PULSATILE TINNITUS: AUDIOLOGIC AND MR IMAGING DIAGNOSTIC ISSUES

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ABSTRACT

Pulsatile tinnitus often presents a diagnostic and management dilemma to the neurotologists. Correct diagnosis is imperative because in the majority of cases, there is a treatable underlying etiology. In addition, failure to make proper diagnosis may be disastrous because in some patients, a life-threatening intracranial disease may be present. This study was carried out aiming to investigate the feasibility of differentiating the possible causes of pulsatile tinnitus, particularly in patients with normal otoscopy, using clinical, audiological and radiological tools. 29 patients complaining of pulsatile tinnitus were evaluated by history taking, clinical examination, laboratory investigations, otoscopy, basic audiological evaluation, MRI for

petrous bone and brain and MRA. Glomus jugulare tumor was diagnosed in 3 patients. Benign intracranial hypertension (BIH) was diagnosed in 4 patients. Intraventricular neoplasm in 2 patients. Internal carotid artery stenosis in one patient. Post-traumatic pseudoaneurysm in one patient. Scalp AVM in one patient. Temporal bone metastasis in one patient. Severe anemia in one patient and no identifiable cause (idiopathic) in 15 patients. A unilateral mild low frequency pseudosensorineural hearing loss was identified in 10 patients with normal otoscopy. Hearing loss was normalized after elimination of tinnitus by applying a light digital pressure over the ipsilateral internal jugular vein.

INTRODUCTION

Tinnitus is a very common otolog-

ic symptom that may affect up to 30% of the population worldwide (Heller, 2003). Despite the recent great advances in otology, otorhinolaryngologists are very often in the difficult situation of dealing with a symptom (tinnitus) for which they may not know the exact pathophysiology or treatment. Although the methods of treatment were different, it is clear that all societies have recognized the high prevalence of this annoying symptom and tried to understand it, clarify it and treat it.

First of all, a distinction should be made between subjective and objective tinnitus. Another important distinction is that between pulsatile and non-pulsatile tinnitus. Pulsatile tinnitus can be classified further as either vascular or non-vascular in origin. Non vascular causes are usually related to myoclonus of the palatal musculature, stapedius muscle or tensor tympani. Vascular causes can be differentiated further into venous or arterial (Xenellis et al., 2005).

Pulsatile tinnitus, although an infrequent otologic symptom, often presents a diagnostic and manage-

ment dilemma to the neurotologists especially in patients with normal otoscopy. Correct diagnosis is imperative because in the majority of cases, there is a treatable underlying etiology. In addition, failure to make proper diagnosis may be disastrous because in some patients, a life-threatening intracranial disease may be present (Sismanis, 1998). Accordingly, the present study was designed to investigate the feasibility of differentiating the possible causes of pulsatile tinnitus, particularly in patients with normal otoscopy, using clinical, audiological and radiological tools.

PATIENTS AND METHODS

This study was carried out on 29 patients complaining of pulsatile tinnitus. Tinnitus was defined pulsatile when the patient described sounds synchronous with the heart rate. The patients were 21 females and 8 males. Their age ranged from 21 to 63 years with a mean age of 40 years. Patients were referred from otolaryngologists to the audiology unit of Mansoura university hospital during the period from January, 2003 to February, 2005. All patients were subjected to :

- 1-Full history taking.
- 2-Clinical examination including:
 - a. General examination for obesity, anaemia, hypertension and hyperthyroidism.
 - b. Local head and neck examination for audible bruit, palpable masses or pulsatile swelling.
 - c. Neurological examination for the presence of cranial nerve palsy or signs of increased intracranial pressure
- 3-Otoscopy.
- 4-Pure-tone and speech audiometry in a sound booth using a Madsen pure-tone and speech audiometer model OB822.
- 5-Immittancemetry using an immittancemeter, Interacoustics model AZ7.
- 6-Laboratory investigations for anaemia and hyperthyroidism.
- 7-MR imaging of the temporal bone and brain: MR imaging examinations were carried out using a 1.5 Tesla system unit. Evaluation of the temporal bone region was done using thin sections axial and coronal non contrast T1 and T2 weighted spin echo sequences. Axial T1 weighted images with repetition time (TR) of 500 msec, echo time (TE) of 20 msec, slice thickness =

3 mm and axial T2 weighted images with TR of 5000 msec, TE of 90 msec and slice thickness = 3 mm. Evaluation of the whole brain was done using thicker slice thickness (5-7 mm) axial, coronal and sagittal planes T1 and T2 weighted spin echo sequences. Post contrast T1 weighted images were obtained in 8 patients after intravenous injection of 10 cc of gadolinium. Magnetic resonance angiography (MRA) was done using non contrast 3D time of flight sequence (3D TOF) for all cases.

RESULTS

Pulsatile tinnitus was unilateral in 27 patients (right sided in 15 and left sided in 12 patients) and bilateral in 2 patients.

Otoscopic examination did not reveal anything remarkable except in 3 female patients aged 45 to 52 years, where there is a reddish retrotympanic mass. Their pure-tone audiometry revealed a unilateral mild conductive hearing loss. Immittancemetry showed a humped curve with cyclic perturbations consistent with the patient pulse. Glomus jugulare tumor

was highly suspected and diagnosis was confirmed by MR imaging (figures 1 & 2).

In the remaining 26 patients with normal otoscopy, a unilateral mild low frequency sensorineural hearing loss with excellent speech discrimination was identified in 10 patients. Hearing loss was normalized in all patients after elimination of pulsatile tinnitus by applying a light digital pressure over the ipsilateral internal jugular vein (figure 3). Immittanceometry was normal in all the 26 patients.

MR imaging in patients with normal otoscopy revealed attenuated ventricles and empty sella in 4 patients and considered suggestive of benign intracranial hypertension (BIH) which was proved by papilloedema in fundus examination and lumbar puncture. Intraventricular space occupying lesion was identified in 2 patients, provisionally diagnosed as benign tu-

mors and proved by surgery and histopathology in one patient as ependymoma (figure 4). Internal carotid artery stenosis was identified in one patient aged 63 years (figure 5). A post-traumatic pseudo aneurysm involving the left external carotid artery was identified in one patient (figure 6). Scalp arteriovenous malformation (AVM) was identified in one patient with a history of trauma and complaining of a tender retroauricular swelling (figure 7). Temporal bone metastasis was identified in one patient (figure 8).

Diagnosis of severe anemia as a cause of pulsatile tinnitus was established clinically and laboratory in one patient aged 50 years.

In 15 patients (12 females and 3 males), no cause could be found and diagnosed as idiopathic. Table (1) summarizes the causes of pulsatile tinnitus in the present study.

Table (1): Causes of pulsatile tinnitus in the study group

Diagnosis	No. of cases
Benign intracranial hypertension (BIH)	4
Glomus Jugular tumour	3
Intraventricular neoplasm	2
Internal carotid artery stenosis	1
Post-traumatic pseudoaneurysm	1
Scalp AVM	1
Temporal bone metastasis	1
Severe anemia	1
Idiopathic	15
Total	29

Figure (1): Left glomus jugular tumor. Axial T2 WI showing slight hyperintense lesion in the left jugular foramen region with multiple signal void areas.

Figure (2): Large right glomus jugular tumor. Axial post contrast T1 WI with fat suppression showing the marked contrast enhancement of the mass.

Figure (3): PTA before and after applied digital pressure over the ipsilateral internal jugular vein in a patient with pulsatile tinnitus and normal otoscopy.

Figure (4): Intraventricular ependymoma. Axial T1 W1. The tumor in the left lateral ventricle is isointense to the white matter and shows small signal void areas. There is a subsequent mild dilatation of the left lateral ventricle.

Figure (5): Right ICA stenotic segment. MRA showing the stenotic segment within the left internal carotid artery (arrows).

Figure (6): left external carotid pseudo aneurysm. MRA showing large aneurysm on the left side related to branches of the external carotid artery.

Figure (7): Left temporal scalp AVM. MRA showing the scalp AVM with intracranial extension

Figure (8): Left skull base and temporal bone metastasis. Axial post contrast MRI of temporal bone shows enhancing soft tissue mass involving the clivus and skull base on the left side.

DISCUSSION

Pulsatile tinnitus is an interesting clinical symptom, frequently associated with identifiable and treatable causes when compared to the more common non-pulsatile tinnitus. Therefore, misdiagnosis may have serious consequences as it may be related to intracranial disease such as tumors and aneurysms that may have fatal complications (Carmody, 2000). Pulsatile tinnitus usually originates from vascular structures (venous or arterial vessels) as a result of either increased blood flow or lumen stenosis. The responsible vascular structures are usually within the cranial cavity, the head and neck region and the thoracic cavity (Sismanis, 2003).

In our study, abnormal otoscopy (reddish retrotympanic mass) was found in only three female patients. Their audiologic evaluation revealed unilateral conductive hearing loss and humped tympanogram with cyclic perturbations consistent with the patient pulse. MRI showed marked contrast enhancement and alternating punctuate areas of hyperintensity and hypointensity. A classic appearance for glomus jugular tumor that was

called salt and pepper appearance. The "pepper" component represents the multiple areas of signal void interspersed with the "salt" component seen as hyperintense foci due to slow flow or hemorrhage.

Patients with normal otoscopy (26 patients) :

In 4 patients, history taking and clinical examination raised the suspicion of BIH; all were young females and overweight. They all complained of headache and pulsatile tinnitus which was unilateral in three patients and bilateral in one patient. One patient complained also of blurred vision. The tinnitus was of the venous type in all of them. Their pure-tone audiometry revealed a unilateral low frequency pseudosensorineural hearing loss which was normalized and tinnitus disappeared by applying a light digital pressure over the ipsilateral internal jugular vein. We referred the patients suspected to have BIH to ophthalmology department for fundus examination and papilla edema grade II was present in all patients. The diagnosis of BIH was proved by lumbar puncture. The importance of MR imaging in such patients is to exclude a

cause for intracranial hypertension. The findings on MRI are often too subtle and non-specific to allow the diagnosis of BIH on the basis of MRI scans alone (Binder et al., 2004). Attenuated small sized ventricles and empty sella were the only MR abnormality. In our study, BIH was the second most common cause of pulsatile tinnitus with normal otoscopy. However, in a study carried out on 145 patients by Sismanis (1998), BIH was the most common cause of pulsatile tinnitus with normal otoscopy.

Two other patients had a history of persistent pulsatile tinnitus for about two years, both had normal otoscopy and normal hearing. MRI revealed an intraventricular space occupying lesion. Surgical excision was done in one patient and the pathologic diagnosis was ependymoma. The mechanism of pulsatile tinnitus was unclear, but these patients had mild dilatation of the ventricular system which is reported in literature as a potential cause of pulsatile tinnitus (Lo and Maya, 2003).

A stenotic segment of internal ca-

rotid artery (ICA) on the side of pulsatile tinnitus was diagnosed by MRA in one patient. MRA showed the site and extent of the stenotic segment which also was clearly visible on reviewing the axial source images. Westwood et al. (2002) reported that MRA was found to be highly sensitive and specific in diagnosing carotid artery stenosis.

A scalp AVM may cause pulsatile tinnitus. Controversy still exists about the etiology of scalp AVMs, but it is accepted that they may be either of congenital or traumatic origin, with the congenital lesion being the more commonly encountered variety (Fisher-Jeffes et al., 1995). One of our patients complained of pulsatile tinnitus associated with a retro auricular swelling. On examination, it was compressible and pulsatile. Although the condition was diagnosed clinically as a scalp AVM, imaging was still indicated for confirmation and to delineate the extent and connections of the AVM. MRA study revealed a scalp AVM with an intracranial connection to branches of the middle ce-

rebral artery.

A history of a swelling in the parotid region following a penetrating trauma was given by one patient. By examination, this swelling was cystic and pulsatile suggesting a vascular swelling. MRA revealed a pseudoaneurysm in a branch of the external carotid artery. Transmitted pulsation was the cause of pulsatile tinnitus.

From the literature, other systemic causes of pulsatile tinnitus include conditions associated with hyperdynamic circulation such as chronic anemia, pregnancy and thyrotoxicosis (Noell and Meyerhoff, 2003). History, clinical examination and appropriate laboratory investigations are the key to diagnosis. The tinnitus is usually heard on the side of dominant jugular vein (the right side in 70% of normal subjects). Characteristically, tinnitus is abolished by light digital pressure over the ipsilateral internal jugular vein.

After exhausting all the investigations, there remained 15 patients with no identifiable cause of pulsatile tinnitus and with no associated radiologic abnormalities. Their mean age was

35 years, most of them were females. Tinnitus was unilateral in most of them. There was no associated ear symptoms and no retrotympanic masses on otoscopy. In the literature, this group was termed essential tinnitus, venous hum tinnitus or more recently idiopathic tinnitus (Lo and Maya, 2003). The percentage of cases with no identifiable abnormalities explaining pulsatile tinnitus is variable in the reported series, but is always present. Shin et al. (2000) found no radiologic abnormalities to explain pulsatile tinnitus in nine out of thirty three patients evaluated by MRI and MRA. Dietz et al. (1994) found no MRI or MRA abnormality in 21 patients out of 49 patients evaluated for pulsatile tinnitus.

Conclusion :

If otoscopy and audiological profile suggest a glomus jugular tumor, MRI is recommended. If pure-tone audiometry revealed a low frequency sensorineural hearing loss in a patient with pulsatile tinnitus, Pure-tone audiometry should be repeated with digital pressure over the ipsilateral internal jugular vein to alleviate tinnitus as pure-tone thresholds could be nor-

malized. Non contrast MRI of the brain and temporal bones is the initial investigation together with MRA of the head and the neck vessels. If no abnormality is detected by these procedures, the probability of a significant disease becomes minimal.

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طنين الأذن النابض : التشخيص بالاختبارات السمعية والرنين المغناطيسى

ملخص البحث :

يجد الكثير من أطباء الأذن بعض الصعوبة فى تشخيص وعلاج طنين الأذن النابض. ويتحتم التشخيص الصحيح لسبب هذا العرض لأنه فى حالات كثيرة يكون من الممكن علاجه. أما عدم الوصول إلى تشخيص صحيح لسبب هذا الطنين ممكن أن يؤدي إلى كارثة نظراً لاحتمالية وجود مرض داخل الدماغ يهدد حياة المريض. تهدف هذه الدراسة إلى إمكانية إستخدام الاختبارات السمعية والتصوير بالرنين المغناطيسى لتشخيص الأسباب المختلفة لطنين الأذن النابض وبالأذات فى المرضى ذى فحص منظاري طبيعى للأذن.

أجريت الدراسة على ٢٩ مريض يشكون من طنين الأذن النابض. وقد تم تقييم هؤلاء المرضى بأخذ التاريخ المرضى، الفحصى الأكلينيكي، فحوص معملية، اختبارات السمع الأساسية بالإضافة إلى التصوير بالرنين المغناطيسى للمخ.

تم الوصول إلى سبب طنين الأذن النابض فى ١٤ مريض وكانت الأسباب كالآتى :

ورم بكتلة الأوعية الدموية الكهفية فى ٣ مرضى، إرتفاع حميد للضغط داخل الدماغ فى ٤ مرضى، ورم داخل البطين الخى فى ٢ مريض، ضيق بالشريان السباتى الداخلى فى مريض واحد، ورم وعائى كاذب فى مريض واحد، تشوه شريانى وريدى بفروة الرأس فى مريض واحد، انبثاث سرطانى بالعظمة الصدغية فى مريض واحد، فقر دم حاد فى مريض واحد. ولم يتم الوصول لأى سبب فى ١٥ مريض.

ووجد أن هناك ١٠ مرضى ممن يعانون من طنين الأذن النابض وفحص منظاري طبيعى للأذن، كان القياس السمعى بالنغمات النقية يشير إلى وجود ضعف سمع حسي/ عصبى بدرجة بسيطة بالترددات الرخيمة إلا أنه بمجرد الضغط على الوريد الوداجى الداخلى لنفس جهه الطنين، اختفى الطنين وأصبح قياس السمع طبيعياً.

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