Imaging examinations in pulsatile tinnitus. A delicate choice

Original Article

Authors

Miguel Padrão
ULS Região Aveiro, Portugal

Diogo Dias
ULS Região Aveiro, Portugal

Margarida Bulha
ULS Região Aveiro, Portugal

Isa Eloi
ULS Região Aveiro, Portugal

Sandra Augusto
ULS Região Aveiro, Portugal

Maria Luísa Azevedo
ULS Região Aveiro, Portugal

Abstract

Pulsatile tinnitus is a symptom/sign that should be quickly identified and properly studied. Their aetiology can be vascular or non-vascular, and their investigation requires imaging characterisation.
This study evaluates the results of imaging exams initially requested in patients with pulsatile tinnitus in the ENT department at ULS Região de Aveiro between 2019 and 2023.
Of the 98 exams analysed, 54.1% corresponded to patients with pulsatile tinnitus. 84.9% of these underwent carotid Doppler ultrasound and 15.1% angio-CT/MRI.
We observed a significant prevalence of hypertension, dyslipidaemia and diabetes in our sample. CT angiography was more effective in detecting alterations compatible with a neurovascular aetiology of pulsatile tinnitus.
The choice of imaging exam should be based on pre-test probability and the need to exclude clinically relevant pathologies.
The use of CTA/MRI should be prioritised in cases of suspected skull base/temporal bone pathology, paraganglioma or neurovascular conflict.

Keywords: Tinnitus; Imaging; Angio-TC; Angioressonance; Echo-Doppler

Introduction

Tinnitus is defined as the perception of sound without an auditory stimulus. It can be classified using various criteria, but it is important to distinguish between pulsatile and nonpulsatile tinnitus. Unilateral pulsatile tinnitus may be the first symptom of severe disease, while nonpulsatile bilateral tinnitus is often benign[1]. Less than 10% of tinnitus cases are pulsatile[2], which may have a vascular or nonvascular etiology. Vascular etiologies comprise arterial (arteriosclerosis, aberrant carotid artery, arteriovenous fistula [AVF] , arteriovenous malformation [AVM], and increased vascularization in Paget's disease) and venous causes (high

Correspondence:
Miguel Padrão
miguelpadrao@campus.ul.pt

Article received on February 26, 2024.
Accepted for publication on June 20, 2024.

jugular bulb and benign intracranial hypertension)[3]. Nonvascular etiologies include paraganglioma, temporal bone pathology, idiopathic intracranial hypertension, and hyperdynamic state (anemia).

Different approaches can be used to confirm the etiological diagnosis of tinnitus[4,5]. A complete clinical history and thorough objective examination are essential, in addition to complementary imaging tests[6]. Doppler ultrasound and computed tomography/ magnetic resonance angiography (CTA/MRA) are useful tools for evaluating the bone and neurovascular structures in patients with tinnitus[7].

Objectives

This study aimed to evaluate the results of complementary imaging tests of outpatients with pulsatile tinnitus treated between 2019 and 2023 at the otorhinolaryngology (ORL) service of the Local Health Unit of the Aveiro Region (ULS-RA).

Materials and methods

We collected the results of 98 imaging tests, of which 53 (54.1%) were of patients with pulsatile tinnitus, and consisted of 45 (84.9%) carotid Doppler ultrasounds and eight (15.1%) CTA/ MRA. Three patients underwent both tests concomitantly.

Results

The sample included 35 (69.4%) women and 15 (30.6%) men. The average age was 58.2 years (16–87 years), with a standard error of the mean of 2.26. Patients without imaging changes were younger (56.98 ± 2.35 years) than patients with imaging changes (73.75 ± 3.35 years) ($p = 0.049$), which included 12 cases (24%) of bilateral tinnitus, 24 (48%) cases of exclusively or predominantly right-sided tinnitus, and 14 (28%) cases of exclusively or predominantly left-sided tinnitus. Among the patients who initially underwent CTA, four (44.4%) presented with changes compatible with pulsatile tinnitus of neurovascular etiology; in contrast, none (0%) of the patients undergoing Doppler ultrasound demonstrated these changes ($p < 0.001$). 25 (55.5%) of the patients who underwent a doppler ultrasound subsequently performed a CTA/MRA. Among them, 19 (76%) exhibited no changes, one (4%) had an acoustic neuroma, and four (16%) had a neurovascular contact between the branches of the anterior inferior cerebellar artery (AICA) and the cisternal segment of the ipsilateral acoustic-facial bundle.

Twenty-five (50%) patients had no hearing loss, nine (18%) presented with right-sided hearing loss, four (8%) with left-sided hearing loss, and nine (18%) with bilateral hearing loss. Three patients (6%) lacked a complete audiometric study. The pure-tone averages (PTA) of patients undergoing audiometry were calculated at 500, 1000, 2000, and 4000 Hz, averaging 29.3 ± 3.4 (Fig. 1). Tinnitus laterality had a statistically significant correlation with the hearing loss laterality ($p = 0.002$), with 45% cases of bilateral tinnitus being associated with bilateral hearing loss. All patients with right sided hearing loss had right-sided tinnitus, and 75% (four) of patients with left-sided hearing loss had left-sided tinnitus (Fig.2).

Figure 1
Pure-tone average (PTA) distribution

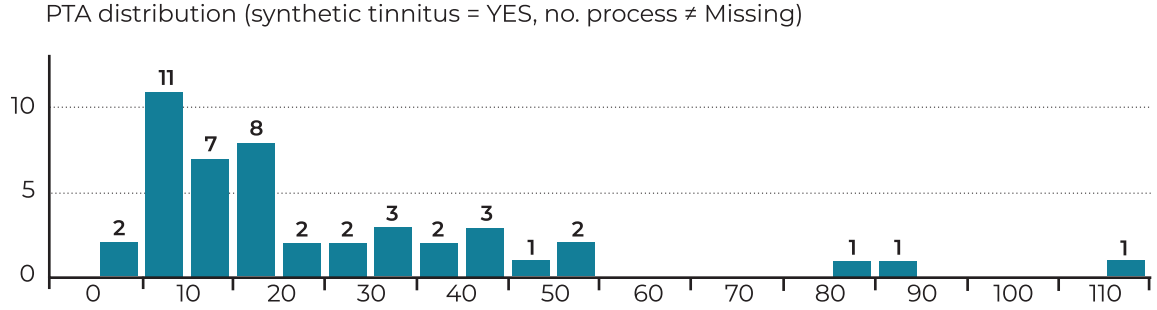
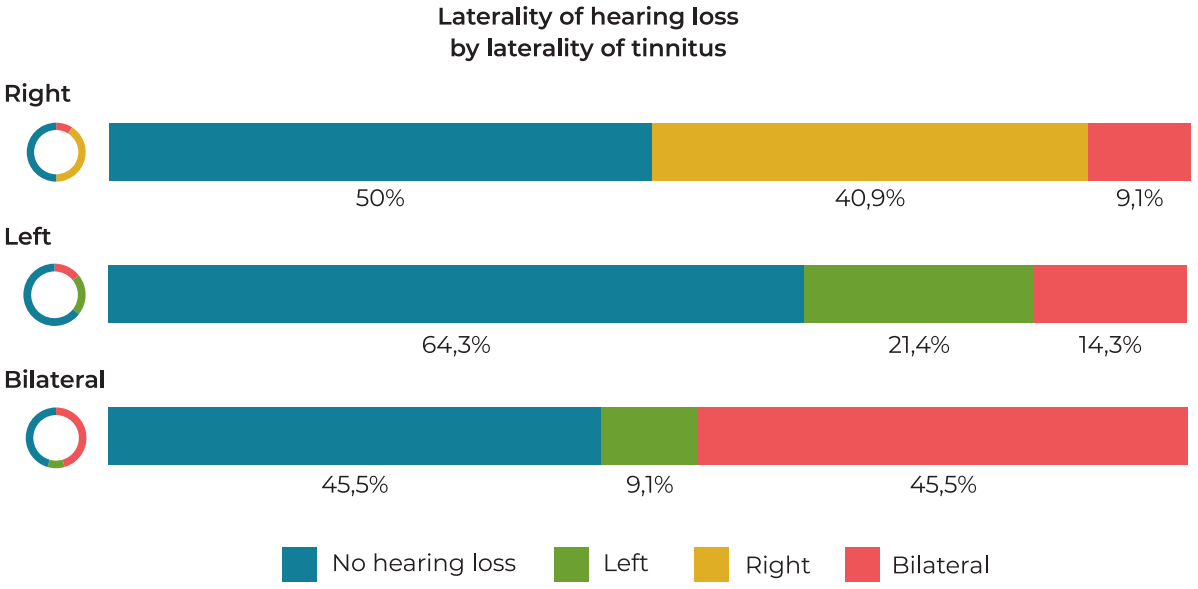


Figure 2
Distribution of the laterality of hearing loss according to the laterality of tinnitus

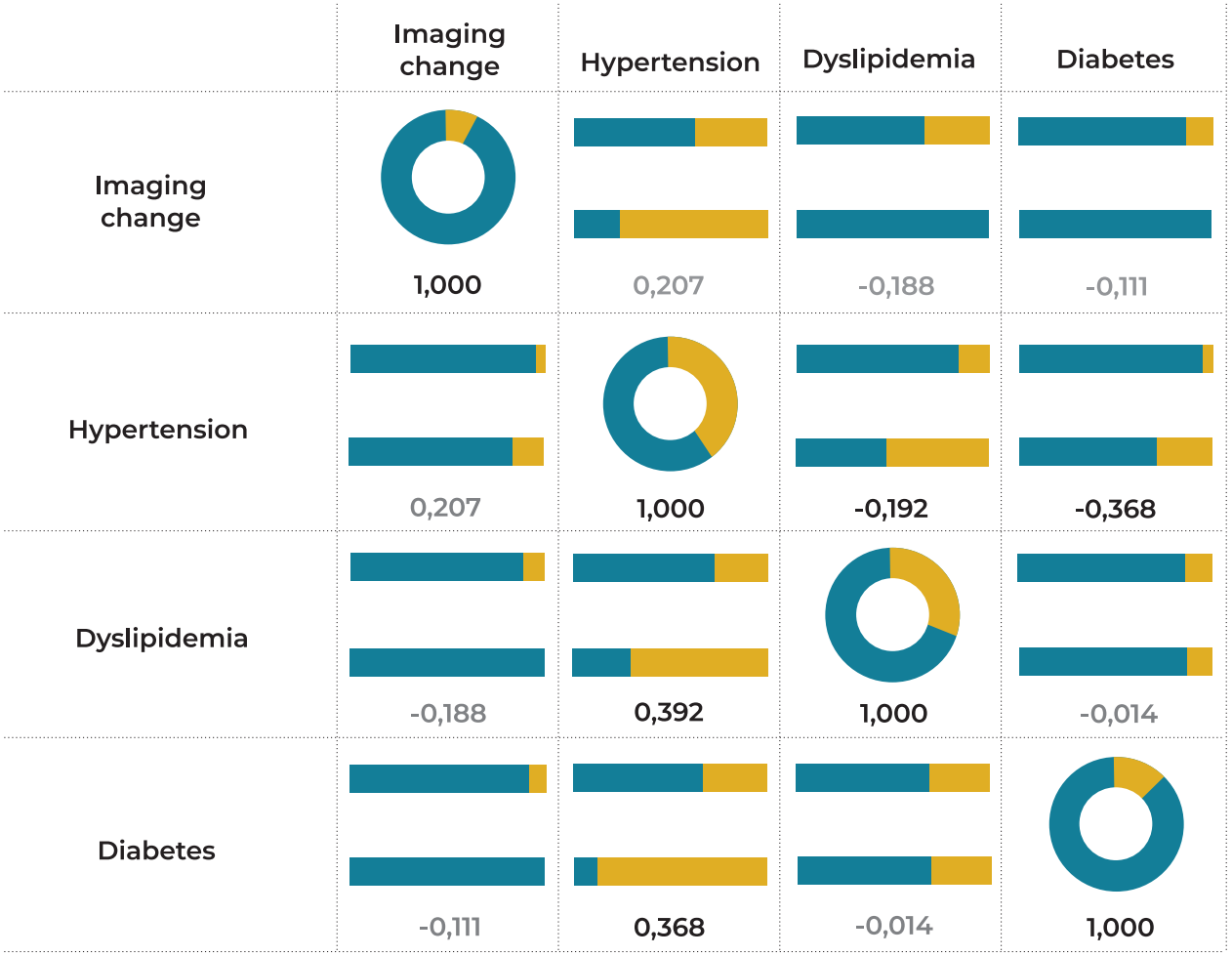


Among all patients, 21 (42%) had arterial hypertension, with no statistically significant difference in the prevalence between the group with imaging changes (75%) and the group without changes (36.7%) (p = 0.0132).
Fifteen (30%) patients had dyslipidemia, which had a prevalence of 32.7% in the group without imaging changes and 0% in the group with changes, without statistical significance (p = 0.171). Seven (13.2%) patients were diagnosed with diabetes, but the prevalence of diabetes did not show a statistically significant difference between the groups with and without imaging changes (14.3% and 0%, respectively, p = 0.417).
Figure 3 shows the correlations between imaging test changes and each of these risk factors.

Discussion

Doppler ultrasound is a noninvasive, safe, and easy-to-implement test to evaluate the superficial vascular structures. It is highly sensitive to carotid blood flow disorders[7], but rather limited in diagnosing other causes of pulsatile tinnitus. In our study population, most Doppler ultrasound results reported "no changes," and the most frequently detected changes without hemodynamic significance were atheromatous plaques (seven cases, 15.9%) and increased intima-media thickness (four cases, 9.1%). In contrast, CTA/MRA enabled the etiological diagnosis of four cases of pulsatile tinnitus.
The first case of pulsatile tinnitus was due to an AVF between the external carotid collaterals and the jugular bulb. An AVF is an anomalous direct connection between dural arteries and veins or between the dural arteries and a venous sinus, without a vascular nidus[8]. It is the most frequent cause of pulsatile tinnitus due to vascular lesions (2–20% of all cases of pulsatile tinnitus), accounting for 10–15% of all cerebrovascular malformations[9]. Our sample included a 74-year-old man diagnosed with AVF and moderate, bilateral, and symmetrical sensorineural hearing loss. This was associated with tinnitus at 500 Hz and 20 Db in the right ear, where a more audible carotid bruit was heard. His CTA report described "marked accessory artery vascularization from the external carotid artery in the right lateral cervical region." It may be difficult to diagnose an AVF by CTA/MRA because these tests usually detect only indirect signs, such as blood vessel dilation, cerebral edema, and (micro)bleeds.

Figure 3
Correlation matrix between imaging changes and cardiovascular risk factors. Values are tetrachoric correlation coefficients



They also have a limited capacity to assess the blood flow dynamics, with conventional angiography being the gold standard for detecting and assessing intracranial dural arteriovenous fistulas (IVAF)[10]. Therefore, the patient underwent conventional angiography, which showed "intense vascular blush appearing to be extracranial in the sub-mastoid and retroauricular region on the right, supplied by the ascending pharyngeal, posterior auricular, and occipital arteries, which are dilated. This high-output blush is almost fistulous and drains into the jugular vein (particularly into the jugular bulb)."

The second case of pulsatile tinnitus involved a neurovascular contact between the left AICA and cisternal segment of the ipsilateral acoustic-facial bundle, next to the acoustic pore. The patient reported bilateral tinnitus, more symptomatic on the left, and concomitant rotational vertigo. The audiogram revealed mild asymmetric sensorineural hearing loss (worse on the left). An initial carotid Doppler ultrasound showed no relevant changes. Neurovascular contact is a controversial etiology for pulsatile tinnitus[11]. The vessels most commonly involved in cases of contact with the vestibulocochlear nerve are the AICA, posterior inferior cerebellar artery (PICA), and basilar artery, typically presenting unilaterally[12]. Magnetic resonance imaging (MRI) is the preferred imaging modality for identifying neurovascular conflicts in patients with neuro-otological disorders, since it

enables the visualization of structures smaller than 1 mm in the cerebellopontine angle and internal acoustic meatus regions.

The third case was due to the recurrence of a right jugulotympanic paraganglioma after mastoidectomy in a patient experiencing right instability, tinnitus, and hearing loss. Paragangliomas (previously called glomus) are neuroendocrine cell tumors that originate from a paraganglion. They represent 0.6% of all head and neck neoplasms and affect the carotid and jugular glomus in 80% of cases. These tumors are frequently recurrent, but rarely malignant[13]. Paraganglioma is the most common tumor-related cause of pulsatile tinnitus. Computed tomography (CT) and MRI are useful tools for diagnosing this disease, with bone invasion being better evaluated on CT, which facilitates the classification of the tumor according to Fisch[14] and Glasscock-Jackson[15] and has therapeutic implications. MRI enables better assessment of the relationship between the tumor and neighboring vascular structures[16], typically showing a salt-and-pepper pattern, which is a mixture of hypo and hyperintense areas on T1- and T2-weighted sequences due to multiple areas without signal interspersed with hyperintense foci[17]. The fourth case was a right-sided pulsatile tinnitus caused by a temporal meningioma in a patient who had been experiencing tinnitus and associated hearing loss for two years. The audiogram was normal. Temporal meningioma is a rare cause of tinnitus that can mimic fibrous dysplasia. Depending on its location, it can spread to the middle ear or labyrinth[18]. MRI or CT are necessary for diagnosis in such cases[19].

This patient's CT report describes "an expansive lesion in the prepontine cistern on the right, over the petrous apex and with apparent extension to the cisternal segment of the trigeminal nerve and wall of the right cavernous sinus, suggestive of a meningioma." MRI confirmed these findings. The patient simultaneously underwent a Doppler ultrasound, which did not show significant changes.

Conclusion

The small sample size and retrospective design of this study hindered epidemiological inferences. Nevertheless, our data align with those in the literature, which recommend selecting an imaging test based on the pretest probability and the need to exclude rarer diseases that are clinically relevant. The etiological diagnosis of pulsatile tinnitus requires a thorough and focused clinical history. Although Doppler ultrasound has limitations, it can be useful in screening for vascular pathologies. The investigation should begin with a CTA or MRA if there is a high index of suspicion of skull base/temporal bone pathology[20], paragangliomas[21], or neurovascular conflict[22].

Conflict of Interests

The authors declare that they have no conflict of interest regarding this article.

Data Confidentiality

The authors declare that they followed the protocols of their work in publishing patient data.

Human and animal protection

The authors declare that the procedures followed are in accordance with the regulations established by the directors of the Commission for Clinical Research and Ethics and in accordance with the Declaration of Helsinki of the World Medical Association.

Privacy policy, informed consent and Ethics committee authorization

All the processed data were based in published reports that fulfilled privacy policy and ethical considerations.

Financial support

This work did not receive any grant contribution, funding or scholarship.

Scientific data availability

There are no publicly available datasets related to this work.

References

1. Conlin AE, Massoud E, Versnick E. Tinnitus: identifying the ominous causes. CMAJ. 2011 Dec 13;183(18):2125-8. doi: 10.1503/cmaj.091521.
2. Kircher ML, Leonetti JP, Marzo SM, Standring B. Neuroradiologic assessment of pulsatile tinnitus. Otolaryngol Head Neck Surg. 2008 Aug; 139 (S21). doi. org/10.1016/j.otohns.2008.05.380
3. Herraiz C, Aparicio JM. Diagnostic clues in pulsatile tinnitus (somatosounds). Acta Otorrinolaringol Esp. 2007 Nov;58(9):426-33.
4. Henry JA, Zaugg TL, Myers PJ, Kendall CJ, Michaelides EM. A triage guide for tinnitus. J Fam Pract. 2010 Jul;59(7):389-93.
5. Mattox DE, Hudgins P. Algorithm for evaluation of pulsatile tinnitus. Acta Otolaryngol. 2008 Apr;128(4):427-31. doi: 10.1080/00016480701840106.
6. Pegge SAH, Steens SCA, Kunst HPM, Meijer FJA. Pulsatile tinnitus: differential diagnosis and radiological work-up. Curr Radiol Rep. 2017;5(1):5. doi: 10.1007/s40134-017-0199-7.
7. Terzi S, Arslanoğlu S, Demiray U, Eren E, Cancuri O. Carotid doppler ultrasound evaluation in patients with pulsatile tinnitus. Indian J Otolaryngol Head Neck Surg. 2015 Mar;67(1):43-7. doi: 10.1007/s12070-014-0756-9.
8. An YH, Han S, Lee M, Rhee J, Kwon OK, Hwang G. et al. Dural arteriovenous fistula masquerading as pulsatile tinnitus: radiologic assessment and clinical implications. Sci Rep. 2016 Nov 4:6:36601. doi: 10.1038/srep36601.
9. Oh SJ, Chon YI, Kong SK, Goh EK. Multiple dural arteriovenous fistulas presenting as pulsatile tinnitus treated with external manual compression. J Audiol Otol. 2017 Sep;21(3):156-159. doi: 10.7874/jao.2017.00115.
10. Farb RI, Agid R, Willinsky RA, Johnstone DM, Terbrugge KG. Cranial dural arteriovenous fistula: diagnosis and classification with time-resolved MR angiography at 3T. AJNR Am J Neuroradiol. 2009 Sep;30(8):1546-51. doi: 10.3174/ajnr.A1646.
11. Peters TTA, van den Berge MJC, Free RH, van der Vliet AM, Knoppel H, van Dijk P. et al. The relation between tinnitus and a neurovascular conflict of the cochleovestibular nerve on magnetic esonance imaging. Otol Neurotol. 2020 Jan;41(1):e124-e131. doi: 10.1097/MAO.0000000000002432.
12. Serra A, Chiaramonte R, Viglianesi A, Messina M, Maiolino L, Pero G. et al. MRI Aspects of Neuro-Vascular Conflict of the VIII Nerve. Neuroradiol J. 2010 Dec;23(6):700-3. doi: 10.1177/197140091002300609
13. Rao AB, Koeller KK, Adair CF. From the archives of the AFIP. Paragangliomas of the head and neck: radiologic-pathologic correlation. Armed Forces Institute of Pathology. Radiographics. 1999 Nov-Dec;19(6):1605-32. doi: 10.1148/radiographics.19.6.g99no251605.
14. Fisch U. Infratemporal fossa approach to tumours of the temporal bone and base of the skull. J Laryngol Otol. 1978 Nov;92(11):949-67. doi: 10.1017/s0022215100086382.
15. Jackson CG, Glasscock ME 3rd, Harris PF. Glomus Tumors: Diagnosis, Classification, and Management of Large Lesions. Arch Otolaryngol. 1982 Jul;108(7):401-10. doi: 10.1001/archotol.1982.00790550005002.
16. Olsen WL, Dillon WP, Kelly WM, Norman D, Brant-Zawadzki M, Newton TH. MR imaging of paragangliomas. AJR Am J Roentgenol. 1987 Jan;148(1):201-4. doi: 10.2214/ajr.148.1.201.
17. Lee KY, Oh YW, Noh HJ, Lee YJ, Yong HS, Kang EY. et al. Extraadrenal paragangliomas of the body: imaging features. AJR Am J Roentgenol. 2006 Aug;187(2):492-504. doi: 10.2214/AJR.05.0370.
18. Hamilton BE, Salzman KL, Patel N, Wiggins RH 3rd, Macdonald AJ, Shelton C. et al. Imaging and clinical characteristics of temporal bone meningioma. AJNR Am J Neuroradiol. 2006 Nov-Dec;27(10):2204-9.
19. Ricciardiello F, Fattore L, Liguori ME, Oliva F, Luce A, Abate T. et al. Temporal bone meningioma involving the middle ear: A case report. Oncol Lett. 2015 Oct;10(4):2249-2252. doi: 10.3892/ol.2015.3516.
20. Bathla G, Hegde A, Nagpal P, Agarwal A. Imaging in pulsatile tinnitus: case based review. J Clin Imaging Sci. 2020 Dec 20:10:84. doi: 10.25259/JCIS_196_2020.
21. Thelen J, Bhatt AA. Multimodality imaging of paragangliomas of the head and neck. Insights Imaging. 2019 Mar 4;10(1):29. doi: 10.1186/s13244-019-0701-2.
22. Peters TTA, van den Berge MJC, Free RH, van der Vliet AM, Knoppel H, van Dijk P. et al. The relation between tinnitus and a neurovascular conflict of the cochleovestibular nerve on magnetic resonance imaging. Otol Neurotol. 2020 Jan;41(1):e124-e131. doi: 10.1097/MAO.0000000000002432.