Efficacy of endovascular stenting in dural venous sinus stenosis for the treatment of idiopathic intracranial hypertension

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Multiple pathophysiological mechanisms have been proposed for the increased intracranial pressure observed in idiopathic intracranial hypertension (IIH). The condition is well characterized, with intractable headaches, visual obscurations, and papilledema as dominant features, mainly affecting obese women. With the advent of MR venography and increased use of cerebral angiography, there has been recent emphasis on the significant number of patients with IIH found to have associated nonthrombotic dural venous sinus stenosis. This has led to a renewed interest in endovascular stenting as a treatment for IIH. However, the assumption that venous stenosis leads to a high pressure gradient that decreases CSF resorption through arachnoid villi requires further evidence. In this paper, the authors analyze the published results to date of dural venous sinus stenting in patients with IIH. They also present a case from their institution for illustration. The pathophysiological mechanism in IIH requires further elucidation, but venous sinus stenosis with subsequent intracranial hypertension appears to be an important mechanism in at least a subgroup of patients with IIH. Among these patients, 78% had complete relief or improvement of their main presenting symptoms after endovascular stenting. Resolution or improvement in papilledema was seen in 85.1% of patients. Endovascular stenting should be considered whenever venous sinus stenosis is diagnosed in patients with IIH. (DOI: 10.3171/2009.9.FOCUS09165)

Key Words • idiopathic intracranial hypertension • pseudotumor cerebri • venous sinus • stent • endovascular stent

Idiopathic intracranial hypertension has been variously known as pseudotumor cerebri or benign intracranial hypertension in the past.20,33 The word benign belies the seriousness of the visual field loss and significant risk of blindness that accompany this condition. The term idiopathic has gained favor and also emphasizes the most pertinent diagnostic criterion for this syndrome: increased ICP in the absence of any known causative factor. Dandy had first proposed a set of criteria to distinguish IIH from secondary intracranial hypertension.12 These criteria have undergone several modifications,4 but can be summarized as follows: 1) The patient must be awake and alert, with symptoms and signs that reflect only the generalized intracranial hypertension. Therefore, there should be no localizing signs, although false localizing signs, such as abducens nerve palsy, are permitted. 2) Raised ICP (> 25 cm H2O in the obese patient) should be confirmed during lumbar puncture in the lateral decubitus position, and the CSF composition should be normal. 3) Neuroimaging should reveal normal findings, without evidence of ventriculomegaly, intracranial lesions, or dural sinus thrombosis. 4) There is an absence of other causes of raised ICP.

Clinically, IIH presents most commonly with headaches. Transient visual disturbances and pulsatile tinnitus are also experienced in about 70% of patients.4,41 Papilledema due to raised ICP is almost ubiquitous and rarely unilateral. In its absence, a diagnosis other than IIH should be sought. Visual field loss is almost always present, although up to 25% of patients may not be aware of any visual loss.51 Left untreated, there is a high risk of continued deterioration and eventual blindness due to excessive papilledema.

Idiopathic intracranial hypertension is most common in overweight or obese women, with the female to male ratio ranging between 4:1 and 15:1.15,59 Between 71 and 94% of patients are clinically obese, as defined by a BMI > 30 kg/m2. In North America, there is an overall annual incidence of between 0.9 and 1.07 per 100,000.
However, the incidence is approximately 15 times higher in women between 20 and 44 years, who are more than 20% over their ideal weight. Children are rarely affected, although the occurrence in older teenagers is well documented. Although the exact pathophysiological mechanism is not known, various mechanisms have been considered, including increased CSF production, decreased CSF absorption, idiopathic brain swelling, and idiopathic intracranial venous hypertension. There are also associated conditions, although the cause-and-effect relationship is not very well known. These include vitamin A deficiency, several pharmacological agents (oral retinoids, tetracycline derivatives, indomethacin, rofecoxib, lithium, cimetidine and anabolic steroids, tamoxifen, dana-zol, and corticosteroid withdrawal), pregnancy, menstrual dysfunction, and several disease conditions (systemic arterial hypertension, diabetes mellitus, thyroid disease, hypoparathyroidism, iron deficiency anemia, ulcerative colitis, systemic lupus erythematosus, sickle cell disease, cystinosis, and renal transplantation).

The intracranial known causes of raised ICP should be excluded through radiological investigation. A CT scan will most often be the first test to evaluate patients with persistent headaches, although it is less sensitive than MR imaging. Brodsky and Vaphiades have reported a series of signs that may be predictive of intracranial hypertension including flattening of the posterior sclera (80%), a partially empty sella (70%), dilation (45%) or tortuosity (40%) of the optic nerve sheath, or Gd enhancement of the optic disc (50%). Since IIH is a clinical diagnosis supported by normal radiological findings, we suggest that the main benefit of MR imaging is to exclude other causes of raised ICP, but also to exclude venous sinus thrombosis, which could also cause raised ICP. The recommended imaging modality for evaluation of dural sinuses is MR imaging in addition to MR venography. However, MR imaging and MR venography may still be insufficient in detecting a transverse sinus outflow obstruction. Nevertheless, these tests should be carried out in any patient before the diagnosis of IIH can be given, and a positive result may alert the clinician to any venous sinus stenosis that may be amenable to stenting as a treatment for IIH.

The most commonly used medical treatment is acetazolamide, a carbonic anhydrase inhibitor that can reduce ICP and intraocular pressure by decreasing CSF and aqueous humor production. Surgery is reserved for patients in whom medical therapy has failed. This corresponds to approximately 20% of all patients with IIH. The CSF diversion procedures (placement of LP or sometimes VP shunts) can be done to decrease the ICP. Less commonly, optic nerve sheath fenestration can also be performed to improve visual function. Currently, there are no controlled, randomized trials comparing these surgical modalities to each other or to medical management. Failure is common, occurring in up to 30% of patients over variable time periods in both shunt and ONSF procedures. Subtemporal decompression as a surgical treatment for IIH is still controversial.

Recently, stenting of stenotic dural sinuses demonstrated in cases of IIH has gained popularity. It is unclear whether the increased ICP may be a cause or consequence of the dural venous stenosis. We analyze the relatively small number of cases published thus far to evaluate the efficacy of stenting as a treatment for IIH associated with dural sinus stenosis. We also present a case to illustrate this mode of treatment.

**Methods**

We performed a PubMed search for studies reported in the English and French languages after 1970 using the keywords “idiopathic intracranial hypertension,” “benign intracranial hypertension,” or “pseudotumor cerebri ” in several combinations with the key words “endovascular,” “stent,” and “venous sinus stenting.” We analyzed the data according to available information in the studies. GraphPad Instat (version 3.05, GraphPad Software, Inc.) was used for statistical analysis. Means were compared using the Student t-test, and p values < 0.05 were considered statistically significant.

**Results**

A total of 8 studies, representing 31 patients, were found that reported the use of stents for the treatment of IIH associated with dural sinus stenosis. The follow-up period ranged from 2 to 36 months with a median of 12 months. The demographic information, treatment, and outcome of each patient are summarized in Table 1. Patients ranged in age from 15 to 60 years with a mean of 35 years. All patients had headaches, and most had several visual symptoms (decreased visual acuity, diplopia, obscurations, constricted visual field); 84% of patients had documented papilledema. The duration of symptoms ranged from 3 weeks to 12 years. The BMI values ranged from 22 to 45 kg/m² with a median of 30 kg/m². Forty-five percent of the patients had BMI values of more than 30 kg/m².

The CSF pressures ranged from 25 to 59 cm H₂O (mean 36 cm H₂O). From the available data (see Table 1), 61% of the cases had bilateral stenosis (11 of 18). Of these 11 cases, unilateral stenting was done in 10 (7 on the right side and 3 on the left). In 1 case bilateral stenting was performed.

The venous pressures ranged from 15 to 58 mm Hg (mean 29.1 mm Hg) in the pre-stenotic part and from 2 to 18 mm Hg (mean 9.6 mm Hg) in the post-stenotic part. There was a pressure gradient ranging from 8 to 45 mm Hg (mean 19.5 mm Hg).

Stenting significantly improved both the prestenotic transverse sinus pressures and pressure gradients (this was from the available data in 13 and 14 patients, respectively, p < 0.0001). After stenting, 43.7% of the patients (14 patients) became asymptomatic, 34.3% (11 patients) improved, and 21.8% (7 patients) did not show any change in their symptoms. Stenting failed to maintain the patency of the sinus in only 1 patient 1 week after the operation, hence requiring further CSF diversion intervention. Papilledema resolved in 77.7% of patients and improved in 7.4%. Two patients had optic atrophy.
### TABLE 1: Information regarding patients reported on in the literature who underwent stenting for the treatment of IIH associated with dural sinus stenosis

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<th>Previ. Ops</th>
<th>Visual &amp; Other Sx (apart from headache)</th>
<th>Preop Papilledema</th>
<th>Last CSF Pressure (cm H₂O)</th>
<th>Pre-Stent Values (mm Hg)</th>
<th>Post-Stent Values (mm Hg)</th>
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Efficacy of endovascular stenting in dural venous sinus stenosis

Illustrative Case

History and Presentation. This 51-year-old woman presented with a 1.5-year history of headaches and right-sided pulsatile tinnitus. She also experienced transient visual obscuration and was diagnosed initially with migraine. Her BMI was 29 and she had mild papilledema bilaterally. Her lumbar puncture revealed an opening pressure of 31 cm H₂O. She received a course of acetazolamide, and later, topiramate, but derived no benefit clinically from either drug. The drug was terminated eventually due to intolerance of side effects. A subsequent cerebral angiogram revealed stenosis of the transverse sinus on both sides.

Operation. Cerebral angiography was performed after induction of general anesthesia, using a coaxial microcatheter supported by a guide catheter positioned in the internal jugular veins from a common femoral puncture. Pressure measurements were taken throughout the venous sinuses using a transducer referenced to zero at the level of the midaxillary line. The pressures measured during angiography were 27 and 25 mm Hg in the prestenotic segments of the left and right transverse sinuses, respectively, and 12 mm Hg in the post-stenotic segments bilaterally (Fig. 1). Thus, the right and left transverse sinuses demonstrated a pressure gradient of 12–15 mm Hg without any thrombosis. Under general anesthesia, a Luminex 10 × 40-mm stent was placed across the right transverse sinus, reducing the pressure gradient to 2 mm Hg bilaterally (Fig. 2).

Postoperative Course. Postoperatively, the patient continued on a regimen of clopidogrel 75 mg/day as well as aspirin. Her headache and tinnitus improved within several hours after stenting. At 2 months, the patient remained free of her presenting symptoms of headache and tinnitus. Computed tomography venography showed patent flow through the right transverse sinus stent.

Discussion

Idiopathic intracranial hypertension is a condition whose origin is obscure. One of the proposed underlying mechanisms is impaired CSF absorption. This may be due to increased pressure in the venous system.

Stenting for IIH was first performed by Higgins et al. Since then, there have been 31 more cases, including the case presented here. All cases involved stenting of the transverse sinus in the setting of idiopathic stenosis of the sinus. Overall, most (78%) of the patients showed improvement or complete resolution of the symptoms. Only one patient exhibited a failure to maintain the patency of the sinus and required subsequent insertion of a VP shunt. Based on this one case, the authors concluded that CSF stenting could not be recommended in cases of IIH with dural sinus stenosis. While we agree that further studies comparing CSF diversion procedures to stenting in cases of isolated dural sinus stenosis are needed, the experience recorded in this review does support the use of stenting as an effective treatment option in cases of IIH associated with nonthrombotic dural sinus stenosis.
The mechanism of how the stenosis occurs is unclear. King et al.\textsuperscript{28} proposed that increased ICP was the major reason causing dural sinus stenosis. They showed regression of the stenosis after serial lumbar punctures were performed to decrease the ICP. Additionally, De Simone et al.\textsuperscript{13} reported on a 28-year-old woman in whom IIH and associated papilledema with narrowing of the transverse sinuses were diagnosed. After removal of 20 ml of CSF, they reported a dimensional increase in the transverse sinuses with clinical remission after 2 months of follow-up.

In their study on the prevalence and morphology of sinovenous stenosis in IIH, Farb et al.\textsuperscript{19} showed that more than 90% of patients with IIH have transverse sinus stenotic appearance compared with healthy patients, and that there were 2 types of sinovenous narrowing. The first is a long, smooth narrowing due to external compression by brain parenchyma and the second is an acutely margined filling defect due to an enlarged arachnoid granulation, swollen by increased CSF pressure. These observations lend support to the view that the stenosis may be due to increased ICP.

Stenosis of the venous system can also be due to several other reasons, one of which might be the occurrence of chordae willisii. These fibers were first described by Willis\textsuperscript{49} as “many fibers, cords or strings such as we have observed to be variously stretched out in the ventricles of the heart.” Three basic forms of chordae willisii have been described:\textsuperscript{45} cordlike (trabecular), longitudinal (lamellar or laminar), and valvelike (mixed or valvular). In a postmortem study in which 25 cadaveric superior sagittal sinuses were studied, Sharifi et al.\textsuperscript{46} found valvelike chordae willisii as the most common form, covering 50% of the openings of the superior cerebral veins. The trabecular type was the next most frequent with single or complex networks. The least frequent type was the longitudinal type, which can divide the sinus into 2 or 3 separate channels of different diameters. Several other studies also identified these structures as causing filling defects\textsuperscript{38} or septations.\textsuperscript{38}

Bateman et al.\textsuperscript{7} recently proposed a mathematical model explaining the relationship between arterial inflow, CSF pressure, and venous outflow. They found that in patients with low-grade venous stenosis, a high arterial inflow was required for the patient to be symptomatic. Furthermore, their modeling enabled a very high prediction rate, which suggested that the reason for elevated pressures in IIH with stenosis could be due to a variable venous outflow collapse together with a variable arterial inflow. Their model also provided the basis for the use of lumbar puncture in patients with > 40% stenosis to obtain a remission, whereas such an approach would not work when the stenosis was < 40%. This may explain the variability of the results seen in the literature. The model also gave insight into the potential pathophysiological mechanisms of IIH showing that increased CSF production or decreased absorption of CSF were not required to account for the elevated CSF pressures. Overall, their study supported the use of venous sinus stenting in patients with sinus stenosis. However, the use of stents in patients with sinus stenosis remains controversial.\textsuperscript{6,42}

Endovascular stenting has its own risks. Complications include perforation of the vessels, stent migration,
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increased risk of thrombus formation, and restenosis. Because of this, anticoagulation therapy during the procedure and antiplatelet therapy after the procedure are necessary.

Conclusions

Notwithstanding the overall positive outcome of the 32 patients analyzed here, venous sinus stenting for the treatment of IIH remains controversial. However, in selected cases, this treatment method seems promising, with good outcomes reported from others and ourselves. There is no doubt that this treatment modality requires further evaluation. It is hoped that the results presented here encourage more thorough investigations in patients with IIH, and where appropriate, to the selection of endovascular treatment.

Disclosure

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References


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Fig. 2. Intraoperative venogram showing the stent (a). Postoperative left (b) and right (c) transverse sinus venograms showing the pressure values (in mm Hg).
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