Venous Abnormalities Leading to Tinnitus
Imaging Evaluation

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KEYWORDS

• Tinnitus • Idiopathic intracranial hypertension • Dehiscent sigmoid sinus
• Sigmoid sinus diverticulum • Dehiscent jugular bulb • Posterior fossa emissary veins

KEY POINTS

- Venous anomalies are the most commonly identified abnormalities in the work-up for pulse synchronous tinnitus.
- Subtle abnormalities such as focal sigmoid sinus wall dehiscence without diverticulum formation can often be overlooked if not incorporated into the standard search pattern for patients with tinnitus.
- Sigmoid sinus wall anomalies are an increasingly recognized cause for pulse synchronous tinnitus. Innovative treatment options including transmastoid sigmoid sinus wall reconstruction have a high rate of success with resolution of symptoms.

INTRODUCTION

Tinnitus can be categorized as subjective or objective and continuous or pulsatile. Pulse synchronous tinnitus (PST) is a repetitive somatosound commonly caused by vascular flow murmurs.[1,2] PST related to vascular flow murmurs can be the result of numerous arterial causes but only a limited set of venous causes. For detailed discussion on Arterial abnormalities leading to tinnitus, (See Miller TR, Serulle Y, Gandhi D: Arterial abnormalities leading to tinnitus, in this issue.) Even with the narrowed differential diagnosis for tinnitus caused by the venous system, these anomalies are the most commonly encountered cause in the work-up of PST.[2] Hence, it is important that radiologists familiarize themselves with the imaging features of these abnormalities. Potential venous causes of PST include idiopathic intracranial hypertension, sigmoid sinus wall anomalies, transverse and sigmoid sinus stenosis, jugular bulb anomalies, and prominent posterior fossa emissary veins.[2] Idiopathic intracranial hypertension (IIH) is discussed first, and, as noted, there seems to be an overlap of clinical features with patients diagnosed with sigmoid sinus wall anomalies.[3]

IDIOPATHIC INTRACRANIAL HYPERTENSION

IIH, known in the past as benign intracranial hypertension and pseudotumor cerebri, is a disorder primarily affecting obese women in the age range of 20 to 45 years. Its increasing incidence seems to be related to the ever-growing percentage of patients with morbid obesity.[4,5] The most common presenting symptoms are headache, followed by transient visual obscurations, back pain, PST, and visual loss, with approximately 10% of patients developing bilateral blindness.[5]

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PST is usually bilateral. Papilledema is an important clinical sign on ophthalmoscopic examination, although not necessarily present in all patients. Opening cerebrospinal fluid (CSF) pressure during lumbar puncture is typically greater than 25 cm H_2O. A diagnosis of IIH is made according to the modified Dandy Criteria (**Box 1**).

Several theories have been proposed to explain the presence of increased CSF pressure in these patients, including increased CSF production, decreased CSF absorption, increased interstitial fluid volume with resultant cerebral edema, and dural sinus stenosis.[4,6] Impairment of CSF absorption and compromise of dural sinus blood flow have been suggested to be interrelated.[7,8] There is still debate as to whether transverse sinus stenosis is the cause or result of IIH. Several reports have shown resolution of stenosis within the transverse dural venous sinus after intracranial pressure normalization via ventriculoperitoneal shunting, lumboperitoneal shunting, or lumbar puncture.[7,9–12] However, there are also reports showing persistent transverse sinus stenosis after normalization of the intracranial pressure.[13] A study using computed tomography (CT) venograms analyzed the morphology of the transverse dural sinus and its adjacent bony grooves in both patients with IIH and control subjects, with results suggesting a varied cause for sinus stenosis, including developmental or fixed narrowing and narrowing caused by compression secondary to increased ICP.[14]

Magnetic resonance (MR) imaging without and with contrast, including MR venogram (MRV), is the study of choice in the work-up for IIH. Orbit-specific sequences can aid in identification of the subtle ocular findings. Imaging findings associated with IIH are outlined in **Box 2** and **Fig. 1**. An interesting association exists between IIH and the presence of spontaneous CSF fistulas. In theory, the persistent increased intracranial pressure leads to the development of prominent arachnoid granulations within the skull base. When these prominent arachnoid granulations develop over pneumatized bone, bony thinning and disruption of the dura can occur.[15] Typical locations for CSF fistulas to develop include the tegmen tympani, floor of the middle cranial fossa, lateral wall of the sphenoid sinus, and cribriform plates.[16] Treatment options for IIH include weight loss/lifestyle modification, medical treatment with carbonic anhydrase inhibitors and inhibitors of growth hormone/insulinlike growth factor 1, CSF diversion, optic nerve sheath fenestration, and endovascular transverse sinus stenting (See: Hui FK, Abruzzo T, Ansari SA. Endovascular Interventions for Idiopathic Intracranial Hypertension and Venous Tinnitus-New Horizons, in this issue) (**Fig. 2**).[4,17,18]

Box 1
Modified Dandy Criteria for IIH

1. Signs and symptoms of increased intracranial pressure
2. No localizing symptoms on neurologic examination
3. Awake and alert patient
4. Absence of deformity, displacement, or obstruction of the ventricular system
5. No other cause of increased intracranial pressure
6. Normal neuroimaging except for typical findings of increased intracranial pressure

Data from Wall M, Kupersmith MJ, Kieburtz KD, et al. The idiopathic intracranial hypertension treatment trial: clinical profile at baseline. JAMA Neurol 2014;71(6):693–701.

SIGMOID SINUS WALL ANOMALIES

Sigmoid sinus wall anomalies (SSWAs) are an increasingly recognized diagnosis in the work-up of PST. This spectrum of anomalies includes a thin but intact sigmoid sinus plate, focal dehiscence of the sigmoid sinus plate, diverticulum formation with a focal protrusion of the vascular elements into the mastoid air cells, and ectasia with smooth bulging of the sigmoid sinus into the

Box2
Imaging features associated with IIH

1. Partially empty sella
2. Distal transverse dural sinus stenosis
3. Small or slitlike lateral and third ventricles
4. Distension and tortuosity of the optic nerve sheath
5. Optic nerve sheath enhancement secondary to venous congestion
6. Posterior globe flattening at the lamina cribrosa
7. Intraocular protrusion of the optic nerve head
8. Dural ectasia
9. Bony thinning and remodeling of the skull base
10. Spontaneous CSF fistulas or meningoencephaloceles

Data from Refs.[6,16,19]