Pulsatile Tinnitus Associated with a Laterally Placed Sigmoid Sinus

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Summary: We describe a case of subjective pulsatile tinnitus associated with a laterally placed sigmoid sinus. CT showed an enhancing eroding through the medial wall of the right mastoid. Two-dimensional time-of-flight MR angiography and conventional cerebral angiography revealed the tortuous and laterally deviated sinus.

Index terms: Hearing; Temporal bone, abnormalities and anomalies

Pulsatile tinnitus can have numerous causes. Although pathologic causes for the symptoms are always given strong consideration, normal variations should also be included in a possible differential diagnosis. We describe a case of pulsatile tinnitus associated with a laterally placed sigmoid sinus shown by computed tomography (CT), two-dimensional time-of-flight magnetic resonance (MR) angiography, and conventional cerebral angiography.

Case Report

A 36-year-old man had a 4- to 6-week history of intermittent right-sided tinnitus that was pulse synchronous as described by the patient. The patient had been in otherwise good health with no similar previous episodes and no history of trauma.

Physical examination revealed a normal tympanic membrane with no evidence of a vascular mass. No bruit was audible. No hearing deficit could be detected. Maneuvers suggested for evaluation of cervical venous hum such as slow head turning and mild pressure on the internal jugular vein (1) were not performed.

CT showed an enhancing abnormality in the region of the right sigmoid sinus. It extended to and caused thinning of the lateral wall of the right mastoid sinus (Fig 1A and B). Cerebral angiography showed markedly tortuous right sigmoid sinus (Fig 2) that drained into a normal right internal jugular vein.

Two-dimensional time-of-flight MR angiography was done with a 1.0-T unit for evaluation of the intracranial venous structures. The tortuous and laterally placed sigmoid sinus was again demonstrated (Fig 3A and B). No flow was seen within the left transverse and sigmoid sinuses.

Discussion

Possible causes of pulsatile tinnitus are many. Pathologic causes include paraganglioma, dural arteriovenous malformation, and acquired stenotic lesions of the carotid system (2, 3). Normal vascular variants as a cause of pulsatile tinnitus include an aberrant internal carotid artery, a dehiscent jugular bulb, and a high-riding, nondehiscent jugular bulb (3–5).

One entity that has been described many times in the otolaryngologic literature but often forgotten is the essential type of tinnitus or cervical venous hum. Essential tinnitus is nearly always pulse synchronous in nature because of the distinct pulsation in the intracranial venous system, as well as in the adjacent extracranial venous system (6). Essential tinnitus is thought to be most likely related to the distortion of laminar flow within the internal jugular vein after compression of the vein by the adjacent transverse process of the atlas and sternocleidomastoid muscle when the head is turned, usually away from the side of the tinnitus (1, 7).

The laterally placed sigmoid sinus has been described (8, 9). Knowledge of such a variant before surgical exploration or biopsy would be critical because of the potentially disastrous complications. Contiguity of the enhancing abnormality with the transverse sinus on CT may suggest this variant. In the present case, the abnormal CT findings and the lack of familiarity
with the entity along with uncertainty as to whether this truly was the cause of the tinnitus prompted further investigation with conventional cerebral angiography. It is debatable whether the sigmoid sinus abnormality could have been detected on a routine MR examination of the brain as the lesion was extremely difficult to discern on standard axial imaging, even in retrospect.

The exact cause of the tinnitus and its pulsatile nature are difficult to understand. It is most likely related to turbulent blood flow within the tortuous sigmoid sinus, as has been suggested in the case of a jugular megabulb deformity (10, 11) and similar to the turbulent flow expected within the internal jugular vein in the case of cervical venous hum.

Therapeutic options in such a situation are limited. Occlusion of the sigmoid sinus or ligation of the jugular vein has been suggested if an expanding vascular mass has been excluded and adequate contralateral venous drainage is
seen to be present (6, 11). In this case, MR and conventional angiography demonstrated no significant flow through the contralateral left transverse and sigmoid sinuses. Therefore, no definitive therapy was offered to the patient.

In summary, MR angiography and conventional cerebral angiography can be helpful in excluding a vascular anomaly, such as a laterally placed sigmoid sinus, when an expansile lesion is encountered in the region of the petrous bones. Such differentiation can be critical in avoiding potentially disastrous intraoperative complications.

References