Objective pulsatile tinnitus: case report

Acufene oggettivo pulsatile: caso clinico

P. VERDE, E. MARCIANO, R. DE FALCO, R. TESTA, S. BUONAMASSA[1], G. MARINIELLO[1]
Audiology Unit, Department of Neuroscience and Behavioural Sciences
[1] Department of Neurologic Sciences, Neurosurgery Clinic, University of Naples "Federico II", Naples, Italy

Key Words

Tinnitus • Pulsatile tinnitus • Diagnosis

Parole Chiave

Acufeni • Acufene pulsatile • Diagnosi

Summary

Anomalies in the vascular structures of the neck, cranial base, temporal bone, and intracranial circulation may give rise to pulsatile tinnitus. If the anomalous sound is perceived also by others, then it is defined as objective tinnitus. Herein, the case is reported of right pulsatile tinnitus of one year's duration, which appeared after cranial trauma. Magnetic resonance angiography showed that the jugular bulb was dominant on the same side as the tinnitus. The tinnitus was recorded with a high-sensitivity microphone, while otoacoustic emissions were measured by distortion products during follow-up.

Riassunto

Anomalie di decorso delle strutture vascolari del collo, del basicranio, dell'osso temporale e del circolo intracranico possono dare origine ad un acufene pulsatile. Nei casi in cui tale suono può essere percepito non solo dal soggetto affetto, ma anche dagli altri, l'acufene viene definito oggettivo. Riportiamo il caso di una paziente affetta da acufene pulsatile destro esordito circa un anno dopo un trauma cranico. Allo studio angiografico mediante risonanza magnetica il bulbo giugulare appariva dominante dallo stesso lato del tinnito. Quest'ultimo veniva registrato con un microfono ad alta sensibilità, mentre durante il follow-up si misuravano le emissioni otoacustiche mediante i prodotti di distorsione.

Case Report

M.T.S., a 28-year-old female, came to our observation complaining of right objective tinnitus for about 12 months, which was synchronized with her pulse. The patient had a college degree in classical languages and defined the noise with the Latin term "*susurrus*". Clinical examination was negative for both otologic and systemic disorders, but revealed a closed head concussion in the right temporal region, in addition to fracture of the nasal septum. The trauma had occurred about 2 years earlier. Otologic examination was negative and revealed neither rhythmic contractions of the soft tissues nor palpable murmurs of the preauricular region. Ocular examination did not reveal any signs of increased intracranial pressure. Measurements of the auditory canal were carried out in a sound-proof room; the tinnitus was clearly audible upon listening carefully on the right side of the patient. Rotation of the patient's head towards the right or the application of modest pressure on the sternocleidomastoid muscle led to a decrease in the tinnitus. Tonal audiometry revealed neurosensorial hearing loss at low frequency (125-500 Hz) of about 50 dB HL on the left side. A slight improvement (10 B HL) was obtained by repeating the audiometric test when light pressure was applied to the homolateral sternocleidomastoid muscle (Fig. 1).

Upon measurement, the tinnitus showed a pitch between 125-250 Hz, with an intensity of 50 dB HL. Vocal audiometry, auditory brainstem responses (ABR), impedance audiometry, vestibular tests (pursuit tracking, thermal stimulation), and Doppler examination of the supra-aortic trunk were within normal limits. Cerebral nuclear magnetic resonance (NMR) studies, with and without contrast medium, and high-resolution computed tomography (CT) examination of the temporal bone did not reveal any pathological alterations. High-resolution MR angiography revealed asymmetry of the venal drainage system of the posterior cranial cavity, with dominance of the transverse sinus, sigmoid, inferior petrous, and right jugular bulb (Fig. 2). During follow-up, otoacoustic emissions (OAEs) were evaluated as well as stimulus frequency (SOAEs) and distortion products (DPOAEs). The patient refused to undergo more invasive diagnostic procedures (cerebral angiography). At present, the intensity of tinnitus has decreased; the patient is in psychotherapy and ascertains that she is able to live with her "*susurrus*".

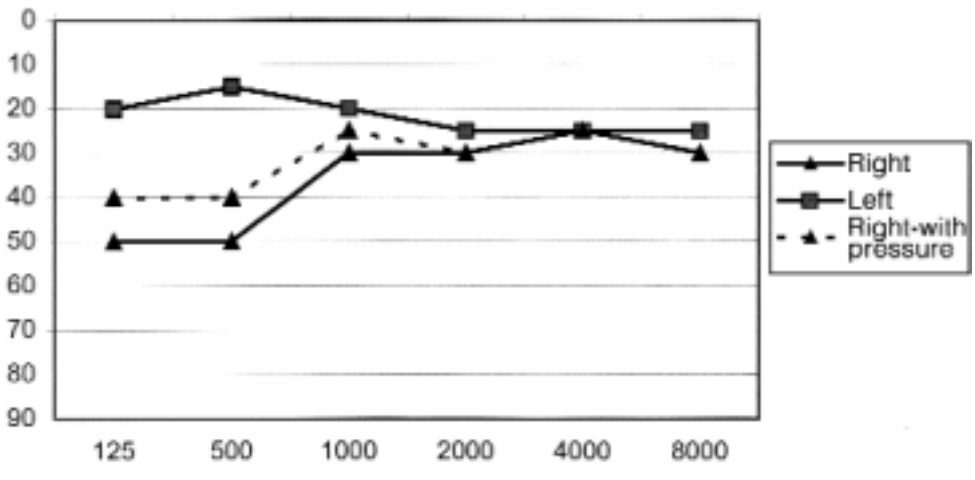

Fig. 1. Left and right tonal audiogram. Slight pressure to right sternocleidomastoid muscle decreases hearing loss by about 10 dB in frequency range from 125-1000 Hz (dx-dg-pres).

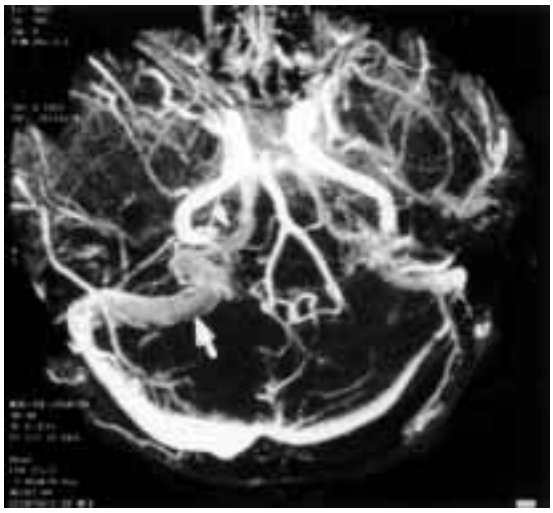

Fig. 2. Axial cerebral angiographic magnetic resonance. After injection of contrast medium, ectasia of right sigmoid sinus is evident (arrow) confluent in a large jugular bulb (arrowhead).

Materials and Methods

To understand the nature of the tinnitus, recordings were made with the intention to characterize the tinnitus by spectral analysis (Fig. 3). For this purpose, a high-sensitivity microphone (Larson and Davis model 2541), a pre-amplifier adaptor (Larson and Davis model PRM902), and a spectral analysis instrument with 1/3 octave analysis (Larson and Davis model 824) were used. All the listeners identified the tinnitus as a venous murmur. Frequency analysis was carried out on the signal that was recorded in digital format (WAW) and filtered to eliminate environmental noise. The sound was then elaborated with a sonogram using Cool-Edit software (Fig. 3). Within a 30-second recording time, 14 events (between 650 msec and 890 msec) were identified as tinnitus. Two of these events are shown in Figure 3. The first, lasting 690 msec, was followed by a pause and then by a second tinnitus lasting 886 msec. Comparison between the spectra in the presence and absence of tinnitus showed that in the former case, much more sound energy was present (particularly between 30 and 700 Hz). Both the SOAEs and OAEs were measured using a Madsen-Capella otoacoustic emission analyzer. All the measurements were carried out in a soundproof room with the patient in a relaxed supine, position. The DPOAEs were obtained using a stimulation level of 70 dB for F1 and F2 in the 500-800 Hz range. The SOAEs were recorded twice bilaterally, in a frequency range from 0-6 kHz. In both instances, the otoacoustic emissions observed, on the right, were 1.2, 2.8, 3.1, and 5.8 kHz, and 1.0, 1.3, 2.4 and 4.2 kHz on the left.

Discussion

Anomalies in the arterial-vascular structures of the intracranial region and/or the cranial base may give rise to pulsatile tinnitus [1,2]. In the case of venous aetiology, the tinnitus decreases when slight pressure is applied to the entire ipsilateral jugular vein [3]. Rota-

Fig. 3. Graph showing temporal changes in the auditory signal: 2 events were identified as tinnitus, while central portion was background noise.

tion of the head towards the affected side likewise causes compression of the entire jugular vein on transverse process of the atlas and the sternocleidomastoid muscle [4]. In patients with arterial aetiologies, these manoeuvres have no effect on the tinnitus [5]. In the present case, the venous origin of the sound was confirmed by the improvement of hearing in the low frequency range when slight pressure was applied to the homolateral sternocleidomastoid muscle; this pressure also eliminated the masking effect of the tinnitus [3] (Fig. 1).

In a series of 7 patients with pulsatile tinnitus, Rodgers et al. [1] reported that 4 cases presented asymmetry of the venous drainage system in the posterior cranial base, diagnosed by homolateral MR angiography. Our case also showed ectasia, most likely congenital in nature, of the transverse, sigmoid, and lower right petrosal sinuses (Fig. 2). These anomalies suggest an increased turbulence in the flow of the sigmoid sinus and the right jugular bulb. As hypothesized by Cutforth et al. [6], the vibrations produced by the turbulent flow in the jugular vein on the venous walls would be transmitted to the external auditory canal, generating tinnitus. Other Authors [7 8] have established that turbulence in blood flow may be caused by the presence of fibrosis in the sigmoid sinus or jugular bulb. In our patient, it is tempting to hypothesize that, following trauma, a rearrangement of the osteo-trabecular structure of the mastoid cells caused thickening of the bony walls in contact with the venous sinuses, increasing the transmission of turbulence produced by venous flow, causing tinnitus. However, this hypothesis cannot be verified, as no pre-trauma CT is available. Pulsatile tinnitus, due to a post-trauma vascular lesion, is relatively rare [9]. Low-frequency sensorineural hearing loss in a patient with a history of earlier trauma, in whom the tinnitus appeared after a considerable period of time favours a post-traumatic origin of the tinnitus [10]. During the follow-up period, oto-acoustic emissions were also recorded in order to better define the origin of the tinnitus and verify the integrity of the cochlea. Various studies have been reported on the use of oto-acoustic emissions in the diagnosis of tinnitus [11-13]. However, to our knowledge, no reports have appeared in the literature concerning pulsatile tinnitus

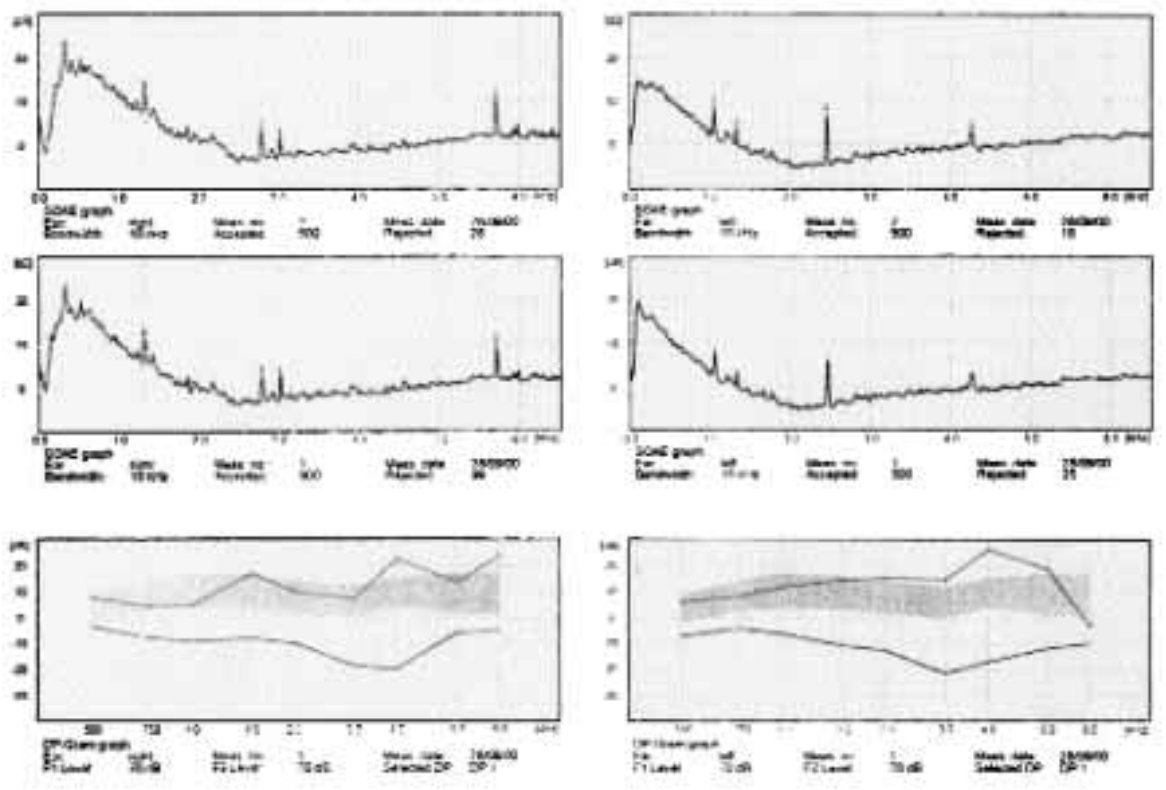

Fig. 4. Recording of OAE. SOAEs (top 4 graphs), DPOAEs (lower 2 graphs). Integrity of external acoustic cells on both sides is confirmed by absence of deficits in focal frequency in DPOAEs and by presence of SOAEs. The latter appear to be increased, at low frequency, in left ear, as present sound energy was greater.

associated with the presence of oto-acoustic emissions.

In our case, the otoacoustic emissions were present bilaterally, confirming normal function of the external acoustic cells. The DPOAEs did not reveal any deficits in the focal frequency, which would have indicated damage to the same cells (Fig. 4). The SOAEs, on the other hand, appeared to be increased in the left ear, in the low frequency range, as if the sound energy were greater.

Conclusions

Asymmetry of the venous drainage system in the posterior cranial base, with the dominant side corresponding to the symptomatic side, may be the cause of objective tinnitus. Correct diagnosis must be supported by detailed case history and by thorough, objective clinical examination, which should be associated with other procedures. Conventional cerebral angiography is the diagnostic exam of choice, allowing a correct therapeutic management. In those patients who refuse to undergo invasive examinations, a correct radioneurological evaluation must include high-resolution MR angiography and CT of the petrous bone. Eventual post-traumatic microanomalies involving bone, may be the cause of objective tinnitus due to the strict continuity of the auditory apparatus. MR angiography is useful for differential diagnosis, especially if associated with a scrupulous clinical exam (the possibility to alleviate tinnitus by applying pressure to the jugular vein). With respect to conventional angiography, magnetic resonance is low-risk and less invasive. Unfortunately, detailed resolution is not always optimal and, therefore, other studies should be taken into consideration. In particular, oto-acoustic emissions are easy to carry out, inexpensive, and can, therefore, contribute to identifying the nature of the sound. Recording of the tinnitus also has a dual purpose: it reassures the patient, who might otherwise be neurotic and better defines what the patient means by "*susurrus*".

References

[1] Rodgers GK, Applegate L, De La Cruz A, Lo W. *Magnetic resonance angiography: analysis of vascular lesions of the temporal bone and skull base*. Am J Otol 1993;14:56-62.

[2] Austin JR, Maceri DR. *Anterior Communicating Artery Aneurysm presenting as pulsatile tinnitus*. ORL 1993;55:54-7.

[3] Sismanis A. *Otologic manifestations of benign intracranial hypertension syndrome: Diagnosis and Management*. Laryngoscope 1987;97(Suppl.42):1-17.

[4] Sismanis A, Smoker WRK. *Pulsatile tinnitus: recent advances in diagnosis*. Laryngoscope 1994;104:681-8.

[5] Sismanis A, Stam MA, Sobel M. *Objective tinnitus in patients with atherosclerotic carotid artery disease*. Am J Otol 1994;15:404-7.

[6] Cutforth R, Wiseman J, Sutherland RD. *Genesis of cervical venous hum*. Am Heart J 1970;80:488-92.

[7] Glanville JD, Coles RRA. *A family with high tonal objective tinnitus*. J. Laryngol 1971;85:1.

[8] Huizing EH, Spoor A. *An unusual type of tinnitus*. Arch Otolaryng 1973;98:134-6.

[9] Shiomi Y, Tsuji J, Naito Y, Fujiki N, Yamamoto N. *Characteristics of DPOAE audiogram in tinnitus patients*. Hear Res 1997;108:83-8.

[10] Legent F, Boutet JJ, Beauvillain C, Galiba J. *Acouphènes pulsatiles par lesions vasculaires pré-auriculaires d'origine traumatique*. Ann Oto-Laryng 1985;102:251-3.

[11] Norton SJ, Schmidt AR, Stover LJ. *Tinnitus and otoacoustic emissions – Is there a link?* Ear Hear 1990;11:159-66.

[12] Khabouri S. *Acouphènes pulsatiles par lésions vasculaires pré-auriculaires post traumatiques: à propos d'une observation*. Rev Laryngol Otol Rhinol 1997;118:195-8.

[13] Ceranic BJ, Prasher DK, Luxon LM. *Presence of tinnitus indicated by variable spontaneous otoacoustic emisisons*. Audio Neurotol 1998;3:332-444.

■ Received: 24 April 2002
Accepted: 9 October 2003

■ Address for correspondence: Dr. P. Verde, Sezione di Audiologia, Dipartimento di Neuroscienze e Scienze del Comportamento, Università degli Studi di Napoli "Federico II", Via Sergio Pansini 5, 80131 Napoli, Italy. Fax +39 081 7463581.