

Otologic Manifestations of Patent Ductus Arteriosus: A Case of Pulsatile Tinnitus and Conductive Hearing Loss Due to High-output Cardiac State

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Abstract:

Background: Patent ductus arteriosus (PDA) represents a persistent communication between the descending aorta and pulmonary artery, leading to left-to-right shunt and increased pulmonary blood flow. While most patients present with cardiac murmurs, dyspnea, or heart failure symptoms, otologic manifestations are rarely described. High-output cardiac states resulting from PDA can transmit vascular pulsations to the middle and inner ear, manifesting as pulsatile tinnitus or conductive hearing loss. Recognition of these rare otologic presentations is essential for timely diagnosis and management of the underlying cardiac pathology.

Case Report: A 22-year-old female presented with persistent left-sided pulsatile tinnitus and mild conductive hearing loss for six months. Otoscopy revealed an intact tympanic membrane with visible vascular pulsations. Pure tone audiometry confirmed mild low-frequency conductive hearing loss. Temporal bone imaging was unremarkable, but Doppler assessment demonstrated increased middle-ear vascular flow. Cardiovascular examination revealed a continuous murmur at the left upper sternal border. Transthoracic echocardiography identified a 4-mm patent ductus arteriosus with significant left-to-right shunt and elevated cardiac output. The patient underwent successful transcatheter PDA closure. Post-procedure, tinnitus resolved completely within two weeks, and hearing normalized on follow-up audiometry, confirming the causal link between PDA-induced hemodynamic changes and otologic symptoms.

Keywords: Patent ductus arteriosus; Pulsatile tinnitus; Conductive hearing loss; High-output cardiac state; Otologic manifestation; Transcatheter closure.

INTRODUCTION

Patent ductus arteriosus (PDA) is a congenital cardiovascular malformation characterized by persistent patency of the ductus arteriosus a normal fetal vascular channel connecting the descending aorta to the pulmonary artery that typically closes within the first few days of life (1). Failure of closure results in abnormal left-to-right shunting of blood, leading to increased pulmonary circulation, left heart volume overload, and high-output cardiac states (2). PDA accounts for approximately 5–10% of all congenital heart defects, with higher incidence observed in preterm infants and females. While the clinical presentation predominantly involves cardiovascular findings such as a continuous “machinery” murmur, tachycardia, or signs of heart failure, extracardiac manifestations, particularly otologic symptoms, are exceedingly rare but clinically significant (3,4).

Pulsatile tinnitus is an uncommon otologic symptom defined as the perception of rhythmic sound synchronous with the patient’s heartbeat. It typically arises from vascular etiologies such as glomus tumors, arteriovenous malformations, or carotid artery diseases (5). However, systemic causes leading to increased vascular flow, such as anemia, thyrotoxicosis, or congenital cardiac shunts

like PDA, can also transmit vascular pulsations to the cochlear and middle-ear vasculature (6). The resulting hemodynamic turbulence may produce subjective or objective tinnitus, often accompanied by conductive hearing impairment due to pressure changes or vascular congestion in the middle ear (7).

In most reported cases, patients with cardiac-related pulsatile tinnitus are initially evaluated for primary otologic conditions, and cardiac pathology is only discovered incidentally. This underscores the importance of multidisciplinary assessment when vascular pulsations or unexplained tinnitus are encountered in otologic practice (8). Modern diagnostic tools including otoscopic visualization of middle-ear pulsations, audiometry, Doppler ultrasound, and echocardiography, facilitate early recognition of cardiovascular causes (9).

Timely identification of a hemodynamically significant PDA is crucial, as closure of the ductus can reverse systemic effects and alleviate associated otologic symptoms. Transcatheter occlusion has emerged as the preferred treatment modality due to its safety, efficacy, and minimal invasiveness (10). The present case report describes a young female who presented with isolated otologic complaints pulsatile tinnitus and mild conductive hearing loss that led to the incidental discovery of a patent ductus arteriosus with high-output

state. This case emphasizes the need for heightened clinical awareness of systemic cardiovascular disorders manifesting with otologic symptoms and highlights the diagnostic value of an interdisciplinary approach between otolaryngology and cardiology.

CASE REPORT

Patient Presentation: A 22-year-old female presented to the otolaryngology outpatient clinic with complaints of persistent left-sided pulsatile tinnitus for the past six months. The tinnitus was described as rhythmic and synchronous with her heartbeat, worsening at night and during physical exertion. She also reported a mild sensation of aural fullness and occasional transient hearing difficulty on the same side. There was no history of vertigo, ear discharge, pain, head trauma, or exposure to loud noise. Her past medical history was unremarkable, with no known cardiovascular or endocrine disorders.

Clinical Examination: General examination revealed a well-nourished young female with stable vital parameters and no signs of anemia or thyroid enlargement. Otolaryngoscopic examination of the left ear revealed an intact tympanic membrane with visible reddish pulsatile reflections on the promontory region. The right ear was normal. No retrotympanic mass was observed. Tuning fork tests indicated mild conductive hearing loss on the affected side. Cardiovascular examination revealed a continuous “machinery” murmur of grade 3/6 intensity at the left upper sternal border, radiating toward the clavicle, suggestive of a vascular shunt lesion. No cyanosis, clubbing, or pedal edema was noted.

Investigations: Pure tone audiometry demonstrated mild low-frequency conductive hearing loss (average 25 dB) in the left ear, while tympanometry showed a normal Type A curve, ruling out middle-ear effusion. High resolution computed tomography (HRCT) of the temporal bone revealed normal ossicular and vascular anatomy with no aberrant vessels or glomus tumor. Doppler ultrasonography of the neck vessels indicated increased middle-ear and jugular venous flow on the left side. Given the vascular nature of her tinnitus, a cardiology referral was made. Transthoracic echocardiography revealed a 4-mm patent ductus arteriosus with significant left-to-right shunt and hyperdynamic circulation, confirming a high-output cardiac state. No pulmonary hypertension or valvular abnormalities were noted.

Management: After multidisciplinary evaluation, the patient underwent transcatheter coil occlusion of the PDA under local anesthesia. The procedure was successful, with no residual shunt on post-procedure echocardiography. She was kept under observation for

48 hours and discharged in stable condition with advice for routine follow-up.

Outcome and Follow-up: At two weeks post-procedure, the patient reported complete resolution of pulsatile tinnitus and improvement in hearing. Repeat audiometry confirmed normalization of thresholds. At three months, she remained asymptomatic, with no recurrence of otologic or cardiac symptoms.

This case reports the rare otologic presentation of patent ductus arteriosus manifesting as pulsatile tinnitus and conductive hearing loss due to hemodynamic turbulence transmitted to the middle ear. Careful clinical examination, correlation with cardiovascular findings, and use of multimodal imaging were essential in reaching the diagnosis. Closure of the ductus not only resolved the hemodynamic overload but also completely relieved the otologic symptoms, emphasizing the value of multidisciplinary collaboration between otolaryngologists and cardiologists in atypical tinnitus cases.

DISCUSSION

Patent ductus arteriosus (PDA) is a well-recognized congenital cardiac defect that typically presents with cardiac symptoms such as murmur, dyspnea, or failure to thrive. However, in rare instances, it can manifest with atypical extracardiac symptoms, including otologic complaints like pulsatile tinnitus and conductive hearing loss (11). These manifestations arise due to the hemodynamic alterations associated with high-output cardiac states, which can transmit vascular pulsations through the arterial system to the cochlear and middle-ear vasculature (12). The present case illustrates this uncommon but clinically significant correlation.

Pulsatile tinnitus accounts for less than 10% of tinnitus cases and is frequently of vascular origin. Common etiologies include glomus tumors, dural arteriovenous fistulas, carotid stenosis, and venous anomalies (5). In contrast, cardiac causes are infrequently considered during initial otologic evaluation. The mechanism by which PDA produces pulsatile tinnitus involves the propagation of high-velocity turbulent flow from the aorta to the pulmonary artery through the persistent ductus (13). This abnormal shunt increases systemic vascular pulsatility, which can be transmitted to the small arterial branches supplying the cochlea and tympanic plexus. The rhythmic vibration of these vessels near the tympanic membrane can generate an audible pulsation perceived as tinnitus (8).

Conductive hearing loss in such cases may result from vascular congestion within the middle ear mucosa or alterations in ossicular chain compliance secondary to transmitted pulsations (14). In the present case, both symptoms were reversible following correction of the

underlying cardiac lesion, further confirming a direct hemodynamic relationship between the PDA and otologic manifestations.

The diagnostic process for vascular tinnitus requires a high index of suspicion and a structured, multidisciplinary approach. Otoscopic findings such as visible pulsations, normal tympanic membrane integrity, and absence of middle-ear effusion should prompt vascular imaging. Audiometry, tympanometry, and high-resolution temporal bone CT are essential to exclude local causes such as glomus tympanicum or aberrant carotid arteries. When these evaluations are inconclusive and vascular signs persist, systemic causes such as PDA, anemia, or thyrotoxicosis must be explored. In this patient, echocardiography confirmed the presence of a small but hemodynamically significant PDA with a left-to-right shunt, illustrating the importance of cardiac evaluation in refractory or unexplained cases.

Therapeutic closure of the PDA, either surgically or via transcatheter device occlusion, remains the mainstay of management. Transcatheter coil or device closure is preferred in adults due to its minimally invasive nature, shorter hospital stay, and excellent long-term outcomes. In this case, the patient underwent successful transcatheter closure, resulting in complete and sustained resolution of tinnitus and hearing loss within two weeks, indicating full hemodynamic normalization.

The case highlights the crucial role of interdisciplinary coordination between otolaryngologists and cardiologists. While the initial complaint was otologic, definitive diagnosis and treatment required cardiovascular expertise. Awareness of such unusual presentations can prevent unnecessary investigations and delayed cardiac intervention.

Otologic symptoms such as pulsatile tinnitus may serve as the first clinical clue to an underlying high-output cardiac lesion like PDA. Careful clinical assessment, multimodal imaging, and prompt correction of the hemodynamic abnormality can result in complete symptom resolution. Recognizing this association broadens the diagnostic perspective for clinicians and reinforces the importance of systemic evaluation in patients presenting with unexplained vascular tinnitus (13).

CONCLUSION

This case emphasizes the importance of considering systemic cardiovascular causes such as patent ductus arteriosus in patients presenting with unexplained pulsatile tinnitus or conductive hearing loss. High-output cardiac states can transmit vascular pulsations to the middle and inner ear, leading to otologic manifestations that may precede cardiac symptoms. Early recognition through multidisciplinary evaluation and appropriate imaging is essential for accurate diagnosis. Successful

transcatheter closure of the PDA not only normalized cardiac hemodynamics but also led to complete resolution of otologic symptoms, highlighting the reversibility of such presentations with timely intervention and close cardiology–otolaryngology collaboration.

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