Venous Sinus Stenting for Idiopathic Intracranial Hypertension: Where Are We Now?

Marc J. Dinkin, MD[a,b,c,*], Athos Patsalides, MD, MPH[d]

KEYWORDS

• Idiopathic intracranial hypertension • Venous sinus stenosis • Stenting
• Papilledema • Visual fields • Magnetic resonance venography • Angiography

KEY POINTS

- An increasing amount of evidence suggests that stenosis at the junction of the transverse and sigmoid sinuses contributes to increased intracranial pressure in idiopathic intracranial hypertension.
- Stenting of the stenosis in medically refractory, medically intolerant, or fulminant patients seems to result in improvement in symptoms, papilledema, and intracranial pressure in most patients assessed.
- Prospective, controlled trials comparing venous stenting with alternative surgical therapies or maximal medical therapy are needed to better assess the efficacy of venous sinus stenting.

Video content accompanies this article at http://www.neurologic.theclinics.com.

INTRODUCTION

Idiopathic intracranial hypertension (IIH) is a condition that causes increased intracranial hypertension, typically in obese women of childbearing age, in the absence of

Conflicts of Interest: No conflicting relationship exists for any author of this study.
Financial Support: None.
[a] Department of Ophthalmology, New York Presbyterian Hospital, Weill Cornell Medical College, New York, NY, USA; [b] Department of Neurology, New York Presbyterian Hospital, Weill Cornell Medical College, New York, NY, USA; [c] Department of Neurosurgery, New York Presbyterian Hospital, Weill Cornell Medical College, New York, NY, USA; [d] Division of Interventional Neuroradiology, Department of Neurological Surgery, New York Presbyterian Hospital, Weill Cornell Medical College, 525 E 68th Street, New York, NY 10065, USA
* Corresponding author. Department of Ophthalmology, New York Presbyterian Hospital, Weill Cornell Medical College, 1305 York Avenue, 11th Floor, New York, NY 10065.
E-mail address: mjd2004@med.cornell.edu

Neurol Clin 35 (2017) 59–81
http://dx.doi.org/10.1016/j.ncl.2016.08.006 **neurologic.theclinics.com**

mass lesion, venous thrombosis, or meningitic process. Although first described as a meningitis serosa by Quincke[1] in 1893, it was later called pseudotumor cerebri, then benign intracranial hypertension, and then, in recognition of its unclear cause, IIH. Symptoms typically include positional headache; horizontal diplopia from abducens palsies; pulse synchronous tinnitus (PST); and, most significantly, visual field loss and transient visual obscurations (TVOs) from papilledema. Although papilledema typically resolves with treatment of the disease, prolonged or severe disc edema may leave up to 25% of patients with visual field loss,[2] and, in more severe cases, even visual acuity may be permanently affected.

In Walter Dandy's[3] seminal article on the disorder, in which he laid out criteria, including increased intracranial pressure (ICP) without mass, and with normal contents on spinal fluid examination, he pondered the possible causes of increased ICP in these patients:

> *We may well be dealing with a condition that has more than one underlying anatomic or etiologic basis.*

Although the dynamics of cerebrospinal fluid (CSF) flow are likely to play a role in IIH, it is interesting that Dandy[3] entertained a vascular contribution as well:

> *The only other possible explanation of the increased intracranial pressure is by variance in the intracranial bed probably by vasomotor control.*

Dandy's[3] recognition of the importance of hemodynamics in ICP foretold an interest in venous hemodynamics and its role in IIH that has emerged over the last 2 decades.

Although some CSF drains through lymphatics and from spinal roots,[4] a primary means of CSF outflow is through the arachnoid granulations and into the cortical venous sinuses. Thus, venous sinus thrombosis or occlusion by an adjacent tumor may mimic IIH and cause significant vision loss from papilledema. Many practitioners therefore include magnetic resonance venography (MRV) in the work-up of IIH, although this practice remains controversial.[5] However, it has become clear over the last 2 decades that most patients with IIH harbor stenosis at the junction of the transverse sinus (TS) and sigmoid sinus (SS) on 1 or both sides,[6] leading to the hypothesis that this stenosis might be playing a role in the increase of ICP. In 1995, King and colleagues[7] showed an increase in venous sinus pressure in the superior sagittal sinus and proximal TS of 7 patients with IIH, with a mean pressure gradient along the TS of 13.3 mm Hg, (vs a mean of 1.4 mm Hg in controls), suggesting that stenoses could affect venous flow and therefore CSF dynamics. In 2 patients with minocycline-induced IIH, there was no observed gradient, suggesting that the gradient played a role in truly idiopathic IIH as opposed to secondary IIH induced by a medication.

Although IIH is typically treated with acetazolamide, which has been shown to be more effective than diet alone in mild IIH in the Idiopathic Intracranial Hypertension Treatment Trial (IIHTT),[8] the condition can be resistant to the medication in up to 10% of patients,[9] requiring a surgical therapy such as optic nerve sheath fenestration or ventriculoperitoneal shunt (VPS) or lumboperitoneal shunt (LPS). Up to 2.9% of patients present with a fulminant picture with central visual field and significant acuity loss[10] and cannot wait for acetazolamide to take effect, also requiring the quick fix of a surgical procedure. Although fenestration and shunts have a long record of efficacy in IIH, they both carry certain risks of complication, including diplopia and rare traumatic vision loss in fenestration[11] and overdrainage and infection with shunts.[12] It was therefore not long before groups began attempting to treat IIH with stenting of venous sinus stenosis (VSS) in the hope that this would reverse the gradient observed in King and colleagues'[7] study and therefore the increase in ICP.[13] The first