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Review Article

What Should Otolaryngologists Know About Dural Venous Sinus Stenting?

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Abstract

Dural venous sinus stenting is an emerging and exciting area in otolaryngology in collaboration with neurosurgeons and neuroradiologists. The first cases were reported 20 years ago. It is now considered part of the routine treatment of increased intracranial pressure due to transverse sinus stenosis. ENT doctors are the first to see these patients in their clinics, as sinus headaches, pulsating tinnitus, and dizziness are the most common symptoms. Previously, with limited success, high-dose diuretics and intracranial shunts had been the only options for treating these patients. Other methods, such as covering the sigmoid sinuses with graft material, appear to cause a sudden increase in intracranial pressure that can lead to blindness and even death. This overview summarizes the clinical and imaging characteristics of patients who will benefit from endovascular sinus stenting for elevated intracranial pressure.

Keywords: Air-bone gap; Anticoagulants in otolaryngology; Dural sinus stenting; Empty sella; Hearing loss; Increased intracranial pressure; Inner ear type conductive hearing loss; Pulsatile tinnitus; Sigmoid sinus dehiscence; Vestibular migraine

Introduction

When one of us (BM) first encountered a patient with transverse sinus stenosis in 2019, we did not know what to do or how to treat them. The patient came to us to treat her hearing loss and to stop the loud, whooshing sound from her right ear. She was in her 30s and looked healthy, overweight, but not obese. She used a long list of medications to control her daily headaches, which she said were not working. Her magnetic resonance imaging was reported as unremarkable, except for an empty sella. Her computed tomography angiography revealed normal intracranial arteries. Her audiology and otologic exams were unremarkable. A second neuroradiologist's interpretation of the imaging studies suggested a blockage of the right transverse sinus. This finding concerned me as I thought this must be due to a severe cerebrovascular event. She also had an extensive right sigmoid sinus wall dehiscence with three small diverticula. Given my uncertainty regarding this patient's management, I prescribed a high dose of acetazolamide and made a referral to the neurosurgery department for possible CSF diversion.

We needed to learn more about transverse sinus stenosis and how these patients may present to our clinics. I reviewed the last ten years of our recent otolaryngology publications and found little to no information regarding dural venous sinus stenting for pulsatile tinnitus. This review presents a brief overview of pulsatile tinnitus and idiopathic intracranial hypertension, oriented toward the otolaryngologist, with specific attention given to patient presentation and management with venous sinus stenting.

Presentation Patterns & Pathophysiology

CSF Circulation

Before delving into management, we should review cerebrospinal fluid circulatory dynamics and the pathophysiology of pulsatile tinnitus. Conventional theory suggests that CSF absorption through arachnoid granulations is due to a pressure differential [1]. Arachnoid granulations are pia mater's extension into dural venous sinuses distributed throughout the sinus and are most prominent in sagittal and transverse sinuses [2]. They appear similar to grapes growing into the vessel lumen. The pressure gradient between CSF and the dural sinus absorbs excess fluid into the venous system. This tenet is supported by research describing a lower number of arachnoid granulations in patients with idiopathic intracranial hypertension and shunt-dependent hydrocephalus following subarachnoid hemorrhage [3,4].

On the other hand, recent publications suggest a relationship between CSF resorption and the glymphatic system. CSF in the periarterial space is filtered into the interstitial space via aquaporin channels and driven toward the perivenous space surrounding veins. The CSF is then reabsorbed via dural lymphatics into the lymphatic circulation or via arachnoid granulations into the venous circulation. Reabsorption also occurs through the lymphatic sheaths of small vessels of the cranial pia mater as well as the perineural sheaths of the cranial and spinal nerves. It is hypothesized that impaired glymphatic drainage due to inflammation from obesity may result in congestion of the lymphatic pathway, increasing the CSF of the interstitial space and causing excess CSF to flow into the veins that travel out of the brain [2]. As the skull can only accommodate a fixed volume, the buildup

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of CSF results in elevated intracranial pressure. This principle may also explain the excess CSF surrounding cranial nerves and the empty sella sign seen on neuroimaging.

Symptomatology

Headaches, pulsating tinnitus, and dizziness are commonly associated with elevated intracranial pressure [5,6]. However, there is no universally pathognomonic representation. One may suffer more from headaches, while another may be debilitated by pulsatile tinnitus. Patients with pulsatile tinnitus murmurs describe hearing a deep, throbbing sound synchronized with a continuous pulse. It usually worsens when the patient lies down, exercises, or strains on the toilet. In diagnosing pulsatile tinnitus, otologic and audiometric assessment, including periauricular, orbital, and neck auscultation, additionally, it is essential to rule out middle ear and vascular pathologies such as an aberrant carotid artery or glomus tumor [7]. Pulsatile tinnitus can be divided into vascular and non-vascular types. Vascular tinnitus can be both of arterial or venous origin. Common cerebral vascular diseases such as atherosclerotic carotid stenosis and arteriovenous fistula are examples of arterial origin. Venous pulsatile tinnitus refers to those related to structural abnormalities associated with the sigmoid sinus wall, including both sigmoid sinus diverticula and sigmoid sinus dehiscence [8]. Because venous pressures and blood flow are lower than those in the arterial system, the pitch (frequency) of venous pulsatile tinnitus is noticeably lower than that of arterial pulsatile tinnitus, and it is harder to detect by auscultation. Furthermore, when attributable to venous sinus stenosis, the location of the lesion is often at the transverse-sigmoid sinus junction, making it difficult to appreciate even on computer tomography angiography or magnetic resonance angiography [7]. For this reason, cerebral angiography is often employed for further evaluation when other causes have been ruled out (Figure 1).

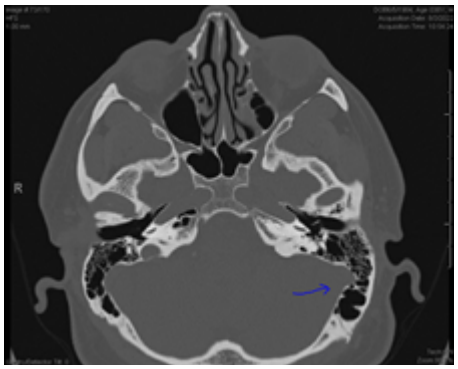

Figure 1: This patient has a BMI of 43. He is referred for his "sinus pressure" as a candidate for nasal surgery; his lumbar puncture opening pressure was 27cm H20. Acetazolamide resolved his "sinus pressure". The arrow shows the diverticula in the left sigmoid wall.

Pulsatile tinnitus is also often seen in the setting of idiopathic Intracranial Hypertension (IIH). IIH can be summarized in a pathological triad consisting of restriction of the venous CSF outflow pathway and congestion of the glymphatic system [2,9]. Dandy proposed the original diagnostic criteria for the diagnosis of IIH. However, these criteria have been modified with advances in neuroimaging and our understanding of the disease pathophysiology. Currently, establishing a diagnosis of IIH relies on signs and symptoms attributable to elevated intracranial pressure, including with or without papilledema, a CSF opening pressure of >25cm H_2O, normal CSF composition, and a lack of mass lesion or other structural intracranial pathology [10,11].

Anatomic Considerations

When there is an increase in venous return, our body initially tries to compensate by increasing the capacity of the vein, i.e., by increasing the size of the lumen. We develop venous collaterals if that does not work or the lumen is obstructed. In the case of intracranial hypertension secondary to an increase in the volume of CSF, the dural sinuses must accommodate and drain more fluid to prevent elevating intracranial pressure. Per the Monro-Kellie doctrine, the skull can only accommodate a certain volume of brain tissue, blood, and CSF. This is particularly true at the junction of transverse and sigmoid sinuses, with little room for expansion. However, as the dural sinus continues towards the temporal bone, a well-aerated mastoid composed of thin septa can absorb part of the venous sinus dilation.

The transverse sinus makes a turn at its distal end, where it transitions to the sigmoid sinus. This juncture, or "kink," is located at the posterolateral edge of the petrous part of the temporal bone. This spot may act as an a border between the middle and posterior cranial fossa. This juncture is the most frequent location of transverse sinus stenosis [12]. Imagine a flexible pipe, as in the picture (Figure 2). If the pipe is tightly attached to surrounding structures, as in the dural sinuses, the pipe will increase its diameter to allow more water to run through. As the pipe is tightly attached to the adjacent tissue and bone at the juncture and cannot change its diameter at this juncture, the juncture will turn into a kink.

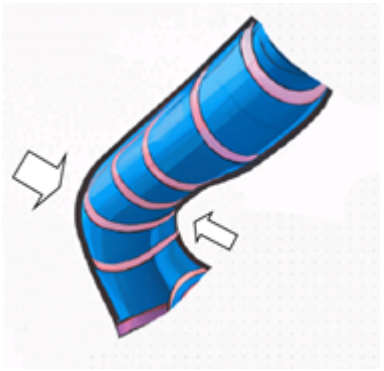

Figure 2: The juncture of transverse and the sigmoid sinus is usually where the stenosis develops (arrows).

Additionally, if we increase the amount of water per time, the increased force of water will try to straighten the pipe to align with the direction of the vector. In other words, the increased flow load in the transverse sinus will be transmitted to the sigmoid while forcing the sigmoid to be straightened. This is often noticed as an anteriorly situated sigmoid sinus during mastoidectomies. A similar mechanism could be applied to the development of a high jugular bulb.

More than 90% of patients with IIH have transverse sinus stenoses, which are usually located bilaterally at the junction with the vein of Labbe [13]. These stenoses result in increased cerebral venous pressure, leading to inefficiency of the venous CSF outflow pathway and resulting in the previously described congestion of the glymphatic system. Two types of venous sinus stenoses have been described in IIH: intrinsic and extrinsic. An intrinsic stenosis can be defined as the presence of a subarachnoid granulation inside the sinus. Extrinsic stenosis can be defined as a long-segment sinus stenosis without an endoluminal abnormality [2]. In a prospective trial of venous stenting

for idiopathic pulsatile tinnitus, Patsalides found that all patients with intrinsic stenosis had either a single or a cluster of enlarged arachnoid granulations located at the transverse–sigmoid sinus junction and all patients with extrinsic stenosis showed brain parenchyma compressing the venous sinus from the distal transverse to the transverse-sigmoid junction [14]. Though there is controversy over whether venous stenosis is a primary cause of IIH, studies revealing a transient improvement of trans-stenosis venous pressures with CSF diversion suggest it is at least a secondary manifestation of IIH [15].

The most referenced article about otological symptoms of intracranial hypertension in literature was written by Dr. Sismanis [16]. He reviewed the clinical presentation of idiopathic Intracranial Hypertension (IIH) for otolaryngologists. Although Sismanis believed that pulsatile tinnitus more likely originated from an arterial source, he recognized the importance of turbulence causing vibration of the dural sinus wall. The pulsatile tinnitus in IIH is usually described as a low-pitched unilateral whooshing sound and can be entirely silenced if the venous flow is brought to a complete stasis by pressure on the ipsilateral internal jugular.

To hear turbulence arising from a dural sinus, there must be a defect over the cortical bone covering the sigmoid sinus, allowing sound to leak out and reach the middle ear. As the blood flow velocity in veins is much slower than arterial blood flow, the vibrations impacting the wall of the dural sinus are much smaller than those arising in an artery. For example, a long-distance runner does not hear this turbulence even when intracranial pressure becomes very high at the peak of their run and turbulence develops in the dural sinus. As long as the cortical bone of the dural sinus remains intact, this structure ensures that this turbulent sound will not reach the cochlea. The primary mechanism of generating the pulsatile sound is the turbulent flow from the stenosis itself and the post-stenotic dilatation of the sigmoid sinus [14]. However, a dehiscent region over the sigmoid sinus wall or on the jugular bulb is needed for this low-pitched sound to be precepted by the subject. Additionally, transverse sinus stenosis causes flow anomalies that slowly erode the overlying bone, resulting in dehiscence, with subsequent weakening of the unprotected sinus resulting in the formation of a diverticula. The anterior bony wall facing the mastoid cavity and adjacent to the sigmoid sinus is relatively thin and weak, which explains why dehiscence occurs at the sigmoid sinus but not at the transverse sinus, which courses along the thicker occipital bone [9].

Sismanis further claimed that increased CSF pressure can be transmitted to the eighth cranial nerve, resulting in dizziness and vertigo. This is not true. An increased or decreased CSF pressure will increase or decrease the perilymphatic pressure, which will be reflected on the endolymph [17,18]. A sole increase in perilymphatic pressure will not cause episodic vertigo but may cause excess perilymph to leak out of the oval window. A long-lasting perilymphatic fistula may lead to endolymphatic hydrops and also explain some of the dizziness in IIH patients [9]. Figure 3 shows the "dramatic" improvement in hearing with pressure on the right jugular vein as it aborts the sound. There is still a tiny air-bone gap, likely caused by decreased compliance of the oval window secondary to elevated perilymphatic pressure [19].

An alternative mechanism to consider is the following: Bernoulli's hydrodynamic principles predict that CSF flow velocity decreases with increased pressure. Consequently, the decreased velocity of CSF flow further slows down the perilymphatic flow, normally at

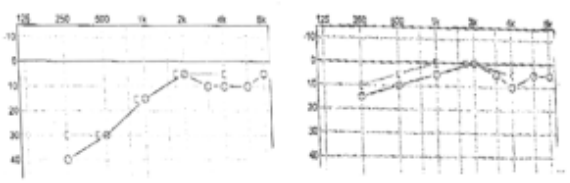

Figure 3: The hearing test of a patient with right transvers sinus stenosis before (left) and after pressure (right) on the jugular vein. The small bone gap is caused by decreased compliance of the oval window [19].

nanometer/second speed. As a consequence, free oxygen levels decrease when the flow slows down. As cochlear hair cells receive part of their oxygen from perilymph, this tampered perilymphatic flow may predispose the inner ear to the progression of hydrops.

Venous Sinus Stenting

Appropriate treatment of pulsatile tinnitus depends upon the nature of the underlying condition. Traditionally, techniques for the management of sinus-associated pulsatile tinnitus included coil embolization of the diverticula or surgical reconstruction of the sinus wall. Frequently, these techniques were fraught with complications, including hearing loss, blindness, and even death [20]. Since the first report of venous sinus stenting for pulsatile tinnitus in 1997, dural venous sinus stenting has been found to have increased utility and success in addressing the shortcomings of conventional medical or surgical techniques [21]. For IIH, the efficacy of stenting is predicated on the notion that venous outflow obstruction plays some part in the etiology of its signs and symptoms [10]. This was first explored by King et al. in 1995, who was the first to describe the contribution of venous stenosis to the pathophysiology of IIH through venous manometry, and then acted upon by Higgins et al. in 2002, who was the first to treat IIH with venous sinus stenting successfully [22,23]. Since then, the techniques and outcomes of venous sinus stenting for IIH (figure 4) and other etiologies of pulsatile tinnitus have been the subject of much attention and research. With the development of more refined techniques came an improvement in efficacy.

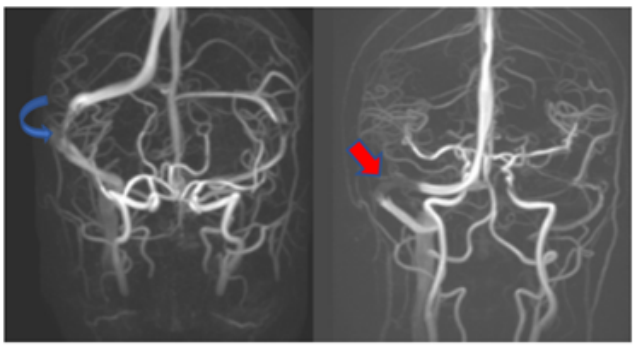

Figure 4: Magnetic Resonance Venography - MRV images of a patient successfully treated with dural venous sinus stenting. The left image reveals the stenosis at the lateral end of the transverse sinus, and the right image was taken one year. Curved arrow shows the stenotic segment and straight arrow points the stent.

Technique

Cerebral venography and manometry are done through femoral vein access, with a guide catheter placed at the internal jugular vein. By using a microcatheter, Superior Sagittal Sinus (SSS) access is gained, and an arterial pressure transducer is used to record pressures at multiple points along the venous system, including segments of the SSS, bilateral transverse sinuses, sigmoid sinus, jugular bulb, and cervical internal jugular vein. Patients with elevated venous pressures or significant trans-stenosis pressure gradient (>8mmHg) are considered for a venous stent [23,24]. Ultimately, in the case of IIH specifically, venous manometry is a decision-making factor for venous sinus stenting.

The goal of the venous stenting has been to normalize the trans-stenosis pressure gradient. The stenting operation is performed under general anesthesia. It is recommended that the pressure gradient between the segment before and after the stenotic segment should be at least 8mmHg [24]. However, practitioners may use clinical judgment to offer stenting in patients with a lesser gradient based on symptoms and degree of stenosis [25]. Prior to the procedure, patients are typically initiated on dual antiplatelet therapy. During the procedure, the patient is treated with heparin. Venous pressures and trans-stenosis pressure gradient are reconfirmed prior to placement of the venous stent. The choice of a stent may vary based on individual patient anatomy. For venous sinus stenting, the stents must be self-expanded with adequate radial force to overcome any external stenosis from elevated ICP and long constructs to ensure they extend >10mm pre- and post-stenosis. The stent is placed with complete coverage across the stenotic/high-pressure gradient segment. For high-grade stenosis, strategies such as the "Cobra" technique, in which a balloon is partially inflated to assist the delivery of an intermediate catheter, may be employed to safely bypass critical stenosis without too much forward catheter pressure [26]. During the postoperative period, patients are administered aspirin and a second anticoagulant. Long-term, only aspirin is needed. Annual follow-up with imaging is recommended.

Outcomes & Complications

Amongst the surgical options, including optic nerve sheath fenestration, CSF diversion, and bariatric surgery for obesity, the one-year outcomes of endovascular stenting in regards to visual function and headache demonstrated the most pronounced success, with improvement in visual acuity in 78% of cases and headache resolution in 77% of cases [10]. Higgins et al.'s first large cohort study (n=12) in 2003 showed a decrease in the mean pressure gradient from 18.9 to 11.3mmHg after venous sinus stenting [24,27]. A more extensive 185-patient review revealed a decreased pressure gradient from 20.1 to 4.4 mm Hg. Within the studies, many patients had significant symptomatic relief even with <8mmHg pre-stent gradient. In a meta-analysis by Nicholson et al., the most substantial symptomatic improvements after venous stenting were seen in papilledema, with 93.7% of patients experiencing an improvement or resolution in papilledema, followed by pulsatile tinnitus and headache, which demonstrated a 90.8% and 79.6% improvement, respectively [28]. Furthermore, stenotic gradient pressure before stenting was correlated with improvements in pulsatile tinnitus, but this did not reach statistical significance (P=0.169). In the most extensive retrospective cohort study available, endovascular stenting resulted in a 93% (40/43 patients) improvement in headaches and a 100% improvement in tinnitus [29].

The most feared complication of endovascular stenting is a thromboembolic event, which is rarely reported. Upon review of the meta-analysis, the overall rate of significant complications, such as intracranial hemorrhage, was 1.9% [28]. Another concerning complication of venous sinus stenting is acute in-stent or stent-adjacent stenosis. The incidence of such stenosis is roughly 18%, but it is symptomatic or requires retreatment in only 10% of cases [30]. A review by Starke et al. found a 3.5% incidence of in-stent stenosis, but only one required retreatment. Stent-adjacent stenosis is more frequently seen, with an incidence and retreatment rate of 11.4% and 6%, respectively [31]. Recurrence of symptoms seems to preferentially affect young, obese patients with extrinsic stenoses and high CSF opening pressures [28]. Interestingly, this group tends to develop stenosis at a secondary location apart from the segment treated by the index procedure. The recurrence rate of symptoms requiring a second invasive procedure is approximately 12% after venous sinus stenting (Nicholson meta-analysis), much lower than the 43% retreatment rate observed after conventional CSF diversion procedures [32]. These recurrences were treated with another stent in 72.8% of those patients, while a CSF diversion procedure was performed in 27.2% [28]. The rate of other adverse effects, including femoral pseudoaneurysm, transient hearing loss, and stent migration, is approximately 5.4% [27].

Closing Remarks

Otolaryngologists, when evaluating a pulsatile tinnitus patient, instead of focusing solely on the middle ear, arteries and cerebellopontine angle, must also pay attention to the dural venous sinuses, specifically transverse sinuses, pituitary fossa, optic nerves, and cerebellar ectopy. The best cost-effective imaging study to order for pulsatile tinnitus is CT head angiography with a request for documentation of post-contrast images of the dural venous sinus. In many patients, lumbar puncture of CSF opening pressure must also be measured.

While other therapeutic options exist for managing pulsatile tinnitus, recent literature provides mounting evidence that venous sinus stenting offers symptomatic relief and possible disease control. Additional follow-up and further studies are necessary to provide prospective and prolonged data to evaluate the durability of venous sinus stenting and long-term clinical outcomes.

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