

Neuroradiologic Assessment of Pulsatile Tinnitus

Matthew L Kircher, MD,

Robert T Standing, MD

John P Leonetti, MD

Department of Otolaryngology– Head and Neck Surgery

Loyola University Medical Center, Maywood, IL: Dr Kircher and Dr Leonetti

Department of Otolaryngology-Head and Neck Surgery

Henry Ford Health System, Detroit, MI: Dr Standing

Corresponding Author: Matthew L Kircher, MD

Department of Otolaryngology – Head and Neck Surgery

Loyola University Medical Center

2160 South First Avenue, Maywood, IL 60153

Phone number: 708 216-1676

Fax Number: 708 216-4834

E-mail address: matthewkircher@gmail.com

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ABSTRACT

Objective: To present neuroradiographic imaging data on patients presenting with pulsatile tinnitus without other directed physical examination findings.

Study Design: Retrospective chart review.

Subjects and Methods: Patients presenting to a tertiary care academic medical center from July 1993 to July 2007 with a chief complaint of pulsatile tinnitus without other directed physical examination findings. Neuroradiographic imaging data were analyzed.

Results: In subjective pulsatile tinnitus, 71% (40/56) of patients had no radiographic findings on magnetic resonance and 63% (10/16) of lesions ultimately identified were still evident with magnetic resonance alone. In objective pulsatile tinnitus, cerebral angiography diagnosed lesions in all patients undergoing angiography and magnetic resonance missed 50% (3/6) of lesions associated with objective pulsatile tinnitus.

Conclusion: Magnetic resonance may be considered a first line diagnostic imaging modality in the assessment of subjective pulsatile tinnitus. However, in patients with objective pulsatile tinnitus, clinicians may choose to proceed directly to cerebral angiography.

INTRODUCTION

The auditory perception of rhythmic noise synchronous with the heartbeat is defined as pulsatile tinnitus. Pulsatile tinnitus is classified into objective and subjective subtypes. These subtypes refer to the ability or inability respectively of the examiner to auscultate a vascular bruit in the periauricular or cervical neck region. Objective tinnitus can result from turbulent or aberrant blood flow or palatal myoclonus^{1,2}. Turbulent arterial flow can be caused by arteriovenous fistula (AVF), carotid stenosis, glomus tumor, arteriovenous malformation (AVM), aberrant carotid artery or cardiac murmur³⁻⁶. A venous hum contributing to pulsatile tinnitus can also be caused by either venous anomalies or raised intracranial pressure³.

Magnetic resonance imaging (MRI) and cerebral angiography are imaging modalities often used to identify vascular lesions responsible for pulsatile tinnitus. MRI is a commonly used noninvasive imaging modality that can be further enhanced by the use of magnetic resonance angiography (MRA)⁷.

Cerebral angiography is a more definitive but invasive test. Practitioners separate pulsatile tinnitus into objective and subjective categories as a means of selecting patients with a higher likelihood of anatomic pathology. Objective pulsatile tinnitus is routinely evaluated with more invasive tests such as cerebral angiography because of the perceived higher likelihood of identifying anatomic lesions, and subjective pulsatile tinnitus is often evaluated with less invasive tests such as MRI/MRA. However, many of the neuroradiologic findings seen in pulsatile tinnitus can also be seen in asymptomatic patients or in patients with contralateral complaints indicating the need for clinical correlation in interpreting these studies.

All patients presenting with a complaint of pulsatile tinnitus require a thorough history and physical examination. Sismanis et al have developed an algorithm to guide the judicious use of diagnostic tests and imaging given the clinical suspicion³. This algorithm begins with a thorough history and physical exam paying close attention to otoscopic and ophthalmoscopic findings, cranial nerve examination, carotid auscultation and cardiac examination. Computed tomography (CT), MRI/MRA, cerebral angiography, carotid duplex, echocardiogram and lumbar puncture (LP) are then utilized appropriately in this algorithm to support clinical suspicion. In this management scheme, patients with pulsatile tinnitus that do not have findings on otoscopy or signs of elevated intracranial pressure are evaluated neuroradiographically based on the degree of clinical suspicion of the treating practitioner.

This study will review the clinical presentation and neuroradiologic imaging data in patients presenting with subjective or objective pulsatile tinnitus without other directed physical examination findings in an attempt to identify the most optimal neurodiagnostic imaging study in this group of patients.

METHODOLOGY

Following approval by the Loyola Research Institutional Review Board, a retrospective chart review of patients presenting to our tertiary care academic medical center from 1993 to 2007 with a chief complaint of tinnitus was conducted. Patients were identified by database search for ICD-9 code 388.30 for tinnitus. Chart review selected out patients from this cohort with pulsatile tinnitus. Neuroimaging data including MRI and/or cerebral angiography studies was collected. Patients excluded from analysis included those with a previously known diagnosis of glomus tumor or with certain physical examination

findings such as an abnormal otoscopic examination, a cervical carotid bruit or clinical signs of elevated intracranial pressure.

All patients underwent a thorough history and physical examination including otoscopy and auscultation of the mastoid, head, neck, and chest, and were evaluated by MRI and/or cerebral angiography at the discretion of the senior author (JPL). MRI was typically the initial neurodiagnostic study chosen before proceeding to cerebral angiogram, and MRA was largely not utilized again at the discretion of the senior author. Tinnitus was defined as pulsatile when the patient described sounds synchronous with the heartbeat. Pulsatile tinnitus was classified as subjective if inaudible to the examiner and as objective when audible to the examining physician. Clinical presentation and neurodiagnostic imaging data were analyzed.

RESULTS

Nine hundred and seventy-six patients were evaluated for a chief complaint of tinnitus by the senior author between July 1993 and July 2007. One hundred and six of these patients were diagnosed with pulsatile tinnitus after careful examination, the rest were found to have nonpulsatile subjective tinnitus. Of the patients with pulsatile tinnitus, 21 were male and 85 were female with age ranging from 19-93 years. The mean age at presentation was 56 years. Pulsatile tinnitus was found to be right-sided in 51 patients, left-sided in 43 patients and bilateral in 12 patients. Of the one hundred and six patients identified, 91 patients presented with subjective pulsatile tinnitus and 15 patients presented with objective pulsatile tinnitus.

In 91 patients with subjective pulsatile tinnitus, 9 patients were excluded from data analysis due to directed physical examination findings. Three patients were noted to have CSF otorrhea on physical examination, 1 patient was diagnosed with encephalocele and the other 2 were diagnosed with meningocele on imaging and operative exploration. Two patients with carotid vascular bruit in the neck underwent ultrasonography and were referred to vascular surgery for evident atherosclerotic carotid artery disease, and 4 patients were noted to have glomus tumor on otoscopic examination and were excluded from data analysis. Of the remaining 82 patients with subjective pulsatile tinnitus, 54 were evaluated by MR, 25 patients were evaluated by cerebral angiography and 24 patients were evaluated by both MR and cerebral angiography. One patient could not undergo MR because of metallic

orthodontics and was evaluated by cerebral angiography alone with no identifiable pathology. Of patients with subjective pulsatile tinnitus and no directed physical examination findings undergoing imaging, 29% (16/56) had radiographic findings to explain pulsatile tinnitus (Table I). Identified pathology in the subjective pulsatile tinnitus group included dural sinus stenosis, arteriovenous malformation (AVM), arteriovenous fistula (AVF), vascular loop, jugular bulb abnormality, aberrant internal carotid artery and intracanalicular acoustic neuroma (Table II).

Of the 24 patients with subjective pulsatile tinnitus undergoing both MR and cerebral angiography, 50% (12/24) had positive radiologic findings to explain pulsatile tinnitus, although 6 false negative MR studies were performed when compared to cerebral angiography. Specifically, 3 AVF, 2 AVM and 1 dural sinus stenosis were not detected on MR. Lesions in patients with vascular loop pathology, aberrant internal carotid artery, jugular bulb abnormality, intracanalicular acoustic neuroma and all but one dural sinus stenosis were detected with MR. Of the patients with jugular bulb abnormality, 1 patient was noted to have a high riding jugular bulb and the other was diagnosed with a dehiscent jugular bulb with an anomalous course.

In 15 patients with objective pulsatile tinnitus, 2 were excluded due to directed physical examination findings. Cervical carotid bruit was present in both patients and atherosclerotic carotid artery disease and carotid body tumor were diagnosed separately on further workup. All 13 patients with objective pulsatile tinnitus and no other directed physical examination findings were evaluated neuroradiographically, 85% (11/13) were evaluated by MR, 46% (6/13) were evaluated by cerebral angiography and 31% (4/13) were evaluated by both MR and cerebral angiography (Table III). Greater than one half or 54% (7/13) of these patients had positive neuroradiologic findings. Five patients were diagnosed with AVM and all underwent MR study. MR identified AVM in 3 patients but failed to identify 2 other AVM lesions. The two negative MR studies were followed with positive cerebral angiogram studies. One patient identified with dural sinus stenosis underwent a false negative MR before cerebral angiography confirmed the diagnosis. In all there were 3 false negative MR studies when compared to cerebral angiography in patients with objective pulsatile tinnitus. In contrast, two patients with AVM were diagnosed with MR only, declining further characterization of lesion by cerebral angiography.

The most commonly identified pathology in objective pulsatile tinnitus was AVM (Figure I) with 5 patients. Of these 5 patients, only 1 underwent both MR and cerebral angiography. Atherosclerotic

carotid artery disease was diagnosed in a patient without directed physical examination findings on cerebral angiogram alone (Table IV). The most common lesion with subjective and objective subtypes combined was AVM with 8 patients diagnosed (Table V).

DISCUSSION

Pulsatile tinnitus may be due to a variety of neoplastic or vascular causes and these lesions may cause significant morbidity and mortality if left untreated^{7,8}. Diagnostic algorithms in the approach to patients with pulsatile tinnitus have been well described by Sismanis³. A systematic approach to the history and physical examination is required with retrotympanic masses requiring CT of the temporal bone and/or neck to evaluate for glomus tumor, aberrant carotid artery or jugular bulb abnormality. In patients with no retrotympanic mass, evaluation for increased intracranial pressure and carotid/cardiac abnormality is recommended. A combination of neuro-ophthalmologic evaluation, MR and/or lumbar puncture are utilized to evaluate for benign intracranial hypertension (BIH). Suspected carotid and cardiac abnormalities undergo duplex ultrasound or echocardiogram to evaluate for atherosclerotic carotid artery disease, carotid tortuosity or valvular heart defect. In the absence of BIH, MR findings or carotid/cardiac abnormality, cerebral angiography may be necessary to evaluate for dural AVM or AVF. However, in patients with an otherwise unremarkable physical examination the choice between MR and cerebral angiography is not always apparent. This uncertainty is due in part to the relatively rare nature of this disease process. In our 15 year experience, 108 patients were identified with pulsatile tinnitus in over 900 patients presenting with a chief complaint of tinnitus. Again, this review has excluded those patients presenting with a known neoplastic or vascular lesion such as glomus or carotid body tumor as this information would dictate the neuroradiographic study chosen. We present our experience with subjective and objective pulsatile tinnitus with an otherwise normal physical examination in the hopes of further refining evidence-based selection of neuroradiographic study in this subgroup of patients.

In patients with objective pulsatile tinnitus, the work-up, including physical examination and/or imaging, often discloses an abnormality⁹. Cerebral angiography is readily utilized in these patients as dural AVM or AVF has been shown to be the most frequent cause of objective pulsatile tinnitus in the patient with a normal otoscopic examination¹⁰. In this review, AVM was not only the most common

lesion seen in objective pulsatile tinnitus, but also the most common lesion overall in objective and subjective subtypes combined. In patients with subjective pulsatile tinnitus, the less invasive MR is often favored over cerebral angiography as this modality has shown useful diagnostically with rates as high as 63% in defining anatomic abnormalities contributing to pulsatile tinnitus^{7,11}. Our data shows radiographically defined anatomic abnormalities contributing to subjective pulsatile tinnitus in 29% (16/56) of patients, excluding those with directed physical examination findings. Clinical correlation must be used in interpreting neuroradiologic findings seen in pulsatile tinnitus as these findings can also be seen in asymptomatic patients or in patients with contralateral complaints. Such discrepancies have been described with vascular loop pathology causing pulsatile tinnitus. Studies have shown conflicting evidence to support the theory of anterior inferior cerebellar artery causing eighth nerve compressive pulsatile tinnitus^{12,13}. In this study, vascular loop pathology was a diagnosis of exclusion after no other identifiable clinical or radiographic pathology was evident. Fortunately none of these patients had debilitating tinnitus complaints and decompressive surgery was not attempted. Likewise jugular bulb abnormality can be an incidental finding in asymptomatic individuals but was seen as a cause for pulsatile tinnitus in 3 patients in this study as a diagnosis of exclusion^{14, 15, 16}. All patients with vascular loop or jugular bulb abnormality pathology were managed nonoperatively.

In patients with subjective pulsatile tinnitus with no directed physical examination findings, 29% (16/56) had radiographic findings to explain pulsatile tinnitus while 71% (40/56) had no radiographic findings. Not all patients were evaluated with cerebral angiogram and/or MR, and this is a major limitation of this retrospective study. Identified pathology included dural sinus stenosis, AVM, AVF, vascular loop, jugular bulb abnormality, aberrant internal internal carotid artery and intracanalicular acoustic neuroma (Table I). MR and cerebral angiogram identified pathology responsible for pulsatile tinnitus in 18% (10/56) and 38% (10/26) of cases respectively. Forty-six patients had negative MR studies but 6 of these studies were false negative when compared to cerebral angiography. Again, not all patients undergoing MR had cerebral angiography performed in addition and so generating sensitivity and specificity for further comparison is problematic. Lesions with false negative MR detected on cerebral angiography included 3 AVF, 2 AVM and 1 dural sinus stenosis. One vascular loop and 1 jugular bulb abnormality were detected on MR with a negative cerebral angiogram although these lesions are perhaps less likely to be life-threatening. These numbers may represent the complementary nature of

MR and cerebral angiogram in evaluating subjective pulsatile tinnitus. Although cerebral angiogram was required to identify 6 lesions responsible for vascular tinnitus, clinicians will try to avoid a more invasive test if MR will give valuable information. MR still seems an appropriate initial diagnostic modality in subjective pulsatile tinnitus without other directed physical examination findings as 71% of patients imaged had no radiographic findings on MR and 63% (10/16) of lesions ultimately identified were still evident with MR alone. Further evaluation of subjective pulsatile tinnitus with cerebral angiography may be reserved for those with debilitating subjective pulsatile tinnitus and negative MR.

In this study, an abnormality was identified in 60% (9/15) of all patients presenting with objective pulsatile tinnitus. Two patients with cervical carotid bruit were excluded from analysis, 1 with carotid body tumor and 1 with atherosclerotic carotid artery disease. Although one patient included in analysis also had atherosclerotic artery disease noted on cerebral angiography, inclusion rested on the fact that a vascular bruit was auscultated at the ipsilateral mastoid as opposed to the lower cervical level. Five patients were diagnosed with AVM and 1 with dural sinus stenosis. MR identified the lesion in 3 patients but failed to identify AVM and dural sinus stenosis in 3 other patients. These 3 false negative MR studies were followed with positive cerebral angiogram studies. Two patients with AVM diagnosed on MR declined further characterization of lesion by cerebral angiography. Given the small number of lesions and lack of uniform radiologic assessment it is difficult to generalize these findings. We can see that cerebral angiography appears effective in diagnosing lesions associated with objective pulsatile tinnitus and it would presumably show lesions in 2 patients diagnosed with AVM on MR that declined cerebral angiography. MR on the other hand missed 50% (2 AVM, 1 dural sinus stenosis) of lesions responsible for objective pulsatile tinnitus. Others have reported on similar patterns, where CT and MR were relatively insensitive in the detection of dural AVF when compared to cerebral angiography^{11,17}. Shin et al recommended that in the absence of objective pulsatile tinnitus, MR is an appropriate initial diagnostic step¹¹. In the patient with an objective bruit, the clinician may choose to proceed directly to cerebral angiography for adequate evaluation, and our data support this clinical approach.

The retrospective nature of this study limits the reliability of these findings as patients were evaluated with MR and cerebral angiography based on clinical suspicion rather than formal criteria. Future prospective studies evaluating pulsatile tinnitus with standardized criteria for selecting neuroradiographic study will be of benefit. Also as noninvasive CT and MR angiography capabilities

advance and become readily available, cerebral angiography may be required less frequently in evaluating pulsatile tinnitus.

CONCLUSION

Magnetic resonance may be considered a first line diagnostic imaging modality in the assessment of subjective pulsatile tinnitus. However, in patients with objective pulsatile tinnitus, clinicians may choose to proceed directly to cerebral angiography. Future prospective studies are needed to better delineate the optimal neuroradiologic study in the assessment of pulsatile tinnitus.

AUTHOR CONTRIBUTIONS

Matthew L. Kircher majority author, chart review and analysis

John P. Leonetti original designer of study, clinician, senior author

Robert T. Standring data collection, minor author

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None

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Figure 1 Cerebral angiogram illustrating a left sigmoid sinus dural arteriovenous malformation (see black arrow) in a patient with left occipital bruit and objective pulsatile tinnitus.



Table I Radiographic findings in patients with subjective pulsatile tinnitus.

Patient	Diagnosis	Cerebral Angiogram	MR
1	Arteriovenous fistula	+	-
2	Arteriovenous fistula	+	-
3	Arteriovenous fistula	+	-
4	Arteriovenous malformation	+	+
5	Arteriovenous malformation	+	-
6	Arteriovenous malformation	+	-
7	Dural Sinus Stenosis	+	+
8	Dural Sinus Stenosis	+	-
9	Dural Sinus Stenosis	∅	+
10	Vascular Loop	∅	+
11	Vascular Loop	-	+
12	Vascular Loop	∅	+
13	Jugular bulb abnormality	+	+
14	Jugular bulb abnormality	-	+
15	Aberrant internal carotid artery	+	+
16	Intracanalicular acoustic neuroma	∅	+

+ Indicates positive study finding, - Indicates negative study finding, ∅ Indicates study not performed

Table II Lesions associated with subjective pulsatile tinnitus

Diagnosis	Number of patients
Arteriovenous Malformation	3
Arteriovenous Fistula	3
Vascular loop	3
Dural sinus stenosis	3
Jugular bulb abnormality	2
Aberrant internal carotid artery	1
Intracanalicular acoustic neuroma	1

Table III Radiographic findings in patients with objective pulsatile tinnitus

Patient	Diagnosis	Cerebral Angiogram	MR
1	Arteriovenous Malformation	+	+
2	Arteriovenous Malformation	+	-
3	Arteriovenous Malformation	+	-
4	Arteriovenous Malformation	∅	+
5	Arteriovenous Malformation	∅	+
6	Atherosclerotic carotid artery disease	+	∅
7	Dural sinus stenosis	+	-

+ Indicates positive study finding, - Indicates negative study finding, ∅ Indicates study not performed

Table IV Lesions associated with objective pulsatile tinnitus

Diagnosis	Number of patients
Arteriovenous Malformation	5
Dural sinus stenosis	1
Atherosclerotic carotid artery disease	1

Table V Lesions associated with pulsatile tinnitus

Diagnosis	Number of patients
Arteriovenous malformation	8
Dural sinus stenosis	4
Arteriovenous fistula	3
Vascular loop	3
Jugular bulb abnormality	2
Aberrant internal carotid artery	1
Atherosclerotic carotid artery disease	1
Intracanalicular acoustic neuroma	1