

Magnetic Resonance Angiography in Pulsatile Tinnitus: The Role of Anatomical Variations

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Abstract: Pulsatile tinnitus (PT) is a perception of a rhythmical sound that is synchronous with the heartbeats. Despite being seen rarely in daily practice, frequently it is associated with identifiable causes, thus warranting special attention in regard to the etiological diagnosis. PT results from blood flow turbulence, which in turn results from changes in flow velocity or in the vessel lumen. One of the most important causes of PT is the paraganglioma, a vascular tumor that appears as a reddish retrotympenic mass. However, a normal tympanic membrane mandates differentiating among other diagnoses, such as arteriovenous malformations or fistulas, intracranial or extracranial aneurysms, a high or dehiscent jugular bulb, and persistent stapedial artery. Owing to the progress of radiological evaluation, magnetic resonance angiography (MRA) has proven to be excellent for evaluating vascular diseases. From January 1995 to June 1997, the authors prospectively studied 16 patients with PT and normal otoscopic examination. The study comprised 1 male and 15 female patients (ages 25–71 years; mean age, 42.5 years). All were subjected to MRA evaluation, which revealed the etiological diagnosis in 13 cases (81.25%), including 2 aneurysms and 1 case of intracranial hypertension. Of the 13 patients, 9 (69.23%) presented with at least one variation of vascular anatomy of the skull, showing a close correlation, in most cases, with the side on which PT occurred. Our results confirm that MRA is an excellent primary screening modality for patients with PT and normal otoscopic findings. The authors point out the importance of making etiological diagnoses in such cases, suggesting that variations of the vascular anatomy of the skull are a possible etiology.

Keywords: magnetic resonance, pulsatile tinnitus, vascular malformation

Pulsatile tinnitus (PT) is a rhythmical auditory sensation that is synchronous with a patient's heartbeat [1]. Despite being seen only rarely in daily practice, frequently it is associated with identifiable causes, thus warranting special attention as regards the etiological diagnosis. PT originates mainly in vascular structures (venous or arterial vessels) as a result of flow turbulence caused by increased blood volume or alterations in the vessel lumen [2–5]. When close to

the skull base, such alterations may produce auditory sensation [3, 5] sufficient to disturb an affected patient's life and sometimes may cause serious psychological disorders [2].

The best-known cause of PT is a paraganglioma. Often, however, this vascular tumor presents with a reddish retrotympenic mass [6] in the otoscopic examination, which strongly suggests the correct diagnosis [2, 7]. A greater challenge occurs when the tympanic membrane is normal. Then, other causes of PT must be considered, such as dural malformation, arteriovenous fistulas, intracranial or extracranial aneurysms, a high or dehiscent jugular bulb, and persistent stapedial artery [3, 5, 6, 8–10].

On 1882, Jacobson [4] was the first to describe a case of auditory sensation of cranial murmur associated

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with atherosclerosis. Since 1936, many authors have described cases of PT associated with several such etiologies as vascular abnormalities [10], venous hum [11], tumors of the cerebellopontine angle, a high jugular bulb, dehiscent internal carotid artery (ICA) [3,7], and described arteriovenous malformations [7]. We are not aware that the literature contains any report regarding the correlation between PT and anatomical variations of the arteries close to the skull base.

Since the establishment of the magnetic resonance angiography (MRA) technique, many vascular abnormalities have been identified, representing a great advance in many areas of medical science, including otoneurology. Considering the high incidence of identifiable and treatable causes of PT, the correct etiological diagnosis is fundamental. Therefore, the objective of our study was to evaluate the effectiveness of MRA in establishing the etiological diagnosis of PT.

CASUISTIC AND METHODS

Our prospective study was conducted at the tinnitus ambulatory unit of the Ear, Nose, and Throat Department of the University of São Paulo from January 1995 to June 1997. During this period, 16 patients (15 female and 1 male) with PT and normal otoscopy were evalu-

ated; their ages ranged from 25 to 71 years (average, 42.5 years).

To rule out systemic causes of PT, such as anemia and hyperthyroidism, a clinical investigation always was accomplished that emphasized the following examinations: complete blood cell count, total cholesterol level and fractions, glucose test (test of tolerance to oral glucose when necessary), levels of triglycerides and thyroid hormones, and serological reaction for syphilis.

Besides a complete audiometric evaluation, all patients underwent magnetic resonance imaging (MRI) with complementary MRA. The examinations always were performed by the same radiologist, using the team-of-flight, three-dimensional spin-echo technique, with T₁- and T₂-weighted sequences. Axial acquisitions and sagittal reconstructions were performed without the use of endovenous contrast.

RESULTS

The clinical investigation and the MRI examinations were normal in all patients. The results of MRA in each patient are summarized in Table 1. Thirteen of 16 examinations (81.25%) presented alterations of the vessel lumen or of the velocity of blood flow (or both), includ-

Table 1. Results of MRA in 16 Patients with PT and Normal Otoscopy

Patient's initials	Age (yr)	Gender	Duration of PT	Side of PT	MRA results
AMB	54	F	< 1 yr	R	Basilarization; aneurysm of right supraclinoid ICA in the exit of the posterior communicating artery
AMS	58	M	1-2 yr	L	Hypoplasia of left A1, basilarization, left vertebral artery dominant with projection to the left cerebellopontine angle
DAS	25	F	2-3 yr	L	Discreet dominance of the left VA
DSA	32	F	4 yr	R	Hypoplasia of right VA
GFO	53	F	1-2 yr	R-L	Multiple murals irregularities in the petrous carotids arteries; hypoplasia of left A1, basilarization
GCS	71	F	1-2 yr	R	Basilarization; blood flow is slow
LBK	29	F	3-5 yr	Head R-L	Hypoplasia of the left VA, right PICA dominant, close to VII and VIII nerves
IV	43	F	4 yr	L	Normal
MDS	37	F	2 mo	R	Mild sinuosity of the basilar artery
MFA	32	F	1 yr	R-L	Mild vascular blush in the projection of left ear
MGF	37	F	3-5 yr	R	Aneurysm of right ICA at exit of ophthalmic artery
MJS	35	F	5-10 yr	R	Intracranial hypertension, hydrocephalus, syringohydromyelia, cerebellar tonsil below the magno forame (Chiari I)
MVR	48	F	2-3 yr	L	Mild basilarization, left PICA dominant, thick right AICA dominant
SOC	32	F	3-5 yr	L	Normal
TSS	43	F	3 yr	Head	Normal
ZPM	51	F	3 yr	R-L	Left posterior cerebral artery origins from ICA (fetal pattern), hypoplasia of left VA

AICA = anteroinferior cerebellar artery; ICA = internal carotid artery; L = left; MRA = magnetic resonance angiography; PICA = posteroinferior cerebellar artery; PT = pulsatile tinnitus; R = right; VA = vertebral artery.

Note: Note the correlation of abnormality with the side on which tinnitus occurred.

ing intracranial aneurysms in 2 patients and intracranial hypertension due to Arnold-Chiari syndrome in 1 patient. Only three patients presented normal examinations (18.75%), which prevented us from making an etiological diagnosis.

Of the 13 patients with altered MRA, 9 (69.23%) presented with at least one variation of the vascular anatomy of the skull, which, in most cases, was closely correlated with the side on which PT occurred. The vertebral artery was the most often involved, with dominance or hypoplasia of one side in two each of the nine cases (22.2%) and "basilarization" in five of the nine cases (55.5%). The carotid artery was involved in three cases (33.3%), the posteroinferior cerebellar arteries (PICAs) in two (22.2%), and the anteroinferior cerebellar arteries (AICAs) in only one case (11.1%).

DISCUSSION

Generally, PT is the otologic manifestation of a blood flow abnormality of the temporal bone. Synchronous with cardiac pulsation [1, 7, 12], PT can reach high intensity and become a reason for psychological disability in some patients [3, 13]. This type of tinnitus is a common reason for imaging examination of this region [2, 6].

PT can be characterized as objective or subjective. *Objective tinnitus* can be noticed by both patients and medical observers. Usually, it is caused by vascular lesions that can be detected during MRI or MRA examinations (e.g., arteriovenous fistulas of the dura or extracranial fistulas, aberrant internal cerebral artery, and abnormalities of the cervical veins) [1, 4, 6, 10, 12, 13]. *Subjective PT* is observed only by affected patients, being demonstrable during imaging examinations in only 42% of cases [6]. Some examples are paragangliomas, a high jugular bulb, a jugular bulb diverticulum, a pseudoaneurysm, arteriovenous malformations, and stenosis of the transversal sinus [1]. In our study, just one patient presented with objective PT, the diagnosis of which was compatible with Arnold-Chiari syndrome (herniation of cerebellar tonsils); the patient showed signs of intracranial hypertension, hydrocephalus, and syringohydromyelia.

The importance of investigating PT is justified by its higher association with identifiable and treatable causes, as compared to the common types of subjective and nonpulsatile tinnitus. In addition, misdiagnosis can be catastrophic, as PT can be related to intracranial diseases, including aneurysms and tumors [12, 13].

The most common causes of PT described in the literature are dural arteriovenous malformations and fistulas [3, 5, 6, 8–10]. These are followed in frequency by paraganglioma, a vascular tumor that may corre-

spond to the tympanicum, jugulare, or jugulotympanic glomus [2, 7]. A paraganglioma presents with a vascular retrotympanic mass, which is an important sign for the physician, strongly suggesting the diagnosis [6].

This study considered only patients in whom otoscopy results were normal. According to our results, MRA was able to identify the cause of the PT in 81.25% of affected patients (13 of 16). Interestingly, we noted that the most common established cause of PT was anatomical variation of the vascular anatomy of the skull, which has not heretofore been described in the researched literature.

According to Brodal [14], "In Neurology, a knowledge of structural features is perhaps more important for an understanding of function under normal and pathological conditions than in any other branch of medicine. The determination of the site of the lesion is therefore an important link in understanding the nature of the disease." The need to understand variations of the vascular anatomy and the need for applying this understanding has increased. The variability of the vascular anatomy, although "normal," represents increased rigidity of the system. It may remain compatible with function for a long time, but a minimal constraint may betray its limited flexibility. Thereafter, the variation becomes "abnormal" and symptomatic [15].

VASCULAR ANATOMY CONSIDERATIONS: A SUMMARY

Within the posterior circulation of the skull, anatomy takes center stage. More than in any other region, accurate diagnosis depends on an intimate familiarity with the brain and vascular anatomy [16]. The vascular supply of posterior circulation is from the paired subclavian-vertebral arteries, which join intracranially to form the basilar artery, which in turn bifurcates into the paired posterior cerebral arteries (PCAs).

Extracranial Large Arteries

The subclavian arteries give rise to their first branches, the vertebral arteries (VAs), which commonly present variations [17]. The VAs, more often the left, may arise directly from the aortic arch (approximately 8%), whereas the right VA may originate directly from the innominate artery. In very rare cases, either VA can arise from the common carotid artery on the same side. Often, the VAs are asymmetrical: In perhaps 45% of individuals, the left VA is larger, whereas in 21% the right VA is dominant; in 34%, both arteries are approximately of equal size [18]. One VA may be atretic or very hypoplastic.

Intracranial Large Arteries

Often, the intracranial portion of the vertebral arteries (ICVAs) are asymmetrical; one may be two to three times the size of the contralateral VA, with the left ICVA more often the larger. The four most important branches of the ICVAs are the anterior and posterior spinal arteries, the PICAs, and the direct lateral medullary branches. The PICAs are the largest and yet the most variable of the ICVA branches [19]. The most distal segment of the ICVAs forms at a different time embryologically, and often this segment is hypoplastic or even absent. In that case, angiograms show that the ICVA seems to end in the PICAs and that the contralateral ICVA usually is larger and responsible for the major basilar artery supply. Often, this situation is called *basilarization* of one ICVA. Usually, when the left ICVA is dominant, the basilar artery deviates to the right, whereas it deviates to the left when the right ICVA is dominant. In only 25% of patients does it have a perfectly straight course [20].

The main branches of the basilar artery are the AICAs. The sizes of the PICAs and AICAs on each side often are reciprocal. Either can be very hypoplastic or absent, in which case the remaining, healthier PICA or AICA supplies the entire territory on that side. The PCAs originate from the terminal bifurcation of the basilar artery. In approximately 10% of individuals, a fetal pattern persists, in which the PCA essentially originates from the ICA and the proximal segment of the PCA from the basilar artery is hypoplastic [21]. One PCA can be unusually large (29%) or small (24%).

Evaluating the MRA of the 13 patients with any alteration, we noticed that 9 (69.23%) presented with variations of the arteriovascular anatomy of the skull only. No previous report in the literature addresses the correlation between such anatomical variation and PT. Once this finding is very relevant, we can hypothesize that the turbulence occurring during the passage of blood flow through such altered vessels can promote the appearance of tinnitus. For example, in one patient (DAS; see Table 1) with PT in the right ear and hypoplasia of the right VA, the disorder might be explained, in the absence of other causes, by the turbulent flow in such a narrow vessel. The same can be said in relation to another patient (LBK; see Table 1), who had PT and MRA showing dominance of the right PICA and hypoplasia of the left VA.

Though the anatomical variation is congenital and PT seems to appear only in adulthood, we still need to determine the predisposing factors that trigger the appearance of tinnitus. We hope that future studies will resolve this issue. As we said, the variation may remain compatible with function for a long time, but a minimal

constraint may betray its limited flexibility. Thereafter, the variation becomes abnormal and symptomatic [15].

CLINICAL AND RADIOLOGICAL INVESTIGATION AND CONSIDERATIONS

Clinical diagnosis of PT is based on a complete neurological evaluation, which includes clinical history, complete physical examination (with special attention to the otoscopic examination), auscultation of the external auditory canal and adjacent areas, and palpation of the high cervical area and preauricular region [1, 5]. Equally important is evaluating an eventual papilledema, which leads to the diagnosis of benign intracranial hypertension syndrome [5]. Reports in the literature described the use of a modified electronic stethoscope, the auscultear, to assist in evaluating PT (mainly objective tinnitus) [22].

Radiological investigation is most important for establishing the etiological diagnosis. Initially, definition of the radiological approach depends on knowing the nature of the PT (objective or subjective) and the aspect of the tympanic membrane. Such data can suggest the possible etiological diagnosis, guiding the use of the most appropriate radiological modalities [12].

Computed tomography (CT) allows diagnosis of bone alterations associated with vascular anomalies [1, 6]. High-resolution CT is the appropriate examination to be requested in patients with retrotympanic masses, allowing one to diagnose such conditions as paragangliomas, aberrant ICA, and abnormalities of the jugular bulb. However, it cannot detect arteriovenous malformations or arteriovenous fistulas of the dura, which are the most important causes of PT, especially in the presence of normal otoscopic findings [1].

Arteriography is more sensitive than is CT, allowing the diagnosis of arteriovenous fistulas and intrinsic vascular anomalies. On the other hand, it is an invasive examination, with a considerable related morbidity index in some series [6]. Thus, it should not be used as a screening examination, being indicated only in such limited cases as preoperative evaluation or embolization (or both), evaluation of collateral circulation with possible vessel occlusions, and therapeutic embolizations [3, 5, 7, 8, 12, 13]. Until recently, radiological investigation of PT was based on performance of a CT scan associated with arteriography [5, 6]. Currently, arteriography has been restricted to those cases in which MRA is normal, because it may detect small abnormalities not shown in MRA.

Due to its poor resolution for vascular and bone structures, the benefit of MRI used alone is limited. Without contrast enhancement, it is not sufficient to differentiate some vascular tumors from arteriovenous

malformations or to identify regions of vascular compressions, thereby limiting its use in patients with PT. When gadolinium contrast enhancement is used, MRI can establish the diagnosis of paraganglioma [6]. In our study, all MRI examination results were normal, establishing that it is not a proper examination for evaluating PT, a finding that is consistent with other reports in the literature.

MRA offers additional information and is superior to MRI alone; hence, arteriography is preferable in almost all cases. It increases the ability to diagnose lesions responsible for PT, mainly dural arteriovenous malformations and fistulas [1, 5, 6]. It also is extremely useful in evaluating vascular tumors, with special attention to paragangliomas. Additionally, it allows the differentiation of vascular neoplasms from those with fewer vascular components, facilitating the differential diagnosis between meningioma and schwannoma. In 1993, Dietz [6] compared MRI and MRA in the evaluation of 49 patients with PT, noting vascular lesions in 28 patients that were either demonstrated better (46%) or only visualized (36%) with MRA. Thus, its superiority is obvious in precise neurovascular evaluations, demonstrating small vascular alterations, and occasionally in vascular compressions [23].

CONCLUSION

Most PT is related to identifiable causes. In their present study, the authors point to variations of the vascular anatomy of the skull as a possible cause of PT. In this study, MRA was able to detect those causes in 81.25% of patients with PT and normal otoscopy. This evaluative technique has had an enormous impact in evaluating patients with PT and normal otoscopic examination, allowing precise etiological diagnosis and adequate treatment of affected patients. It allows screening of patients with PT through only one radiological examination [6]. Presently, this practical, safe, and effective screening method should be used as first choice for these patients, be the tinnitus objective or subjective and accompanied or unaccompanied by retrotympanic mass, cervical lesions, or cranial nerve deficits [11].

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