2O. In Cases 1

and 2, initial symptomatic relief was real and meaningful. The argument is not that stenting was the wrong decision; in each case, the indication was appropriate, the selection was rigorous, and the procedure was executed correctly.

The argument is that stenting should not be the last decision in the management pathway. It should be followed by a surveillance strategy capable of detecting physiological relapse, and by concurrent disease-modifying management that addresses the upstream metabolic drivers stenting cannot reach: weight reduction, optimization of comorbidities, pharmacological ICP suppression where tolerated. In patients with strong upstream drivers—severe obesity, active autoimmune disease, exhausted prior CSF diversion—the durability of venous decompression should be anticipated to be limited, and follow-up should be structured accordingly.

4.6. Limitations

This series is limited by its small size and retrospective design. Specific trans-stenotic gradient values from pre-procedural manometry were not available in the data set, which limits the precision of haemodynamic characterisation; this constitutes the principal limitation of the series and is reported explicitly in the Abstract, Methods, and here. Repeat catheter venography with manometry was not performed at relapse; we therefore cannot formally exclude a gradient in a region not captured by cross-sectional imaging. In Case 3, the relapse LP was performed under conscious sedation, which may moderately reduce the recorded opening pressure relative to the awake state; the true ICP at relapse may therefore have been somewhat higher than the recorded 300 mmH_2O. LP technique was not standardized prospectively across the series. Standardized visual acuity (Snellen or logMAR) and formal papilloedema grading (Frisén scale) were not consistently documented across all time points for all cases; ophthalmological outcome data are therefore incompletely reported in Cases 1 and 2. In Case 1, formal visual acuity was not recorded in the available clinical documentation, though comparative automated perimetry documented progressive visual field deterioration before stenting and improvement following therapeutic LP. Metabolic and endocrine biomarkers were not systematically measured. Prospective studies incorporating standardized pre- and post-stenting gradient documentation, serial LP manometry at defined intervals, formal ophthalmological outcome measurement (Frisén grade, visual acuity, visual fields), and repeat catheter manometry at symptomatic relapse are required to define the frequency, predictors, and natural history of physiological relapse after VSS in IIH.

5. Conclusions

Three patients with medically refractory IIH had transverse sinus stenosis confirmed on venography, trans-stenotic pressure gradients meeting the threshold for intervention documented on catheter manometry, technically successful venous sinus stenting, and documented initial improvement. All three subsequently

relapsed. Post-relapse imaging confirmed patent stents without structural failure in every case. Where LP manometry was performed at relapse, ICP was elevated above the diagnostic threshold.

The selection criteria were met. The procedures were successful. The stents are open. The pressure is not controlled. This pattern—anatomically intact venous reconstruction coexisting with physiological ICP re-elevation—is invisible to current post-stenting surveillance frameworks, which are built to detect structural stent failure rather than to measure ICP directly. Serial LP opening pressure measurement after VSS is the minimum surveillance tool capable of detecting this. We suggest it should be incorporated into standard post-stenting follow-up, with particular priority in patients whose upstream ICP drivers—metabolic load, inflammatory disease activity, or prior exhaustion of CSF diversion—are unlikely to be fully controlled by venous decompression alone.

Ethics and Consent

Conducted as a service evaluation; formal ethics approval not required per institutional policy. Informed written consent obtained from all patients for anonymized publication of clinical data and imaging.

Author Contributions

Mohamed Hassanin: Concept, clinical supervision, critical intellectual revision, and final approval. **Ahmed Samy Sheta:** Concept Study design, data collection, literature review, drafting, and final submission. Both authors approved the final manuscript.

Conflicts of Interest

The authors declare no competing interests.

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