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Pulsatile tinnitus – a review of 84 patients

Daniel Waldvogel
Heinrich P. Mattle
Matthias Sturzenegger
Gerhard Schroth

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D. Waldvogel · H.P. Mattle (✉)
M. Sturzenegger
Department of Neurology,
Inselspital, CH-3010 Bern,
Switzerland
Tel.: +41-31-632-3073
Fax: +41-31-632-9679
e-mail: heinrich.mattle@insel.ch

G. Schroth
Department of Neuroradiology,
University of Bern,
Inselspital, Bern, Switzerland

Abstract Pulsatile tinnitus can be annoying for a patient and can also be the only clue to a potentially devastating and life-threatening disease. In order to understand its clinical spectrum and management better we analysed the files of 84 patients seen at our institution over a 10-year period. Noninvasive techniques (ultrasound, computed tomography, magnetic resonance imaging) and angiography were employed as investigations tailored to the individual patient. A vascular disorder [i.e. arteriovenous fistula, dissection of the internal carotid artery (ICA), fibromuscular dysplasia, aneurysm of the ICA and sinus thrombosis] was found in 36 patients (42%), most commonly a dural arteriovenous fistula or a carotid-cavernous sinus fistula. In 26 patients with a vascular abnormality, pulsatile tinnitus was the presenting symptom. In 12 patients (14%), nonvascular disorders such as glomus tumour or intracranial hypertension with a variety of causes explained the tinnitus. We conclude that patients with pulsatile tinnitus should be investigated with noninvasive techniques. If these are negative or to clarify abnormal findings of noninvasive techniques selective angiography is needed for diagnosis and to guide treatment

Key words Tinnitus, pulsatile · Differential diagnosis · Vascular diseases · Glomus tumour · Angiography

Introduction

Pulsatile tinnitus is a rare symptom or sign in patients with neurological disorders [41, 44]. It is often the first or even sole manifestation of a pathology that potentially affects the nervous system in a serious manner [43]. Therefore, familiarity with pulsatile tinnitus is important, especially for neurologists, otologists and radiologists.

From 1985 to 1995 we examined 84 patients with pulsatile tinnitus at our Department of Neurology. In the following, we describe these patients, the differential diagnosis and the investigation of pulsatile tinnitus.

Materials and methods

We reviewed the records of 84 consecutive patients with pulsatile tinnitus. Tinnitus was defined as pulsatile when the patient described a sound synchronous with the heart beat. When the sound was heard by the auscultating examiner with a stethoscope, tinnitus was considered objective. When it was heard only by the patient, it was classified as subjective. All patients had detailed neurological and general medical examinations. This also included testing of hearing, inspection of the external auditory canal and palpation for neck masses. The further patient study depended on the decisions made by the treating physicians. There was no standard algorithm to follow. Sixty-eight patients were investigated by ultrasonography, 26 by computed tomography (CT), 33 by magnetic resonance imaging (MRI), 7 by magnetic resonance angiography (MRA) and 46 by selective arteriography (Table 1). Sixty-two of the patients had more than one examination, 19 ultrasound only, 2 CT only and 1 MRI only.

Patients without a detected cause of pulsatile tinnitus were contacted by letter for follow-up. When they responded, they were invited for an examination at our outpatient clinic.

Table 1 Diagnostic techniques employed in 84 patients with pulsatile tinnitus

	Number of patients	Ultrasound	Computed tomography	Magnetic resonance imaging	Magnetic resonance angiography	Selective angiography
Patients with vascular pathologies	45	40	14	12	5	36
Patients with nonvascular causes	12	2	8	7	6	6
All patients with known aetiologies	57	42	22	19	5	42
Patients with unknown cause	27	26	4	14	2	4
All patients	84	68	26	33	7	46

Table 2 Causes of pulsatile tinnitus found in 84 patients (*ICA* internal carotid artery)

Causes of pulsatile tinnitus		Patients (n)
Vascular	Dural arteriovenous fistula	17
	Carotid-cavernous sinus-fistula	6
	Atherosclerotic carotid stenosis	7
	Dissection of the ICA with stenosis	5
	Fibromuscular dysplasia with ICA stenosis	5
	Cerebral venous sinus thrombosis	2
	ICA aneurysm	1
	Cerebral venous sinus stenosis	1
	Abnormal loop of the anterior inferior cerebellar artery	1
Nonvascular	Glomus tumour	5
	Intracranial hypertension owing to intracranial tumours	4
	Intracranial hypertension owing to aqueduct stenosis	1
	Meningioma	1
	Sarcoidosis	1
Unknown		27

Results

General characteristics

The mean age of the 84 patients was 51 years (range 3–82; 58 women, 26 men). The evaluation of tinnitus was on average 13 months after its onset (range 3 days–6 years).

Aetiology

In 57 of 84 patients (68%) an underlying pathology was found. It was vascular in 45 (54%), nonvascular in 12 (14%) and remained unknown in 27 (32%) (Table 2). The most frequent cause was a dural arteriovenous fistula or a carotid-cavernous sinus fistula, found in 23 patients (27%). The second most frequent cause was an internal carotid artery stenosis. It was found in 17 patients (20%). Atherosclerosis was supposed to be the cause of the stenosis in 7 patients (8%), although only two of them had arteriography to exclude additional pathology. In 5 patients (6%) each, fibromuscular dysplasia or dissection of the internal carotid artery (ICA) was detected. Two patients developed pulsatile tinnitus after a venous sinus thrombosis. A stenosis of the junction between the sigmoid sinus and the transverse sinus was found by arteriography in 1 patient, probably owing to a former venous sinus thrombosis and subsequent partial recanalization. In 1 patient with hemifacial spasm, a loop of a cerebellar artery was thought to cause both hemifacial spasm and pulsatile tinnitus. In 1 patient, an aneurysm of the ICA was detected.

In 12 patients, a nonvascular pathology was found. Five patients (6%) had a glomus tumour. In 5 patients there was intracranial hypertension, owing to a tumour in 4 and to aqueduct stenosis in 1. One patient had a highly vascularized meningioma and 1 a sarcoid meningoencephalitis.

Thirteen of 27 patients in whom the aetiology of pulsatile tinnitus remained unknown answered a letter asking about the outcome and offering a follow-up examination. The mean length of time between the first investigations and the follow-up was 2.7 years (range 1 month–5 years). In 7 of these 13 patients, pulsatile tinnitus had vanished in the meantime. Two patients still suffering from pulsatile tinnitus came to a follow-up examination, but refused additional investigations such as arteriography. Apart from tinnitus, their neurological examination was normal.

Clinical features

Of the 84 patients in our series, pulsatile tinnitus was the presenting symptom in 57 (68%). Of the 45 patients with a vascular pathology, pulsatile tinnitus was the presenting

symptom in 30 (67%). Of the 27 patients with an unknown cause, pulsatile tinnitus was the presenting symptom in 21 (78%). Two of the 5 patients with glomus tumours became symptomatic with pulsatile tinnitus, but none of the 7 patients with other nonvascular pathologies had pulsatile tinnitus as the presenting symptom.

In 15 of the 17 patients (82%) with a dural arteriovenous fistula and in 4 of the 6 patients (66%) with a carotid-cavernous sinus fistula, pulsatile tinnitus was the presenting symptom. Four of the 7 patients (57%) with atherosclerotic disease presented with pulsatile tinnitus, as did 4 of the 5 patients with a fibromuscular dysplasia. Only 1 of the 5 patients with a dissection presented with pulsatile tinnitus. Pulsatile tinnitus was the presenting symptom in 1 of the 2 patients who had suffered from cerebral venous sinus thrombosis, in the patient with an aneurysm of the ICA and in the patient in whom a stenosis of the venous sinus had been found.

Tinnitus was classified as subjective in 28 of the 84 patients (33%), and as objective in 35 (42%), and it was not stated in 21 (25%). In the group of 45 patients with a vascular pathology, pulsatile tinnitus was subjective in 10 patients (22%), objective in 26 patients (58%), not stated in 9 patients (20%). In 15 of the 17 patients (88%) with dural arteriovenous fistulas and in 5 of the 6 patients (83%) with a carotid-cavernous sinus fistula, pulsatile tinnitus was objective. Pulsatile tinnitus was objective in 4 of the 5 patients with a fibromuscular dysplasia and in 1 of the 5 patients with a dissection of the ICA. In 2 of the 12 patients with a nonvascular cause, pulsatile tinnitus was objective, once in a patient with a glomus tumour. Of the 27 patients with an unknown underlying cause, 10 (37%) had a subjective and 7 (26%) an objective pulsatile tinnitus. In the remaining 10 (37%), it was not stated whether pulsatile tinnitus was subjective or objective.

Investigations: vascular pathology

Carotid ultrasonography was performed in 40 of the 45 patients with a vascular pathology. Ultrasound examination suggested the pathology finally found in 18 patients (40%) and was diagnostic in 12 patients (27%). Thirty-six patients were studied by arteriography, which was diagnostic in 35 cases. In 1 patient with a venous sinus thrombosis, the thrombosis had occurred months before our investigation took place and had been diagnosed elsewhere. Craniocerebral CT was performed in 14 of the 45 patients (31%) with a vascular pathology. It was suggestive of the diagnosis in one dural arteriovenous and one carotid-cavernous sinus-fistula, and it was diagnostic in 3 patients (1 patient with venous sinus thrombosis, 1 with a carotid-cavernous sinus fistula and 1 with an aberrant loop of the inferior cerebellar artery). MRI was performed in 12 patients with a vascular pathology, MRA in only 5. It was helpful in detecting the pathology in 5 of them. In 2 patients, MRI suggested a carotid cavernous sinus fistula and in one each an arteriovenous fistula, an aneurysm or a dissection of the ICA.

Sixteen of the 17 patients with a dural arteriovenous fistula were examined by ultrasonography. The most frequent finding was a low resistance flow profile with increased flow velocities in the ipsilateral external carotid and occipital arteries. In 11 of the 16 patients (69%), ultrasound examination suggested the pathology. All 17 patients had arteriography, which confirmed the suspected diagnosis. Of the 6 patients with a carotid-cavernous sinus fistula, 5 had arteriography, which was diagnostic. One patient refused arteriography. His fistula was suspected by ultrasound examination and CT. MRI was performed in 5 of the 17 patients with a dural arteriovenous fistula, MRA in 3. The fistula was shown in 1 patient only.

All seven patients with atherosclerotic stenosis of the ICA had ultrasonography as an initial examination. Arteriography was performed in two of them and confirmed the diagnosis.

Ultrasonography was most helpful in suggesting a pathology in the five patients with a fibromuscular dysplasia affecting the ICA, which was confirmed in all five by arteriography. The typical finding was the combination of distal stenosis in the retromandibular, high cervical ICA segment and absent sclerotic changes in the carotid bulb on B-mode. Of the five patients with a dissection of the ICA, four had an ultrasound examination, which showed the typical findings in three. Arteriography was performed in four and MRI in only one to demonstrate the haematoma of the arterial wall.

Non vascular pathology

The 12 patients with a nonvascular pathology were investigated by CT (8), MRI (7), arteriography (6) and ultrasonography (2). The diffuse metastases on the skull base in 1 patient were seen by MRI only; in all of the other patients, either CT or MRI was able to show the underlying pathology. Arteriography was preoperatively performed in 1 patient with a meningioma and in 1 patient thought to suffer from basal meningitis owing to sarcoidosis, ultrasonography in 1 patient with a pontine astrocytoma who was referred because of dizziness, visual disturbances, headache and pulsatile tinnitus. The 5 patients with a glomus tumour were investigated either by CT, MRI and arteriography (1), by CT and arteriography (2) or by ultrasonography, MRI and arteriography (1). In 1 patient, it was impossible to retrieve the full medial records.

Unknown pathology

Twenty-six (96%) of the 27 patients had had an ultrasound evaluation, 14 (52%) MRI (2 with MRA), 4 (15%) CT and 4 (15%) arteriography. None of these investigations showed an abnormality that could explain pulsatile tinnitus.

Table 3 Causes of pulsatile tinnitus reported in the literature (*AVM* arteriovenous malformation, *CCA* common carotid artery)

Type of lesion	Aetiology
Arterial lesions	Extracranial AVMs [58]
	Dural arteriovenous fistula [2, 13, 23, 24, 34, 38, 55]
	Carotid cavernous fistula [42]
	Aneurysm of the ICA [3, 12]
	Fibromuscular dysplasia of the ICA [17, 56]
	Dissection of the ICA [33, 50]
	Atherosclerosis [7, 16, 30, 44]
	Occlusion of the contralateral CCA [35]
	Vascular anomalies of the ear [8, 20, 28, 49]
	Vascular compression of the VIII nerve [27, 37]
	Migraine [52]
Venous lesions	Jugular bulb anomalies [1, 39]
	Abnormal condylar and mastoid emissary veins [19, 25]
Skull base, temporal and petrous bone lesions	Glomus tumor [26, 36, 48]
	Paget's disease [14]
	Cavernous haemangioma [51]
	Histiocytosis X [5]
Miscellaneous disorders	Intracranial hypertenison of various causes [14, 31, 32, 45, 46]
	Anaemia, high cardiac output [9, 11]
	Palatal myoclonus [4, 22]
	Abnormally patent eustachian tube [40]

Discussion

Pulsatile tinnitus can be the presenting and only symptom of a serious and potentially life-threatening pathology [44] (Table 3). Pulsatile tinnitus can also be one of many symptoms of a neurological disorder and may guide the clinician to the aetiology of the condition. Many patients have to be asked whether tinnitus is present. On the other hand, pulsatile tinnitus is reported spontaneously when it is distressing to patients. Some distressed patients are willing to take the risk of surgery for correction of harmless vascular variations [6] or may even commit suicide [29].

Pulsatile tinnitus is almost always the result of the sound of nonlaminar blood flow that is transmitted to the inner ear. This can occur in systemic diseases causing a general alteration of the haemodynamics or in local disorders that are anatomically close to or within the petrous bone. Therefore the causes of pulsatile tinnitus are multiple. They include systemic disorders with high cardiac output (anaemia, thyrotoxicosis, valvular heart disease) [9, 11], circumscribed vascular variations [1, 8, 19, 20, 25, 27, 28, 37, 49], arteriovenous malformations, dural arteriovenous fistulas [2, 3, 10, 12, 13, 23, 24, 34, 38, 42, 55, 58] or arterial wall diseases causing stenosis such as dissections, fibromuscular displasias or atherosclerosis [7, 16–18, 30, 33, 35, 44, 50, 56], skull base tumours [26, 36, 48, 51], intracranial hypertension [31, 32, 45, 46] and rare diseases such as Paget's disease [14] or histiocytosis X of the petrous bone [5].

Patients with pulsatile tinnitus may show symptoms or signs suggestive of the aetiology: head or neck pain or Horner's syndrome indicate ipsilateral carotid dissection; proptosis or chemosis suggest ipsilateral carotid-cavernous fistula; amaurosis fugax occurs in ipsilateral carotid stenosis; palpable carotid bifurcation tumour indicates ipsilateral glomus caroticum tumour; vanishing of tinnitus on compression of the occipital artery may indicate dural arteriovenous fistula to sigmoid or transverse sinus; papilloedema is evidence of intracranial hypertension; abnormalities of the ear drum suggest glomus tympanicum tumour. In carotid artery dissection, pulsatile tinnitus may transiently appear during progression of the stenosis, disappear when pseudo-occlusion occurs and reappear transiently when the vessel recanalizes [50].

The diagnostic approach should always be guided by the symptoms and physical findings [41]. For patients with objective pulsatile tinnitus, i.e. tinnitus heard both by the patient and the examiner, the first suggested diagnostic step is high resolution CT, if the abnormality is sought in the petrous bone [21, 41], or MRI combined with MRA if an intracranial pathology is suspected [15, 54]. If CT or MRI do not show any abnormality, ultrasound examination [53], MRA [15, 54] or selective intra-arterial arteriography [41, 43, 57] is employed as the next additional investigation. In our series, dural arteriovenous fistulas were the most frequent cause of objective tinnitus. Some of them were detected only by selective intra-arterial angiography, which was also used for endovascular treatment of these vascular abnormalities.

In a patient with subjective tinnitus, i.e. tinnitus heard by the patient only, and negative noninvasive tests, it is uncertain how to proceed [21]. The questions are therefore how often a serious underlying pathology presents as subjective pulsatile tinnitus and how often this pathology is missed by noninvasive investigations and can be detected by arteriography only.

In this series, pulsatile tinnitus was the presenting symptom and subjective only in 8 (18%) of the 45 patients with a vascular pathology. Five of them had a treatable disorder (2 dural arterio-venous fistulas, 1 carotid-cavernous sinus fistula, 1 aneurysm). In 1 of the patients with

subjective pulsatile tinnitus and a dural arteriovenous fistula, all of the noninvasive tests were negative. Of the 27 patients with an unknown aetiology, 16 presented with subjective or unclassified pulsatile tinnitus. The follow-up questionnaire showed that in 6 of the 13 patients who had responded, pulsatile tinnitus had persisted. An underlying vascular malformation might therefore be suspected in these patients. However, these patients declined further investigations.

A recent prospective study using MRI and MRA demonstrated that 42% of the patients with subjective pulsatile tinnitus suffered from pathologies such as paraganglioma, high jugular bulb, jugular bulb diverticula, carotid dissection, arteriovenous malformation (AVM) and stenosis of the transverse sinus [15]. Patients with negative MRI had no further investigation. Therefore the true number of patients with a clinically relevant cause of pulsatile tinnitus might be even higher. The value of MRA cannot be evaluated in our series, because most patients were seen before we used this imaging method. Most frequently we used neurovascular ultrasonography. It was performed in 40 of the 45 patients with a vascular pathology. In 30 (75%) of them, there was an abnormal finding on ultrasonography. Of the 23 patients with a dural arteriovenous or a carotid-cavernous sinus fistula, 22 had ultrasonography. It was abnormal in 15 patients, resulting in a sensitivity of 68%. According to these data, ultrasonography was a useful initial investigation and helped to direct further study. However, a negative ultrasound result is by no means reliable enough to rule out a serious underlying pathology. Therefore, in patients with negative ultrasound findings, angiography should be considered, which is also needed for endovascular treatment [53].

Unlike other series, the so-called „benign" intracranial hypertension syndrome did not occur in our group. In other series, this was considered to be the most frequent cause of pulsatile tinnitus [44, 46]. In our patients with raised intracranial pressure, an underlying pathology was found in all.

The spontaneous course of pulsatile tinnitus with undetected origin is unknown. Few studies report a follow-up. In 7 of the 13 patients whose cause of tinnitus remained unknown and who responded to our written questionnaire, the tinnitus had ceased. The 6 patients with persistent pulsatile tinnitus were either unwilling to come to a follow-up examination (4) or to undergo any further investigation (2, one with subjective, one with objective pulsatile tinnitus).

In conclusion pulsatile tinnitus whether subjective or objective can be a symptom or sign of a serious pathology. Noninvasive examinations such as neurovascular ultrasonography, CT or MRI and MRA have the potential to disclose the underlying cause. However, to rule out a vascular pathology with certainty and to guide treatment, selective cerebral angiography is needed.

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