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CASE REPORT

Occipital artery anastomosis to vertebral artery causing pulsatile tinnitus

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SUMMARY

Pulsatile tinnitus can result from various vascular etiologies that cause transmission of pulsatile turbulent flow into the inner ear. Less commonly, non-vascular sources cause increased blood flow and transmission of sound perceived as tinnitus. Thorough clinical examination leads to appropriate imaging evaluation and therapeutic planning. Most pulsatile tinnitus results from expected mechanisms, such as dural arteriovenous fistula, jugular bulb dehiscence, or paraganglioma; however, the literature contains reports of numerous rare causes, particularly variant anatomic morphologies. We present the case of a novel cause of pulsatile tinnitus in which collateral vascular flow compensated for decreased normal intracranial cerebral arterial supply and might have caused catastrophic consequences if intervened upon after assumptions based on an incomplete evaluation.

CASE PRESENTATION

A 63-year-old man with hypertension, diabetes mellitus, coronary artery disease with prior stenting, and a history of heavy tobacco use presented with chronic headaches and longstanding pulsatile tinnitus in the back of his head.[1–3] He reported that these sensations worsened when leaning forward, and physical examination yielded bruit but no thrill at the right mastoid process. He underwent MR angiography that demonstrated an out-pouching of the left posterior communicating artery (PComm) most likely representing an infundibulum but concerning for an aneurysm. He was referred for diagnostic cerebral angiography to assess the PComm and the possibility of a dural arteriovenous fistula (DAVF) as the source of the patient's pulsatile tinnitus.

Angiography of the left internal carotid artery (ICA) confirmed a PComm infundibulum, cross filling of the right anterior cerebral artery from the left ICA, and no flow in the right anterior cerebral artery from the ipsilateral ICA. A large right PComm was noted, and the dominant cervical right vertebral artery was occluded just beyond its origin. Left subclavian injection demonstrated a very small patent vertebral artery terminating at the posterior inferior cerebellar artery without connection to the basilar artery.

Right common carotid artery and external carotid artery injection showed no arteriovenous shunting. However, the right occipital artery connected to and supplied the C1 segment of the right vertebral artery, which then continued to supply the right posterior inferior cerebellar artery and basilar artery after its reconstitution (figure 1). This high flow occipital knot anastamosis between the external and internal carotid systems was deemed the source of the patient's symptoms.

TREATMENT

Intervention was not performed given the dependence of the posterior circulation on this anastomosis.

OUTCOME AND FOLLOW-UP

No further imaging analysis has been performed. The patient's symptoms persist.

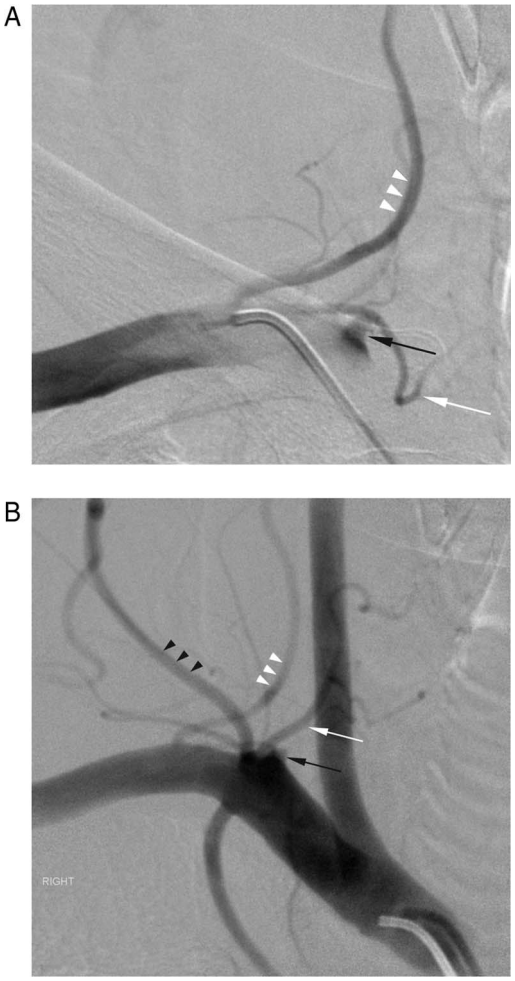

Figure 1 (A) Right subclavian artery and (B) right innominate artery injections in left anterior oblique projection demonstrate complete occlusion of the vertebral artery shortly beyond its origin (black arrow) and proximal to the origins of the inferior thyroidal (white arrow), deep cervical (white arrowheads), and suprascapular (black arrowheads) arteries.

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DISCUSSION

Auditory sensation without external stimuli is known as tinnitus, and this sensation can coincide with one's heartbeat, an entity known as pulsatile tinnitus.[1 4] Vascular etiologies typically cause the sensations, and history and physical examination play useful roles in localizing the source. Classically, pulsatile tinnitus is divided into objective and subjective groups, the former of which can be heard by an examiner.[4] While the diagnostic utility of this approach has been called into question, auscultation of murmur or palpation of thrill can expedite workup and localize potentially life threatening vascular lesions in the head and neck.[4 5] Furthermore, absence of tinnitus on compression of the ipsilateral carotid artery suggests a venous etiology, while persistence indicates an arterial source, which can further narrow the differential diagnosis and guide appropriate imaging workup.[6]

Vascular and non-vascular sources can cause pulsatile tinnitus. Classically, objective pulsatile tinnitus suggests a vascular etiology.[7] Most commonly, DAVF causes pulsatile tinnitus but other arterial pathologies include intracranial arterial aberrancy or redundancy, carotid cavernous fistula, atherosclerosis, vascular stenosis, arteriovenous malformation, aneurysm, dissection, and fibromuscular dysplasia.[1 8 9] Venous etiologies include jugular bulb abnormalities, dural sinus stenosis, and sinus aberrancy.[1 2 4 10] Such vascular etiologies tend to correlate with intracranial location, although thoracic or neck sources can occur.[1] Non-vascular causes include histiocytosis X, glomus tumor, high cardiac output, hydrocephalus, intracranial hypertension, tumor, myoclonus, and valvular heart disease.[1 3 4 7]

With the potential for catastrophic outcome possible with many of these lesions, swift imaging must be performed to complete the workup.[6 8] Additionally, the pulsatile tinnitus from some lesions, particularly DAVFs, can actually subside as they progress in severity and must not diminish the urgency with which evaluation should be completed.[7] History and physical guide the imaging approach. Although catheter angiography remains the gold standard, MR angiography or CT angiography have largely replaced catheter angiography in the early evaluation.[1] Other modalities, including general CT, MRI, or ultrasound, may play a diagnostic role depending on the lesions suspected.[3 4 7]

Turbulence due to increased flow or stenosis in vascular lesions transmits pulses to the inner ear.[1 11] Such turbulence increases or decreases with corresponding alterations in flow, causing changes in symptom intensity with maneuvers such as carotid compression or Valsalva, as seen in the patient presented here. Lesions involving the occipital artery transmit sound through the dense mastoid process to the inner ear.[6] Pulsatile tinnitus has been reported from lesions similar in location but distinct in features from those found in the patient presented here. Cowley *et al*[11] reported an aberrant occipital artery with retrograde flow supplying the ICA. After carotid stenting, flow reversed to the normal direction, and symptoms resolved.[11] Lehmann *et al*[6] described a left vertebral anastamosis to branches of the occipital artery to supply the arm after near total occlusion of the left subclavian artery.[6] Subclavian artery stenting resolved the tinnitus. In the patient presented in this report, antegrade flow in the occipital artery supplied the majority of the posterior circulation. We postulate that progressive stenosis and eventual complete occlusion of the right vertebral artery necessitated collateralization from the external carotid system (figures 2, 3).

The anastomosis in our patient appears to be a typical occipital knot, the persistent potential connection between the occipital artery and C1 vertebral artery posterior and superior to the posterior arch of C1. The occipital artery is a remnant of the type I and type II proatlantal arteries, which are the embryologic C1 and C2 segmental arteries.[12] These remnants connect the occipital artery to the vertebral artery via C1 and C2 radicular branches. The caliber change between the occipital artery, the tortuous radicular anastomotic segment, and the C1 vertebral artery in our patient is more compatible with a persistent occipital knot anastomosis than with a congenital type II proatlantal intersegmental artery (also previously described as a source of objective pulsatile tinnitus), in which there typically is not a significant caliber change along the persistent occipitovertebral anastomosis.[13]

Imaging often identifies treatable sources of the tinnitus, generally with good results after appropriate treatment, which can include endovascular intervention or surgical removal or repair.[1 2 4 7] While prompt assessment and treatment can prevent a tragic outcome, the clinician must exercise diligence in assessing the source of pulsatile tinnitus to avoid errantly eliminating vital compensatory anomalies and causing equally tragic complications.

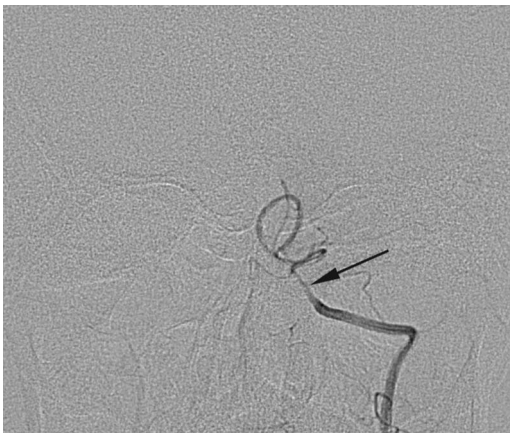

Figure 2 Left subclavian artery injection in anteroposterior projection demonstrates a diminutive vertebral artery (arrow) with termination at the posterior inferior cerebellar artery and no connection to the basilar artery.

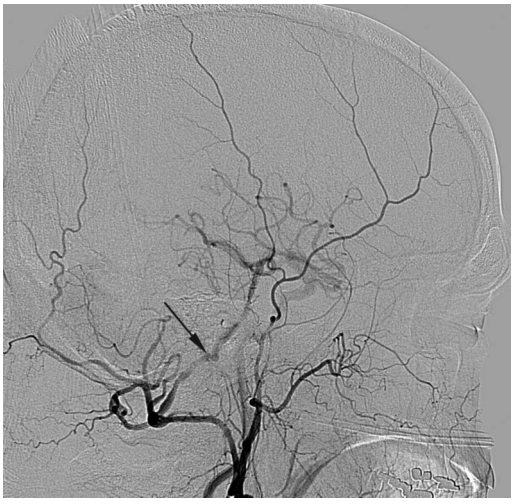

Figure 3 Right external carotid artery injection in lateral projection demonstrates anastomotic connection (arrow) between the occipital artery and reconstituted basilar artery.

Learning points

- The differential diagnosis for pulsatile tinnitus includes arteriovenous fistulae, vascular stenosis, arteriovenous malformations, jugular bulb abnormalities, glomus tumors, and high cardiac output.
- Rare etiologies may cause pulsatile tinnitus, some of which may not be pathologic.
- Diagnostic cerebral angiography plays an important role in the assessment of pulsatile tinnitus. In this case, this modality demonstrated a previously unidentified anastomosis that was critical for arterial supply to the posterior fossa.

Contributors MDA authored the paper. JE and SWH participated in patient care and edited the paper.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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