PULSATILE TINNITUS: A HARBINGER OF A GREATER ILL?

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Abstract: Background. Pulsatile tinnitus is an uncommon otologic symptom, which may be the presenting complaint of a potentially devastating pathology. Understanding this manifestation as a possible symptom of a significant vascular abnormality is crucial to guide management and treatment.

Methods and Results. We describe a 38-year-old woman with sudden-onset right-sided pulsatile tinnitus. A right extracranial internal carotid artery (ICA) dissection was diagnosed with MRI/magnetic resonance angiography (MRA) and treated with anticoagulation. Follow-up MRI/MRA demonstrated complete resolution. Two months later, left-sided pulsatile tinnitus evolved. An MRI/MRA of the neck demonstrated left-sided extracranial ICA dissection. She was treated in a similar fashion and a repeat MRI/MRA demonstrated its resolution.

Conclusion. Spontaneous extracranial ICA dissection may present with pulsatile tinnitus as the only symptom in 4% to 50% of patients. Subsequent evolution of a contralateral dissection is even more uncommon. Generally, treatment of this phenomenon is conservative utilizing anticoagulation or aspirin; however, surgical intervention may be necessary.

Keywords: pulsatile tinnitus; tinnitus; carotid artery dissection; internal carotid artery dissection; spontaneous carotid artery dissection

Spontaneous extracranial internal carotid artery (ICA) dissection is an extremely uncommon manifestation. The estimated incidence of all etiologies of extracranial ICA dissection is 2.6:100,000 with an average age of presentation of 40 to 45 years.1,2 Spontaneous extracranial ICA dissection is an unusual event that may evolve asymmetrically or may cause a milieu of cerebrovascular deficits. Pulsatile tinnitus may be the only clue indicating an underlying pathology in 4% to 50% of affected patients.1,3,4 We present a 38-year-old woman who was seen with a right-sided pulsatile tinnitus and was diagnosed with a right-sided extracranial ICA dissection. After effective treatment and resolution of this process, she then developed a left-sided pulsatile tinnitus and MRI and magnetic resonance angiography (MRA) revealed a left-sided extracranial ICA dissection. This disease process is well described in the neurology and vascular surgery literature. Otolaryngologists should be aware of this rare vascular pathology as an underlying cause of pulsatile tinnitus.

CASE REPORT
A previously healthy 38-year-old woman was seen with a 2-week history of right ear tinnitus, which she described as pulsatile and "swishing" in nature. On physical examination, both tympanic membranes were sclerotic and a bruit was auscul-
tated over the mid right neck. There were no neck masses palpated, neurologic deficits, or cardiac murmurs appreciated. There was no hearing deficit, and she denied any headaches, recent illnesses, recent neck manipulations, amphetamine/cocaine abuse, or history of trauma. She had no personal or family history of Ehlers Danlos syndrome, Marfan’s syndrome, polycystic kidney disease, aortopathy, or osteogenesis imperfecta.

Initial tympanograms were flat bilaterally. A contrast-enhanced CT of the neck showed focal thinning of the carotid canals, which was more pronounced on the right (Figure 1). A contrast-enhanced MRI/MRA of the brain was negative for acute intracranial process. MRA of the neck revealed an extracranial right ICA dissection beginning in the mid portion and extending just proximal to the petrous portion, with an associated 50% to 60% stenosis. She was started on anticoagulation medications and a repeat MRA obtained 3 months later showed resolution. On exam, her bruit and subjective tinnitus had resolved as well.

One month later, she reported with similar symptoms in her left ear along with 1 episode of transient right hand weakness and clumsiness, and left eye blurry vision. Repeat imaging revealed left ICA dissection and was once again started on anticoagulation for 3 months (Figure 2). Follow-up MRI/MRA demonstrated complete resolution. Further workup with ultrasound and abdominal CT revealed normal renal arteries and aorta. Echocardiograph was normal. Laboratory
studies for α-1-antitrypsin, heme profile, complete metabolic panel, erythrocyte sedimentation rate, creatine kinase, and serum lipid panels were all within normal limits. She was switched from warfarin and began long-term daily aspirin. She remains symptom free to this day.

**DISCUSSION**

Pulsatile tinnitus is an uncommon otologic symptom, which may be the presenting complaint of a potentially devastating pathology. Individuals may present with subjective tinnitus which only they may appreciate or with objective tinnitus recognized by both the patient and examining physician. The sound of pulsatile tinnitus is due to turbulent, nonlaminar flow of blood, which is transmitted to the organs of hearing in the inner ear. These individuals may have varying degrees of both onset and duration of tinnitus after arterial dissection. An understanding of this manifestation along with a careful history and physical examination is crucial to guide management and treatment of this disease. Extracranial ICA dissection may occur spontaneously, or more commonly secondary to trauma.

When symptomatic, patients may be seen with a variety of symptoms including Horner’s syndrome, dysgeusia, pulsatile tinnitus, headache, neck or facial pain, cranial neuropathies, and manifestations of cerebral ischemia such as hemiparesis, amaurosis fugax, dysphagia, and aphagia. Fortunately, regardless of the severity of presentation, extracranial ICA dissection has a rather benign course. Greater than 75% of patients recover completely, and only 6% to 8% of patients suffer with moderate to severe neurological deficits.

Pulsatile tinnitus may be the presenting complaint of a very significant and possibly life-threatening etiology. The most common diagnostic algorithm divides pulsatile tinnitus into 2 categories: vascular and nonvascular (Figure 3). Vascular can further be divided into arterial and venous etiologies. Vascular pathology may include cerebral aneurism, arterial dissection, both intracranial and extracranial arterial-venous malformations, carotid cavernous fistulae, or dural venous fistulae. Nonvascular sources of pulsatile tinnitus may include Paget’s disease, histiocytosis x, increased cardiac output, Ménière’s disease, cholesterol granuloma, and palatal myoclonus. Out of the vast etiologies included in the differential, the 3 most common causes of pulsatile tinnitus are benign intracranial hypertension syndrome (pseudotumor cerebri), atherosclerotic carotid artery disease, and glomus tumors, respectively.

To avoid delay in diagnosis, a high degree of suspicion and early radiographic studies are critical. A clear relationship between the causal factors and extracranial ICA dissection remains to be

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**FIGURE 3.** This diagnostic algorithm delineates the common causes of pulsatile tinnitus of both vascular and nonvascular origin. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com.]
elucidated. Current literature suggests predisposing factors may include arterial hypertension, prolonged physical strain or sudden severe strain as in whiplash, minor head or neck trauma, fibromuscular dysplasia, atherosclerotic plaque, oral contraceptive use, connective tissue disorders, hyperhomocysteinemia, and additional genetic causes.4,6

The suspected mechanism of dissection is via an intimal tear in the blood vessel which provides a path for intraluminal blood to dissect longitudinally through the media. The dissection may either enter the vessel lumen, possibly occluding it, or create a pseudoaneurysm. A transient ischemic attack or stroke may result from either luminal occlusion or from the newly created lumen serving as a source for emboli.

Although history and physical examination remain the mainstay of diagnosis, additional modalities are often needed to aid in diagnosis. Evaluation of a patient with pulsatile tinnitus should discern the character of tinnitus, onset, location, factors which alleviate and exacerbate symptoms, and associated symptoms such as hearing changes, vertigo, and auricular fullness. Routine head and neck examination should be performed with special attention to otoscopy for careful evaluation of middle ear pathology and auscultation of the great vessels for bruits. Initial evaluation should also include pure-tone and speech audiologic testing to rule out cerebellopontine angle pathology, otosclerosis, Ménière’s disease, or noise-induced hearing loss. Contrast-enhanced CT of the neck is usually the initial radiographic study of choice and may visualize many vascular anomalies. CT of the temporal bone is also utilized to delineate pathology within the region including aberrant carotid artery, persistent stapedial artery, and paragangliomas of the jugular fossa and middle ear. However, to ensure several vascular anomalies including arteriovenous fistulas and carotid artery dissections are not overlooked, further radiographic workup should include MRI/MRA of the neck.

MRI with MRA is an excellent technique for evaluation because of the high degree of soft tissue contrast with high sensitivity.7 MRI and MRA will provide noninvasive detailed views of an intramural hematoma, arterial dissection, and surrounding vessels. Typical features on T-1 weighted images are a hyperintense crescent-shaped area that increases the external diameter of the vessel while narrowing its lumen resulting in a signal flow void. Radiographically, the pathognomonic findings of an extracranial ICA dissection are a false or double lumen and an intimal flap. However, more common findings include an aneurysmal dilation, a tapering, or stenosis of the lumen referred to as the “string sign,” and arterial occlusion.8,9 CT and CT angiogram also are sensitive and specific sources of imaging for evaluation with similar findings.10,11 Four vessel angiography is an additional modality for evaluation; however, because of its invasive nature it is generally reserved for diagnostically difficult cases or as a complement to endovascular management of unstable dissections.9

The treatment of extracranial ICA dissection is controversial, and data from therapeutic trials is lacking. Early anticoagulation with heparin followed by warfarin and/or aspirin for a 3- to 6-month-period is the most common medical approach.12 A longer regimen may be necessary with evidence of residual vascular disease. Of note, anticoagulation is contraindicated in patients with subarachnoid hemorrhage, intracranial extension, pseudoaneurysm, or additional medical conditions.1,5,12,13 In addition, symptomatic relief of the patient’s perception of tinnitus may be assisted with ambient sound generators.

While a majority of patients improve clinically and radiographically with medical management alone, multiple surgical interventions are described for patients in whom medical management does not suffice. These patients typically have recurrent symptoms or dissections, contraindications to anticoagulation, an expanding pseudoaneurysm, or significant deficiency in cerebral circulation.14 Surgical approaches involve vascular ligation, endarterectomy with patch angioplasty, bypass techniques, and endovascular stenting, coiling, embolization, and aneurism trapping. These techniques may prove technically challenging due to the anatomy, and each has their own associated morbidity and mortality rates.

Tinnitus is a common presenting complaint in an otolaryngologist’s practice. When it is pulsatile in nature, a high index of suspicion and early vascular evaluation with angiography is crucial to avoid delays in diagnosis and treatment. Upon ascertaining the diagnosis, expeditious consultation of vascular surgery or neurology is appropriate due to the urgent nature of the diagnosis and importance of early treatment. A majority of patients with extracranial ICA dissection will have resolution of their symptoms with angiographic normalization of their vessels4; however, the possibility of permanent morbidity or mortal-
ity exists. Because the risk of contralateral dissec-

tion is 2% in the first month and 1% per year thereafer, and the risk of ipsilateral recurrence is rare,12 routine follow-up angiography should be utilized to help guide future treatment decisions and management options.

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