Carotid Doppler Ultrasound Evaluation in Patients with Pulsatile Tinnitus

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Abstract A wide range of conditions, including atherosclerosis, may lead to pulsatile tinnitus. The objective of this study was to evaluate the patients with pulsatile tinnitus by means of carotid Doppler ultrasound parameters. Thirty-four patients with pulsatile tinnitus were enrolled in the study. Internal carotid artery intima-media thickness (IMT), blood flow velocities and arterial lumen diameters (LD) were measured and compared with the control group. Mean end diastolic velocity (EDV) (32.29 ± 17.22) and IMT (1.06 ± 0.36) values were significantly higher and mean LD (6.69 ± 1.20) was significantly lower compared to the control group. Peak systolic velocity and EDV values were significantly higher in patients whose IMT were greater than 1 mm. Pulsatile tinnitus may be the first symptom of a severe, life threatening disease. Doppler sonography is a noninvasive, safe and easily applicable

diagnostic tool for the evaluation of the vascular structures, in patients with pulsatile tinnitus.

Keywords Pulsatile tinnitus · Carotid atherosclerosis · Doppler ultrasonography

Introduction

Tinnitus is perception of sound in the ear in the absence of an external stimulus [1]. Pulsatile tinnitus is defined as tinnitus that is rhythmic and synchronous with heartbeat [2, 3]. Approximately 4 % of all tinnitus patients are pulsatile [4]. Pulsatile tinnitus is usually caused by vibrations from turbulent blood flow in arteries or vessels [5, 6].

Causes of pulsatile tinnitus include arteriovenous shunts, arterio-arterial anastomoses and intraluminal irregularities of the carotid system (carotid atherosclerosis and atherosclerotic carotid stenosis, fibromuscular dysplasia), aberrant carotid artery, heart murmurs and middle ear vascular neoplasms [7–10].

Although there is no comprehensive elucidatory data on pulsatile tinnitus, some authors relate it to venous hum caused by turbulent blood flow through the internal jugular vein, undiagnosed increased intracranial pressure or anatomic abnormalities of IJV in close proximity of transverse process of atlas [9, 11]. Liyange et al. [8] have postulated that patients may become aware of their physiologic pulsatile tinnitus as a result of altered states of arousal and selective attention, otologic disorders such as conductive hearing loss and trauma.

In the present study we investigated the etiologic factors in patients with pulsatile tinnitus and evaluated the patients with no abnormalities by means of internal carotid artery Doppler ultrasound (US).

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Materials and Methods

Thirty-four patients with pulsatile tinnitus were prospectively evaluated, over a period of 16 months in Atatürk Research and Training Hospital, İzmir. The control group consisted of 31 patients who had no history of ear disease. All patients had undergone meticulous clinical evaluation including a thorough medical and otologic history; microotoscopy; auscultation of the mastoid, head, neck and chest. Complete blood count, and clinical biochemistry analysis, pure tone and speech audiometry were performed in all patients. For further investigations, temporal bone magnetic resonance imaging (MRI) and carotid magnetic resonance angiography were done.

All the cases in both groups were evaluated with carotid duplex ultrasonography (NEMIO 20, TOSHIBA Medical, Tokyo, Japan) using 8 MHz transducer. Scanning of the internal carotid arteries in the neck was done bilaterally in longitudinal and transverse projections. Peak systolic velocity (PSV), end diastolic velocity (EDV), lumen diameter (LD) and intima-media thickness (IMT) were examined. PSV, EDV and LD were measured at a level 2 cm distal to the carotid bifurcation. LD was measured between the parallel bright inner layers of the vessel in B-mode images. IMT was defined as the distance between the edge of luminal echo and the edge of media-adventitia echoe.

Comparisons between the control and the study groups were made using independent samples *t* test, Chi square, crosstabs statistical analysis. Statistical analyses were done using SPSS 15.0 for Windows.

The study was approved by the ethics committee of the hospital.

Results

Thirty-four patients (22 women and 12 men) with pulsatile tinnitus comprised the study group (mean age: 53.00 ± 13.57 , range 17–85 years). Thirty-one patients in the control group had a mean age of 48.97 ± 8.54 years (range 32–65 years) and female/male ratio was 22/9.

Pulsatile tinnitus was right sided in 17 of 34 patients; left sided in 11 patients and bilateral in 6 patients. The duration of tinnitus ranged between 1 and 120 months (mean 17.70 ± 4.56). The demographic characteristics of the study and the control groups are given in Table 1. In the study group; seven patients had hypertension, three patients had diabetes mellitus and three patients had both. In the control group one patient had hypertension and one patient had diabetes mellitus (Table 2). Three patients had a high jugular bulb, two patients had atherosclerotic carotid artery disease, two patients had an aberrant carotid artery and one patient had a tortuous internal carotid artery (Fig. 1).

Internal carotid artery doppler US revealed no significant difference for the mean LD, PSV, EDV and IMT values between the two sides, in the study group (Table 3).

The mean values of the ears with pulsatile tinnitus were compared with the control group, and mean EDV and IMT

Table 2 Comorbidity chart

Comorbidity	Study group	Control group
Hypertension	7 (20.6 %)	1 (3.2 %)
Diabetes mellitus	3 (8.8 %)	1 (3.2 %)
Hypertension + diabetes mellitus	3 (8.8 %)	0 (0.0 %)
No comorbidities	21 (61.8 %)	29 (93.5 %)

Fig. 1 Disorders in pulsatile tinnitus patients

Table 1 The demographic characteristics of the study and the control groups

	Study group (<i>n</i> = 34)	Control group (<i>n</i> = 32)	<i>P</i>
Age (mean $\pm$ SD)	53.00 ± 13.57	48.97 ± 8.54	0.161
Weight (mean $\pm$ SD)	75.09 ± 15.23	72.77 ± 14.44	0.533
Height (mean $\pm$ SD)	161.03 ± 8.06	163.10 ± 7.99	0.304
Gender			
Male	12 (35.29 %)	9 (29.03 %)	0.596
Female	22 (64.71 %)	22 (70.97 %)	
BMI (mean $\pm$ SD)	29.24 ± 5.98	27.30 ± 4.48	0.147

Table 3 Comparison of right and left LD, PSV, EDV and IMT in the patient group

	Right Mean $\pm$ SD	Left Mean $\pm$ SD	<i>P</i>
LD (mm)	6.60 $\pm$ 1.23	6.96 $\pm$ 1.54	0.105
PSV (cm/s)	72.00 $\pm$ 21.18	72.12 $\pm$ 23.65	0.958
EDV (cm/s)	32.00 $\pm$ 16.50	33.23 $\pm$ 17.32	0.459
IMT (mm)	1.02 $\pm$ 0.37	1.05 $\pm$ 0.40	0.273

Table 4 Comparison of LD, PSV, EDV and IMT between the ears with tinnitus and the control group

	Study group (<i>n</i> = 31) Mean $\pm$ SD	Control (<i>n</i> = 62) Mean $\pm$ SD	<i>P</i>
LD (mm)	6.69 $\pm$ 1.20	7.35 $\pm$ 1.16	0.012*
PSV (cm/s)	71.26 $\pm$ 23.01	79.06 $\pm$ 88.16	0.630
EDV (cm/s)	32.29 $\pm$ 17.22	23.69 $\pm$ 7.51	0.001*
IMT (mm)	1.06 $\pm$ 0.36	0.86 $\pm$ 0.15	0.000*

* Statistically significant

values were significantly higher than the control group whereas mean LD values were significantly lower (Table 4).

The mean triglyceride level was significantly higher for the study group (173.26 mg/dl) than for the control group (120.42 mg/dl) ($P < 0.05$). Comparison of IMT for different levels of triglyceride, total cholesterol and BMI are shown in Table 5.

Comparison of PSV and EDV values based on IMT revealed statistically significant results between patients with IMT >1 mm and IMT <1 mm (Table 6).

Discussion

Pulsatile tinnitus almost always originates from conduction of non-laminar blood flow sounds to the inner ear. Pulsatile tinnitus emanates from systemic diseases that disturb haemodynamics or local disorders in or in the vicinity of petrous bone [12]. Anemia, thyrotoxicosis, valvular heart disease, vascular lesions, neoplasms, intracranial hypertension belong to the group of diseases which result in

Table 6 LD, PSV, EDV in patients with IMT <1 mm and >1 mm

	IMT <1 mm (<i>n</i> = 40) Mean $\pm$ SD	IMT >1 mm (<i>n</i> = 25) Mean $\pm$ SD	<i>P</i>
LD (mm)	5.96 $\pm$ 3.03	6.72 $\pm$ 1.24	0.239
PSV (cm/s)	54.70 $\pm$ 30.04	85.46 $\pm$ 69.49	0.017*
EDV (cm/s)	21.03 $\pm$ 12.46	29.66 $\pm$ 17.44	0.023*

* Statistically significant

pulsatile tinnitus [5, 8]. The major arterial and venous structures of the head and neck area are in close contact with hypotympanum. The thin bony plate over the internal carotid artery (ICA) separates these vessels from the basal turn of cochlea [8]. Normally, the blood stream is laminar and noiseless in blood vessels. The disturbance of laminar flow and occurrence of turbulence may cause vessel wall vibration which result in audible sounds [11, 12].

The evaluation of patients with pulsatile tinnitus requires whole blood count and thyroid function tests in addition to complete history and physical examination, because anemia and thyroid function disorders may increase the cardiac output and cerebral blood flow which result in pulsatile tinnitus. High serum lipid levels increase the risk of stenosis in cervical arteries [8, 13]. In our patients, hemogram and thyroid function tests (TFTs) were within normal limits. Serum triglyceride levels were significantly higher in the tinnitus group than in controls. Although statistically not significant, mean total cholesterol level was above 200 mg/dl.

Sismanis et al. have noted that diagnosis of pulsatile tinnitus should be individualized according to the symptoms and clinical signs of the patients. Moreover, they suggested that temporal bone magnetic resonance (MR) imaging and carotid MR angiography is required in pulsatile tinnitus patients with normal tympanic membranes; high resolution computed tomography (CT) scan of the temporal bone in patients with retrotympenic lesions; color Doppler US in patients with risk factors for atherosclerosis. They also suggested that selective carotid angiography is not indicated unless tinnitus is objective, auscultation of the head and neck reveals a bruit, presence of an arteriovenous malformations (AVM), an arteriovenous fistula

Table 5 IMT comparisons based on BMI, serum triglyceride and cholesterol levels

	BMI		Triglyceride (mg/dl)		Cholesterol (mg/dl)	
	<25 (<i>n</i> = 12)	>25 (<i>n</i> = 53)	<150 (<i>n</i> = 36)	>150 (<i>n</i> = 29)	<200 (<i>n</i> = 35)	>200 (<i>n</i> = 30)
IMT	0.705 $\pm$ 0.27	0.847 $\pm$ 0.43	0.777 $\pm$ 0.34	0.878 $\pm$ 0.48	0.774 $\pm$ 0.39	0.878 $\pm$ 0.43
<i>P</i>	0.305		0.341		0.319	

(AVF) or fibromuscular dysplasia, or the patient is a candidate for glomus tumor surgery [9, 14–16].

US studies and radiographic examinations are of utmost importance in patients with pulsatile tinnitus [8, 14, 17]. Daneshi et al. [18] found atherosclerosis of the carotid artery as the cause of pulsatile tinnitus using color Doppler evaluation in 11.76 % of the patients. Waldvogel et al. [12] reported ICA stenosis in 17 of 84 patients (20 %) and noted that atherosclerosis was the probable cause in 7 (%8) of these patients. Sismanis et al. [9] reported atherosclerotic carotid artery disease (ACAD) as the cause in 16 % of the patients with pulsatile tinnitus. Bertora et al. had studied supratrochlear artery and evaluated arterial parameters (maximum systolic velocity-MSV and EDV). They found increase in MSV and no significant difference in EDV parameters [19]. In our study group two patients had carotid artery atherosclerosis (5.88 %).

In the present study, we measured the internal carotid artery lumen diameters, IMT and blood flow velocities in patients with pulsatile tinnitus. Comparisons of the mean values from the study and the control groups revealed significant differences between groups: the EDV and IMT values measured from the sides with tinnitus were greater, and the lumen diameters were smaller, which were statistically significant. The difference was not statistically significant for PSV.

Increased carotid IMT and plaque formation are considered as early signs of atherosclerosis [20]. Increased carotid wall thickness has been shown to be a significant predictor of coronary and cerebrovascular diseases [21–23]. In our study, mean IMT was 1.05 and 0.85 mm for the patient and the control groups, respectively. The difference was significant statistically. Moreover, the triglyceride level, IMT and presence of coexisting diseases (HT, DM) were significantly higher in the study group as compared with the control group. This showed an increased risk of atherosclerotic artery disease for the study group.

Doppler sonography is the most common imaging study performed for the diagnosis of carotid disease. ICA PSV and ICA EDV are Doppler parameters which are used in the detection of ICA stenosis [24]. Carotid IMT has been shown to reflect the properties of the vascular wall [25]. Atherosclerosis not only causes stenosis mechanically by means of plaque formation, but also causes endothelial dysfunction and change in vascular compliance [26, 27]. In our study, the PSV and EDV values were significantly higher in patients whose IMT were greater than 1 mm. We suggest that increased IMT, i.e. atherosclerosis causes alteration in blood flow velocity and this results in turbulence in bloodstream. The turbulence generates pulsatile tinnitus despite absence of stenosis in internal carotid artery.

In the present study, increased risk of atherosclerosis and increased flow velocity was seen in patients with PT.

This implies that, despite presence of significant narrowing in carotid arteries, PT may be associated with turbulent blood flow secondary to alterations in vessel compliance and increase in blood flow velocity, even in early stages of atherosclerosis.

Summary

Pulsatile tinnitus may be the first symptom of a severe, life threatening disease. Thus, a comprehensive evaluation is required for diagnosis. CT, MRI, MRA and US are non-invasive methods for the evaluation of the underlying causes. Doppler sonography is a noninvasive, safe and easily applicable diagnostic tool for the evaluation of the vascular structures. In the present study, increased risk for atherosclerosis and increased flow velocity were observed in pulsatile tinnitus patients. This fact suggest that pulsatile tinnitus is secondary to the turbulent blood flow due to the alteration in vessel compliance and increase in blood flow velocity in the early stages of atherosclerosis, although there is no significant stenosis in the carotid arteries.

Conflict of interest The authors declare that they have no conflict of interest.

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