Pulsatile Tinnitus

Aristides Sismanis, M.D., F.A.C.S.
Medical College of Virginia, Virginia Commonwealth University, Department of Otolaryngology-Head & Neck Surgery, Richmond, VA

Pulsatile tinnitus is an uncommon otologic symptom and always deserves a thorough evaluation. In the evaluation history, microotoscopy, auscultation and neuro examination are included.

Auscultation is performed with a modified electronic stethoscope preferably in a sound proof room. Application of digital pressure over the lateral/carotid side of the neck and compressing the jugular vein is very important to determine between arterial or venous type of pulsatile tinnitus. If the tinnitus goes away this is of the venous type. Auscultation of the head, especially behind the ear is important. One should not forget that most of the arterial venous fistula occur in this area.

The audiologic testing is, of course, very important. This is the audiogram (not shown) of a patient with a venous type tinnitus, where you can see a low frequency hearing loss. The same test was performed while the patient was applying pressure over each lateral aspect of the neck which eliminated the masking effect of the tinnitus.

In the evaluation, neuro-ophthalmology consultation is often included. If there is a problem with the carotid artery such as arteriosclerotic artery disease, a vascular surgery consultation is required.

This diagram (not shown) depicts our evaluation. If the examination shows a retrotypanic mass, we proceed at my institution with a CT Scan of the temporal bone which may establish the diagnosis of a glomus tympanicum or jugular bone abnormalities. If you find any of these causes then there is no need for further evaluation. If the CT shows a glomus tumor, and especially if the patient is a surgical candidate, MRI tomography is required.

Most patients fall into this category with normal otoscopy. If you suspect that the patient has increased intracranial pressure, such as the patient who is young, female, and obese, we order an MRI/MRA. We send the patient for a neuro-ophthalmologic evaluation. Diagnosis of a benign intracranial hypertension syndrome is established with lumbar puncture and measurement of the CSF pressure. Other diagnoses which you can derive from an MRI/MRA is hydrocephalus and abnormal lateral sinus. If the patient is suspected of carotid artery abnormalities such as an older individual with previous history of heart attack or hypertension, we proceed with Doppler ultrasound which can confirm the diagnosis of atherosclerotic carotid artery disease. Some of the patients that we have seen with pulsatile tinnitus reflect carotid plaque formation. If this is normal then we proceed with MRI/MRA. When do you do the carotid angiogram? If the patient has a bruit or objective tinnitus I tend to obtain a carotid angiogram. But not everybody with pulsatile tinnitus gets a carotid angiogram. Our results show that most of the patients were diagnosed as having benign intracranial hypertension syndrome followed by atherosclerotic carotid artery disease and glomus tumors. We received various diagnoses of increased intracranial pressure from various etiologies.

Finally you can read the slides (not shown) on 13 patients on whom no diagnosis could be achieved. We call this group idiopathic. Now let me go over the groups of patients. In both BIH groups, almost all of them, except two females, were young and most of them obese, some of them morbidly obese, 39 blacks, and 17 whites. In the majority of patients, the right ear was involved twice as often as the left. Tinnitus was objective in 40 and subjective in 16, and 17 patients had visual disturbances because of papilledema. Not every patient with benign intracranial hypertension syndrome has papilledema. And unfortunately neurologists in the United States do not make the diagnosis of this syndrome if they don't see papilledema. So please insist that you don't have to have a papilledema to have increased intracranial pressure.

BIH syndrome is an idiopathic syndrome characterized by increased intracranial pressure in the absence of focal neurologic signs with exception of occasional 6th and 7th nerve pulses, but I have seen a patient with 5th nerve involvement. Otitic hydrocephalus and idiopathic intracranial hypertension are old terms. These are the classic manifestations which the neurology textbooks describe. It is obvious that there is a group of patients that will come to you and not to a neurologist because of headaches combined with other symptoms such as hearing loss, dizziness, oral fullness and visual disturbances. This is a typical patient with BIH. Treatment of this syndrome is, of course, weight reduction and some patients must have to undergo gastric bypass surgery, if they fail to respond to conservative treatment.

Atherosclerotic carotid artery disease was diagnosed in 24 patients, most of them female, older individuals. Objective tinnitus was present in 12 patients. Almost all of them except one had an ipsilateral neck bruit. The person without the bruit had stenosis in the carotid artery intracranially.

The glomus tumors group were all females. Finally five patients were either on betablocker inhibitors or calcium channel blockers; and tinnitus developed soon after initiation of treatment probably because of decrease of peripheral resistance.

Three patients were diagnosed with arteriovenous fistula and finally the idiopathic group, most of them females, had subjective tinnitus.

In conclusion, the history and physical examination are of the utmost importance. Diagnostic testing should be individualized and there is no need to do a carotid angiogram on every patient. BIH syndrome was the most common diagnosis. The majority of these patients had a treatable underlying etiology.