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CASE REPORT **OLGU SUNUMU**

A RARE CAUSE OF PULSATILE TINNITUS: ENLARGED POSTERIOR CONDYLAR VENOUS

Baki DOĞAN[1], Zehra UYSAL KOCABAŞ[2], Hasan BAYINDIR[3], Fatma GER AKARSU[2], Özlem AYKAÇ[2], Atilla Özcan ÖZDEMİR[2]

[1]Ondokuz Mayıs University Faculty of Medicine, Department of Neurology, Samsun, TÜRKİYE
[2]Eskişehir Osmangazi University Faculty of Medicine, Department of Neurology, Eskişehir, TÜRKİYE
[3]Ankara City Hospital, Neurology Clinic, Ankara, TÜRKİYE

ABSTRACT

Pulsatile tinnitus (PT) is a rare symptom affecting patients quality of life. Detailed clinical history and otoscopic examination are essential in clarifying the etiologic cause. Arterial or venous pathologies should be considered in the differential diagnosis and evaluation with advanced imaging methods. Various vascular conditions such as arterial murmur, venous hum, arteriovenous malformations, and vascular tumors can cause pulsatile tinnitus. Venous pathological abnormalities and variants are often overlooked, although they are among the common causes of PT. These vascular structures may be located close to the auditory pathways and cause pulsatile tinnitus with the effect of turbulence. If non-invasive imaging is found to be normal in the etiology of PT, diagnostic angiography should be performed. In this case report, we present a patient with unilateral pulsatile tinnitus, which we think is caused by abnormally enlarged posterior condylar veins detected on angiographic imaging.
Keywords: Pulsatile tinnitus, condylar veins, diagnosis angiography.

PULSATİL TİNNİTUSUN NADİR BİR NEDENİ: DİLATE POSTERİOR KONDİLLER VENLER

ÖZ

Pulsatil tinnutus (PT) hastaların yaşam kalitesini etkileyen nadir görülen bir semptomdur. Ayrıntılı klinik öykü ve otoskopik muayene etiyolojik nedeni netleştirmede önemli yer tutar. Ayrıcı tanıda arteriyal veya venöz patolojiler düşünülmeli, ileri görüntüleme yöntemleri ile değerlendirme yapılmalıdır. Arteriyel üfürüm, venöz hum, arteriovenöz malformasyonlar ve vasküler tümörler pulsatil tinnutusa yol açabilir. Venöz patolojik anormallikler ve varyantlar, PT'nin yaygın nedenleri arasında olmalarına rağmen genellikle göz ardı edilir. Bu vasküler yapılar işitme yolaklarına yakın yerleşim gösterebilir ve türbülans etkisiyle pulsatil tinnutusa neden olabilir. Etiyolojisinde non-invaziv görüntülemeler normal saptanırsa diagnostik anjiografi yapılmalıdır. Bu yazıda, diagnostik anjiografik görüntülemede saptadığımız dilate posterior kondiler venlerin neden olduğunu düşündüğümüz tek taraflı pulsatil tinnutusu olan hastayı sunuyoruz.
Anahtar Sözcükler: Pulsatil tinnitus, kondiler ven, diagnostik angiografi.

Address for Correspondence: Baki Doğan MD, Ondokuz Mayıs University Faculty of Medicine, Department of Neurology, Samsun, Türkiye.
Phone: 0362 312 19 19 **E-mail:** dr.bakidgn@gmail.com
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ORCID IDs: Baki Doğan 0000-0003-2526-9279, Zehra Uysal Kocabaş 0000-0002-1838-9988, Hasan Bayındır 0000-0002-9260-2230, Fatma Ger Akarsu 0000-0003-3171-4535, Özlem Aykaç 0000-0003-4987-0050, Atilla Özcan Özdemir 0000-0002-9864-6904.
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INTRODUCTION

Tinnitus is the perception of sounds such as ringing, buzzing, humming, or hissing in the absence of an external sound stimulus. It comes from the Latin word "tinnire", which means to play. About 12-30% of the general population experience tinnitus and are aware of it. Although tinnutus is temporary and not bothersome in general, it may affect the daily life of approximately 0.5%-3% of the patients, and its prevalence increases with age. Its risk factors include hearing loss, exposure to noise, family history of hearing or tinnitus, tempormandibular joint syndrome, eustachian dysfunction, hypertension, diabetes, aminoglycosides, salicylates, loop diuretics, and ototoxic drugs such as quinine. The pulse can be synchronous or asynchronous. Tinnitus can be categorized as constant or intermittent, acute, subacute or chronic, unilateral or bilateral (1).

Pulsatile tinnitus (PT) refers to tinnitus that has a rhythmic sound alternating in the high and low frequency range, with interruptions with periods of silence between sounds. PT is subclassified as vascular or non-vascular. Vascular causes are divided into three as arterial, venous or arteriovenous (2) (Table) (3). Arterial causes are atherosclerotic carotid artery disease, aneurysms, fibromuscular dysplasia, dissection, and permanent stapedial artery. Pathological abnormalities of the lateral venous sinus, emissary vein, jugular vein anomalies and variants are venous causes (4). Pregnancy, anemia, thyrotoxicosis, bone dysplasia, Paget's disease, otosclerosis, and tumors such as paraganglioma and cholesteatoma are among the less common non-vascular etiologies (2).

Pulsatile tinnitus is clinically diagnosed based on a comprehensive physical examination with attention to detailed clinical history and otoscopic examination. There is no definite algorithm for examining the etiology. Although non-invasive imaging methods are frequently used for central causes, these methods may not detect some vascular pathologies. Therefore, digital subtraction angiography (DSA) is considered the gold standard in detecting pathologies such as dural arteriovenous fistula, transverse sinus stenosis, sigmoid sinus diverticulum, and enlarged emissary veins (5).

Table. Vascular causes of pulsatile tinnitus.

Arterial
Aneurysm
Carotid artery stenosis
Carotid artery dissection
Fibromuscular dysplasia
Aberrant carotid artery
Persistent stapedial artery
Vascular loop
Arteriovenous
Dural arteriovenous fistula
Carotid-cavernous fistula
Arteriovenous fistula
Venous
Sigmoid sinus dehiscence
Sigmoid sinus diverticulum
Transverse sinus stenosis
High jugular bulb
Jugular bulb dehiscence
Jugular bulb diverticulum
Dilated emissary vein (mastoid, condylar, petrosquamous)
External jugular vein compression

*(Revised from reference 3).

CASE REPORT

A 43-year-old female patient was admitted to our clinic with the complaint of intermittent, high-pitched tinnitus in her left ear. The patient had a history of tinnitus simultaneous with her pulse for 6 years in situations that caused physical stress, such as fatigue, exhaustion, and emotional depression. Her tinnitus increased when she felt tired during the day. The patient described that tinnitus occurs when she stands for a long time and decreases when she presses the left side of her neck with her hand. She stated that on days when she was extremely stressed and tired, she could only sleep laying on her left side. She had no complaints of hearing loss, ear fullness, hyperacusis, dizziness or headache. There was no history of head or neck trauma, exposure to ototoxic drugs, or excessive noise. She was using sertraline 50mg/day due to her depressive symptoms. Physical examination was normal. No papilledema or atrophy was detected at the base of the eye in the neurological examination. Cranial nerve examination was normal; there was no gaze palsy. Motor, sensory and cerebellar examinations were normal. Heart and carotid auscultation were normal. The otoscopic examination also revealed normal external ear canal and tympanic membrane. No objective tinnitus was heard in the examination performed during the symptom. No pathology was detected in the pure tone audiogram. Laboratory examination, lumbar

puncture, temporal computed tomography (CT), cranial magnetic resonance imaging (MRI), and venography were planned for differential diagnosis. Laboratory findings, including thyroid function tests and anemia parameters, were normal. Lumbar puncture could not be performed because the patient refused. Conventional MRI sections and venography showed no imaging findings indicative of increased intracranial pressure or venous thrombus. No dehiscence (separation) or diverticulum anomalies were detected on the temporal bone CT. Significant dilatation of the left posterior condylar veins was observed in the venous phase in four-vessel selective DSA, which was planned to detect vascular pathologies such as treatable arterio-venous fistula and aneurysm (Figure). Our case with clarified diagnosis was followed up conservatively. Informed consent was signed by the patient for this report.

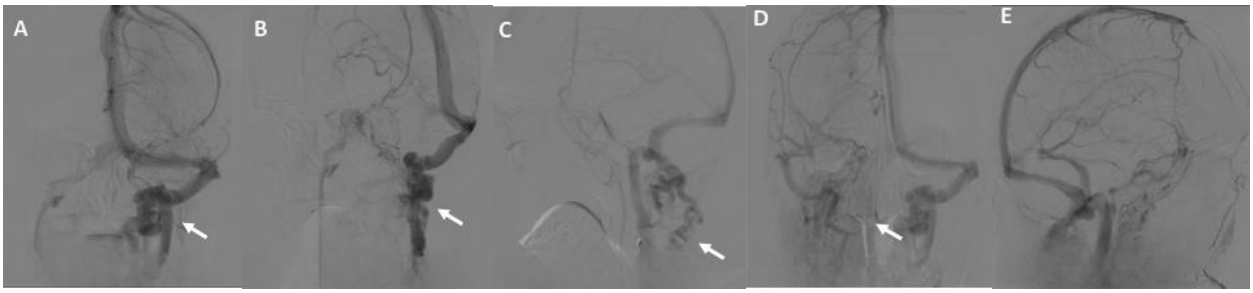

Figure. Anterior-posterior (A), oblique (B), lateral (C) in venous phase with left common carotid artery injection; Significant enlargement of the left posterior condyle veins (white arrows) in anterior-posterior (D), lateral (E) projection in the venous phase with right common carotid injection.

DISCUSSION AND CONCLUSION

The literature on the causes of pulsatile tinnitus is heterogeneous. Idiopathic intracranial hypertension (IIH), arterial stenosis and arteriovenous fistulas, sigmoid wall separation, diverticulum and idiopathic have been reported as the most common causes of PT (2,6-9). Recent literature has characterized the structural and/or flow abnormalities of intracranial and extracranial venous structures as the most common causes of PT (2,7). Venous causes underlie most cases of otherwise unexplained essential, idiopathic, and/or obscure tinnitus. Multiple etiologic causes of venous PT may be present in the same patient. Therefore, it is crucial to identify other possible causes of PT even after identifying a likely cause (4,10,11). Lumbar puncture was recommended for the diagnosis of IIH, but was refused by the patient, so it could not be performed. The diagnosis of IIH was not considered in the foreground due to the absence of visual loss and papilledema, and the absence of findings suggestive of increased intracranial pressure on imaging. Based on all etiological examinations, no cause other than posterior condylar venous enlargement was found in our case.

Emissary veins are small, valveless veins that connect the extracranial and intracranial veins through the skull foramen. They are alternative drainage routes for the meningeal and cerebral venous systems. Due to the absence of valves, they can act as a portal for infection and spread of tumors to the calvarium. Three main groups of emissary veins are categorized by their associated venous sinuses. The superior petro-squamosal emissary vein is formed at the junction of the transverse and sigmoid sinuses. The middle or mastoid emissary vein connects the sigmoid sinus with the posterior auricular or occipital vein. The posterior condylar vein arises from the sigmoid sinus above the jugular bulb. The diameters of these veins generally vary between 1-4 mm (12). Enlarged condylar venous structures can often be seen on normal angiographic examination. These structures may enlarge in pathologies such as increased intracranial pressure, arteriovenous malformation or dural sinus thrombosis (13,14). A case of enlarged posterior condylar emissary vein associated with pulsatile tinnitus in a young woman has been reported in the literature. In this case, the improvement of tinnitus with compression of the left jugular vein on the symptom side suggested that enlarged condylar emissary veins may lead to pathology (15). In vascular pathologies, tinnutus may be exacerbated

by straining, standing, bending or valsalva maneuvers. Patients can realize that they can reduce or eliminate tinnitus by sleeping in an affected-side dependent position. Our case was also able to reduce her symptoms, which had been going on for years, with these methods.

The most common causes of pulsatile tinnitus (PT), venous variants and pathological abnormalities, should be considered in both examination and therapeutic evaluation. These pathologies can be intervened by surgical or endovascular techniques that prevent flow (ligation or embolization) through the abnormal structure (16,17). Although randomized controlled studies are limited, the turbulence region can be selectively eliminated with endovascular treatments. To the best of our knowledge, there is no case of pulsatile tinnitus caused by enlarged posterior condylar venous structure treated with endovascular therapy in the literature.

Enlargement of the posterior condylar veins should be considered among the etiological causes of PT. In fact, more research is needed on the causes of PT in general.

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Ethics

Informed Consent: The authors declared that informed consent form was signed by the patient.

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