Endovascular Interventions for Idiopathic Intracranial Hypertension and Venous Tinnitus: New Horizons

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KEYWORDS

• Venous tinnitus • Intracranial hypertension • Endovascular intervention

KEY POINTS

- Pulsatile tinnitus from intracranial venous abnormalities is an uncommon and increasingly recognized cause of pulse synchronous tinnitus.
- Venous Stenoses associated with idiopathic intracranial hypertension can be treated with venous sinus stenting though randomized data is lacking.
- Venous abnormalities such as venous diverticulae or fenestrations may rarely cause venous tinnitus, and in select cases, may be successfully treated with venous embolization or stenting.

BACKGROUND

The term tinnitus describes a subjective ringing or buzzing in the ear, which may be continuous or pulsatile. Vascular causes of symptomatic, pulsatile tinnitus may include arterial variant anatomy (aberrant internal carotid artery, persistent stapedial artery, neurovascular loop compression syndromes); high-flow arterial diseases, including hypervascular tumors (glomus jugulare or tympanicum); arteriovenous shunt lesions (dural arteriovenous fistula or arteriovenous malformations of the head and neck); and arterial diseases of the head and neck associated with turbulence or flow acceleration (pseudoaneurysms and stenoses related to dissection, atherosclerotic vascular disease, or fibromuscular dysplasia). Venous causes of tinnitus are less well described and may result from congenital or acquired venous anomalies of the jugular bulb or sigmoid sinus (dehiscence, diverticula, aneurysms, fenestrations, webs) or stenoses of the dural venous sinuses (transverse and/or sigmoid sinus), including those associated with idiopathic intracranial hypertension (IIH).[1]

The management of tinnitus remains complex, and a recent review of available randomized data regarding medical management of nonpulsatile tinnitus suggest that data is insufficient for best practice guidelines.[2] An older review by Dobie and colleagues[3] is similar in that the primary focus was nonpulsatile tinnitus, and that venous tinnitus was not described in terms of treatment protocols. Venous causes of tinnitus may also be sufficiently

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rare such that large-scale, randomized data may be difficult to obtain. Other than venous disease in the setting of intracranial hypertension, review of the literature regarding venous tinnitus shows it is marked with multiple small series and case reports.

VENOUS TINNITUS

Venous tinnitus is typically characterized as pulse synchronous and represents a smaller subset of tinnitus in general.[1] Unlike arterial causes of pulse synchronous tinnitus, tinnitus that is caused by venous disease or anomalous venous anatomy can be reduced or extinguished by direct jugular venous compression or by turning the head to the side. Pulsatile tinnitus caused by venous disease is aggravated by straining, bending, or Valsalva maneuvers. In contrast to patients with arterial-type pulsatile tinnitus, patients with venous-type pulsatile tinnitus learn that they can reduce or eliminate their tinnitus by sleeping with the affected side in a dependent position. Dural arteriovenous fistulas, lesions typically located in the dural venous sinuses, are high-flow arteriovenous shunt lesions that may also result in pulsatile tinnitus[4] of the arterial type. For detailed discussion of these lesions, (See Miller TR, Serulle Y, Gandhi D: Arterial abnormalities leading to tinnitus, in this issue.)

IDIOPATHIC INTRACRANIAL HYPERTENSION

Benign or idiopathic, intracranial hypertension (also known as pseudotumor cerebri) may present with adjunctive symptoms of venous tinnitus. In 1998, Sismanis,[5] published a 15-year experience of pulsatile tinnitus and noted 56 out of 145 subjects with tinnitus had a diagnosis of IIH. Forty of these subjects were classified as having objective tinnitus versus 16 with subjective tinnitus. In this series, as in other contemporaneous series, no proposed mechanism for tinnitus was mentioned. However, direct pressure on the ipsilateral jugular vein typically results in cessation of the pulsatile tinnitus, suggesting that this is a venous flow–related phenomenon.

Mechanism of Venous Stenosis and Venous Hypertension

The exact pathophysiology for IIH is unknown. It may be related to reduced cerebrospinal fluid (CSF) absorption or overproduction of CSF. It is thought that decreased CSF absorption may occur in the setting of venous hypertension and, as such, venous hypertension has been put forth as the inciting cause for IIH, with early animal experiments outlining the relationship of increased intracranial pressure and venous pressures.[6] Later work by Karahalios and colleagues[7] proposed that venous hypertension is a universal mechanism for IIH. They found elevated venous pressures in all subjects clinically diagnosed with IIH in their series. Increased venous pressures, intracranially and systemically, may be secondary to focal stenosis or venous outflow obstructions, as well as systemic venous hypertension, often associated with morbid obesity.[8] This may, in part, explain the preponderance of IIH in obese young women.

The presence of venous stenosis in these patients has been gradually recognized, with early work estimating venous outflow obstruction in 19.7% of cases in a series of 188 subjects followed clinically by Johnston and colleagues[9] for 3 decades. This study was hampered by lack of high-resolution assessment of the cervical and intracranial draining veins; a point conceded by the investigators. In 2003, Farb and colleagues[10] prospectively evaluated 29 subjects with clinically diagnosed IIH compared with 50 normal controls and found that significant bilateral venous stenoses were found in 27 out of 29 (93%) of the IIH subjects compared with 4 out of 59 (6%) in controls. Similarly, Rohr and colleagues[11] reported the presence of venous stenosis in the setting of IIH in a smaller series. It is likely that IIH-related venous tinnitus results from turbulent blood flow and flow acceleration across the regions of venous stenosis. However, the cause of venous stenosis remains obscure, though both venous thrombosis (endoluminal obstruction) and extrinsic compression (compression from elevated CSF pressures) have both been postulated. Indeed, there are reports of other processes, such as dural sinus thrombosis or partial thrombosis, as well as dural arteriovenous fistulas, resulting in venous outflow obstruction that have been associated with increased intracranial pressures and attendant symptoms.[12,13]

Clinical Management

The primary goal in managing patients with IIH is to avoid irreversible vision loss and reduce the intensity of headaches. A complete neurologic history and physical examination should be performed including a neuro-ophthalmology evaluation for objective assessment of vision, papilledema, and elevated intraocular pressures. IIH-associated optic neuropathy is a result of prolonged increased CSF pressure within the optic nerve sheath. Given the prevalence of morbid obesity in these patients, weight loss is considered as a first-line management option. Lifestyle modifications with caloric