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Vascular abnormalities – a true cause of pulsatile tinnitus?

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Abstract

Objective. Pulsatile tinnitus can be a symptom of vascular pathology. However, many of these pathologies present as incidental findings on scanning for unrelated symptoms. This study investigated whether the pathologies attributed to pulsatile tinnitus could instead be considered incidental findings.

Methods. This retrospective study evaluated imaging results of 272 pulsatile tinnitus cases for clinically relevant pathologies, and examined correlations between the site of symptoms and the imaging findings.

Results. Of 272 patients, 238 (88 per cent) had normal scans, 17 (6 per cent) had clinically insignificant incidental findings, and 18 (7 per cent) had findings requiring further investigation or intervention; regarding these latter 18 patients, findings for 8 patients (42 per cent) did not correlate with the symptomatic side. The rates of intracranial aneurysm and arteriovenous malformation in the pulsatile tinnitus group were comparable to those in normal populations.

Conclusion. The comparable rates of vascular abnormalities within the symptomatic pulsatile tinnitus group, plus clinically relevant findings contralateral to symptoms, suggest that vascular pathologies could be incidental findings rather than causes of pulsatile tinnitus. Evaluation is recommended of the effectiveness of the new National Institute for Health and Care Excellence guidelines for pulsatile tinnitus investigation.

Main Article

Mr H Walters takes responsibility for the integrity of the content of the paper

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Introduction

Tinnitus is the phantom perception of sound in the absence of any external environmental stimulus. It can be divided into pulsatile and non-pulsatile types. Prolonged tinnitus can have a considerable impact on patients' quality of life and has been linked to reduced mental wellbeing.[1]

Pulsatile tinnitus is an uncommon symptom, representing fewer than 10 per cent of tinnitus patients. It can be further characterised as synchronous or non-synchronous pulsatile tinnitus by checking its timing with vascular pulsations.[1]

Pulsatile tinnitus can be a presenting feature of a variety of underlying neuro-otological disease processes. The most commonly described causes are vascular abnormalities resulting in turbulent blood flow. These can be divided further into arterial and venous causes, such as aneurysms, arteriovenous malformations, arteriovenous fistulas, aberrant vessels and venous anomalies. Non-vascular pathologies include osseous causes and tumours. Vascular and non-vascular causes of synchronous pulsatile tinnitus are summarised in Table 1.[2,3]

Many conditions associated with tinnitus are benign; however, 'red flag' features that should not be ignored include acute onset of symptoms, pain and neurological involvement. These may be signs of carotid dissection or clinically relevant intracranial pathology, including stroke or aneurysm.[4]

A thorough patient history and bedside examination are vital, as these can direct the working diagnosis. Enquiry regarding the character, onset, severity and variations of the pulsatile tinnitus is recommended. The history should include a screen for associated symptoms such as headache, neck pain, vertigo, reduction in hearing and aural fullness. Exacerbating and alleviating factors, such as postural or exertional changes, should be noted. Past medical history may suggest risk factors such as cardiovascular disease, malignancy or previous head injury.

Otoscopy should be undertaken to assess for the presence of a retrotympanic mass. The presence of bruits on auscultation of the neck, scalp or eyes is suggestive of a vascular cause, as are the effects of vascular compression or Valsalva on the intensity of the tinnitus. Neurological signs should raise concerns of a potentially life-threatening pathology. Blood pressure recording and assessment for clinical signs of a hyperdynamic circulatory state such as anaemia, thyrotoxicosis or Paget's disease should be performed. Increased body habitus and optic disc changes on fundoscopy can be suggestive of intracranial hypertension. Audiometric assessment is also recommended.[5]

In March 2020, the National Institute for Health and Care Excellence (NICE) produced recommendations for imaging in pulsatile tinnitus cases in order to standardise practice, as there is no consensus on an optimal choice for imaging. The

Table 1. Causes of synchronous pulsatile tinnitus

Vascular causes				Non-vascular causes	
Arterial	Venous	Arteriovenous	Other	Osseous	Neoplastic
– Atherosclerosis or stenosis	– High-riding jugular bulb	– Arteriovenous fistula	– Hyperdynamic circulatory state	– Paget's disease	– Paraganglioma (glomus)
– Carotid artery dissection	– Dural venous sinus anomalies	– Dural arteriovenous fistula	– Idiopathic intracranial hypertension or pseudotumour cerebri	– Otosclerosis	– Intracranial neoplasm
– Carotid artery aneurysm		– Arteriovenous malformation		– Cavernous haemangioma	
– Aberrant internal carotid artery					

recommendations were based upon expert knowledge, as no evidence to support a particular method of imaging was identified by the committee. Magnetic resonance angiography is the modality of choice, and computed tomography (CT) angiography is the suggested second-line investigation.[6] These investigations do not always uncover evidence of an underlying cause, but can reveal incidental findings of varying significance.[7]

We used retrospective data from 272 patients with symptoms of pulsatile tinnitus who had been investigated prior to the introduction of the NICE guidance, and evaluated the findings. As the recommendations had not been published at the time of data collection, the choice of imaging modality does not follow the new NICE guidelines.

Aims and objectives

These were: (1) to establish whether the radiological evidence of vascular structural abnormalities is attributable to pulsatile tinnitus or if it is more suggestive of an incidental finding, by comparing the location of the abnormality with the laterality of the symptom; (2) to assess the proportion of pulsatile tinnitus patients with clinically relevant findings when imaged; and (3) to establish whether the rate of vascular abnormalities in pulsatile tinnitus patients is similar to the rate found in the asymptomatic, 'normal' population. A similar rate of vascular malformations in the pulsatile tinnitus group when compared with an asymptomatic sample of patients would infer that the vascular malformations are not associated with the tinnitus.

Materials and methods

Patients who had undergone radiological imaging after presenting with hearing loss, tinnitus or vestibular symptoms were identified from Sunderland Royal Hospital records using the Meditech electronic database. The database search was then narrowed, searching by indication to identify scans performed for the investigation of pulsatile tinnitus. The search dates were from 28 May 2013 to 31 December 2018. The date range captures the period from the first date of the electronic records to the end of 2018, providing just over five years of data.

The inclusion criteria were: any patient presenting with pulsatile tinnitus, either unilateral or bilateral; patients with other associated audiovestibular symptoms; and imaging results (i.e. magnetic resonance imaging (MRI) of the internal auditory meatus (IAM), MRA, CT angiography and Doppler ultrasound). All patients aged under 18 years were excluded.

Data from the resulting 272 subjects were collected from the Meditech electronic case note system and picture archiving and communication system, focusing upon presenting features, laterality of symptoms and imaging results.

Imaging results were grouped into the following categories: normal or incidental finding that is not clinically relevant; clinically relevant finding related to pulsatile tinnitus but not requiring intervention; and clinically relevant finding requiring intervention. These groupings were subdivided into findings ipsilateral and contralateral to the patient's pulsatile tinnitus. A similar rate of vascular malformations in the pulsatile tinnitus group when compared with an asymptomatic sample of patients would infer that the vascular malformations may not be associated with the tinnitus.

There is no imaging database for asymptomatic patients; therefore, there is no control group. In order to draw comparison with an asymptomatic, 'normal' population, the rate of vascular abnormalities in the pulsatile tinnitus group has been evaluated against the known prevalence of vascular abnormalities in asymptomatic patients, taken from the rates found in observational studies of normal populations.

Results

Of 272 patients investigated for pulsatile tinnitus, 259 presented with unilateral pulsatile tinnitus; the remainder had bilateral symptoms. A total of 238 patients (88 per cent) had normal scans, 17 (6 per cent) had clinically insignificant or incidental findings, and 1 patient had both a clinically insignificant incidental finding and a clinically relevant finding (a clinically relevant anterior communicating artery aneurysm and a clinically insignificant high-riding jugular bulb). Hence, 18 patients (7 per cent) had clinically relevant findings requiring further investigation or intervention (Figure 1).

Clinically insignificant or incidental findings included: normal anatomical variants ($n = 2$), benign neoplasm ($n = 1$), high or dehiscent jugular bulbs ($n = 10$), middle-ear effusion ($n = 1$), clinically insignificant arterial stenosis ($n = 2$), and partial venous thrombosis ($n = 1$). Within the clinically insignificant findings group, 2 out of 17 patients (12 per cent) had findings contralateral to the site of symptoms: mild external carotid artery stenosis ($n = 1$) and middle-ear effusion ($n = 1$) (Figure 2).

Clinically relevant findings requiring further investigation or intervention included: pituitary macroadenoma ($n = 1$), jugulotympanic paraganglioma ($n = 2$), malignant neoplasm

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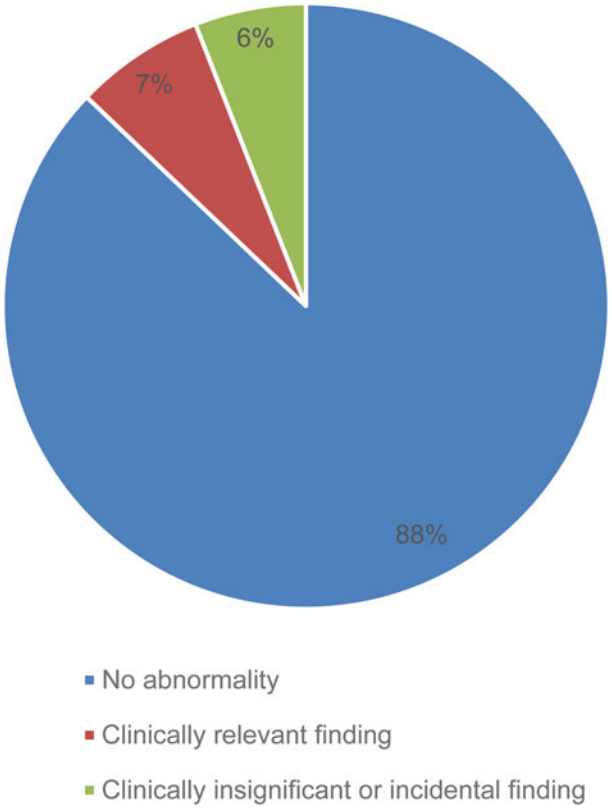

Fig. 1. Rates of abnormalities. (Note that the values exceed 100 following rounding up of the numbers.)

($n = 2$), intracranial aneurysm ($n = 6$), extracranial aneurysm ($n = 1$), arteriovenous malformation ($n = 2$), vestibular schwannoma ($n = 2$) and severe internal carotid artery stenosis ($n = 2$) (Figure 3). One patient was found to have both a clinically relevant anterior communicating artery aneurysm and the clinically insignificant incidental finding of a high-riding jugular bulb.

Of the clinically relevant pathologies, 8 out of 18 (42 per cent) were contralateral to the site of the pulsatile tinnitus, or were midline or bilateral findings in patients with unilateral pulsatile tinnitus, including: glomus tympanicum ($n = 1$), pituitary macroadenoma ($n = 1$), anterior communicating artery aneurysm ($n = 3$), bilateral carotid stenosis ($n = 2$) and vestibular schwannoma ($n = 1$).

Discussion

Rate of incidental findings

Pulsatile tinnitus is thought to have a higher tendency than non-pulsatile tinnitus to have an underlying cause relating to a vascular structural abnormality; however, when one reviews neurosurgical practice, intracranial aneurysms and arteriovenous malformations rarely present with tinnitus.[8,9]

Comparison with normal incidence

In our patient group, the rate of arteriovenous malformations was 2 out of 272 (0.74 per cent) and the rate of aneurysms was 6 out of 272 (2.2 per cent). In the general population, the prevalence of arteriovenous malformations is 18 per 100 000 (0.02 per cent). The reported rate of unruptured intracranial aneurysms ranges from 0.2 per cent to 10 per cent, and the yearly incidence of spontaneous aneurysmal subarachnoid haemorrhages is 6–7 per 100 000.[10,11] Within our patient group, who are symptomatic with pulsatile tinnitus, the rate of intracranial aneurysms has been found to be similar to the rates reported in studies of the normal population. Conversely, the rate of arteriovenous malformations in our group was shown to be higher when compared with studies of the normal population.

Our sample size of 272 patients is relatively small; therefore, we recognise that the reliability of the rates of aneurysms and arteriovenous malformations within the pulsatile tinnitus population is limited. In addition, the reliability of epidemiological data for the prevalence of arteriovenous malformations is questionable in view of their rarity and the unknown prevalence in asymptomatic patients.

We accept that it is difficult to draw any strong conclusions from a direct comparison with epidemiological studies of the normal population because of confounding variables such as demographics, co-morbidities and geographical location.

Symptoms on opposite side to pathology

In our patient group, 18 out of 272 (7 per cent) had clinically relevant abnormal findings, yet 8 of these patients had clinically relevant findings known to cause pulsatile tinnitus either on the contralateral side to their symptoms or in the midline (glomus tympanicum ($n = 1$), carotid stenosis ($n = 2$), pituitary macroadenoma ($n = 1$), anterior communicating artery aneurysm ($n = 3$) and vestibular schwannoma ($n = 1$)).

Of note, a vestibular schwannoma was detected, yet vestibular schwannoma does not commonly present with pulsatile tinnitus. The tumour was also contralateral to the side of the pulsatile tinnitus and is therefore suspected to be an incidental finding.

These findings are comparable to the rates of abnormalities in other contemporary studies, such as Connor, who identified abnormalities in 5–10 per cent of scans.[7]

Other authors investigating pulsatile tinnitus have reported the rate of abnormal anatomical findings on imaging to be 44–91 per cent. This range is high in comparison with our own finding, whereby 13 per cent of patients had pulsatile tinnitus with associated abnormal radiological findings. It is possible that this difference in findings is the result of a higher proportion of patients being referred and imaged for pulsatile tinnitus in modern clinical practice, resulting in a lower diagnostic yield.[7]

Significance of abnormal findings

Recent papers suggest that some structural abnormalities previously attributed to pulsatile tinnitus may not be the underlying cause and are false positive findings, for example vascular loops. Several studies have assessed patients with vascular loops in the IAM and found that their laterality, their site and the severity of compression on IAM structures did not have a statistically significant influence on the presence or severity of pulsatile tinnitus.[12] In our group, five patients with pulsatile tinnitus were found to have vascular loops associated with IAM structures. Within this group, two patients had bilateral loops but unilateral symptoms, one had findings contralateral to the site of symptoms, and two patients had loops ipsilateral to their pulsatile tinnitus.

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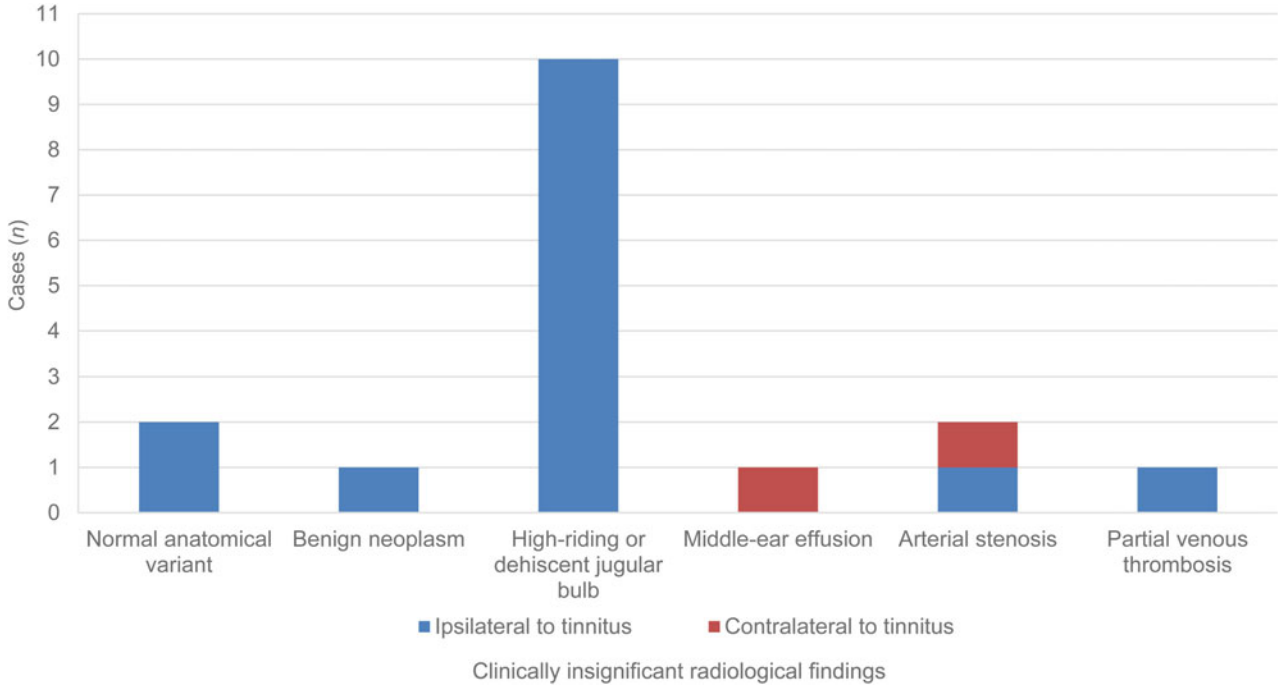

Fig. 2. Clinically insignificant radiological findings.

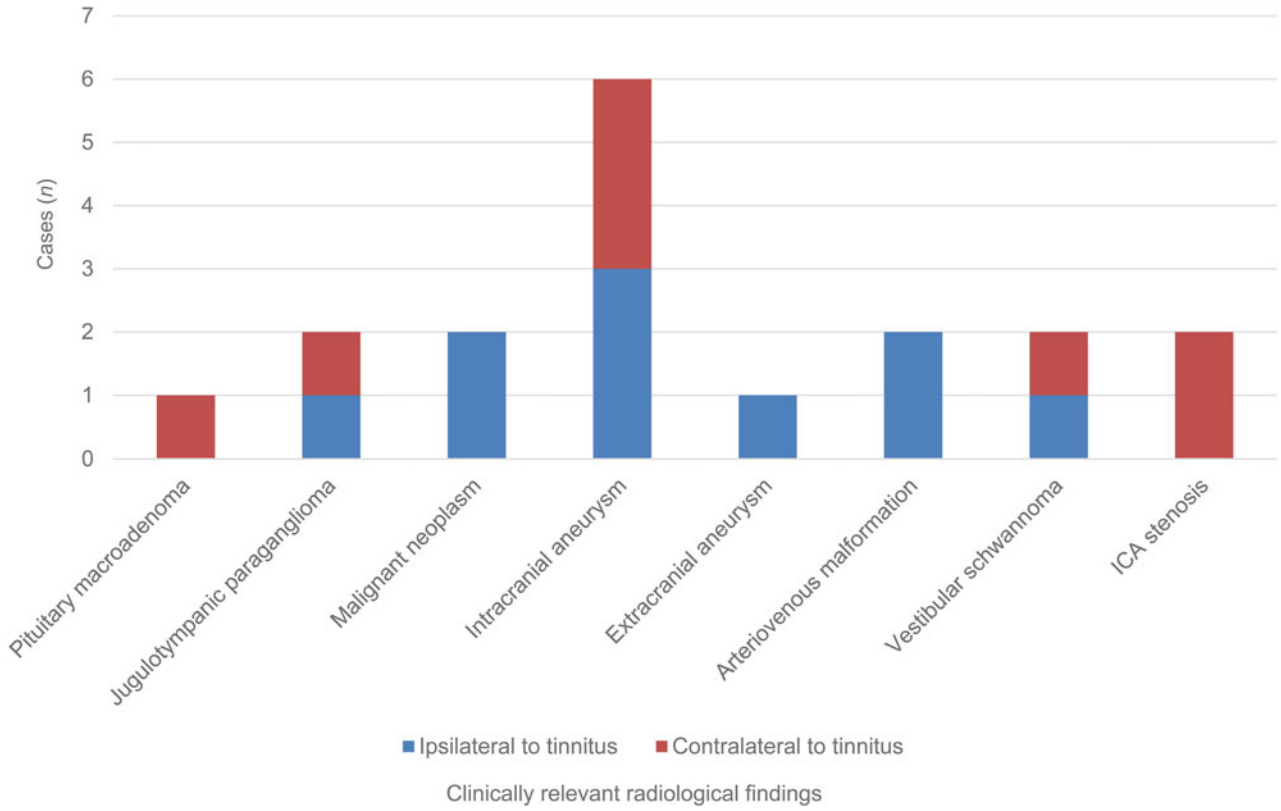

Fig. 3. Clinically relevant radiological findings. ICA = internal carotid artery

Inconsistent laterality of findings

Within our group of patients with abnormal scan findings, 6 out of 37 abnormalities (16 per cent) were contralateral to the site of the symptom. Other studies also comment on abnormal findings contralateral to the symptomatic side. One theory that may explain this phenomenon is a compensatory increase in flow within contralateral vessels as a result of vascular compression by an abnormality. There is also the possibility that the sound caused by the abnormality is conducted to the contralateral side; this may be perceived more obviously because of an ipsilateral hearing loss.[13] There is a lack of strong evidence to support these theories, and it is possible that the contralateral findings are in fact incidental, especially given that our rate of serious vascular abnormalities such as aneurysms in the symptomatic group is comparable to that reported in studies of the normal population.

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Grierson *et al*. provide a valuable review of the topic. They argue that isolated pulsatile tinnitus is unlikely to be associated with a relevant radiological abnormality. Instead, they state that clinically relevant pulsatile tinnitus has other associated features that can be revealed by a thorough history and examination. In their case series of 50 patients with vascular pulsatile tinnitus, only 1 patient had a positive scan finding despite having no documented obvious red flags for vascular pulsatile tinnitus; however, they state that the physical examination had been incomplete, as a vascular compression test had not been conducted. The patient's scan revealed a laterally placed, tortuous sigmoid sinus. If vascular compression had been included in the examination, it could have increased suspicion for a venous abnormality.[14] They contend that a thorough assessment could be used to screen for those patients who are most at risk of having a serious underlying pathology.

- Most patients investigated for pulsatile tinnitus have normal scan findings
- Rates of intracranial aneurysm and arteriovenous malformation in pulsatile tinnitus are comparable to those in a normal population
- Symptoms commonly thought to cause pulsatile tinnitus often do not correlate with the laterality of the symptom
- Vascular pathologies could be incidental findings, rather than the underlying cause of pulsatile tinnitus
- Audit of the new protocol for imaging in pulsatile tinnitus cases is recommended to assess its value in identifying causative pathology

Study limitations

Because of the retrospective nature of our study, it was not always possible to discern the completeness of the history and physical examination from the notes. This information could have produced further data to identify a patient group with a higher yield of diagnostic scans.

We encountered variable imaging techniques using either MRI or CT angiography to investigate pulsatile tinnitus, reducing the reproducibility of the results.

This study is also limited by the lack of a normal, asymptomatic control group to directly compare with, as this was a retrospective review of patients investigated for pulsatile tinnitus. A direct comparison of our results with the normal population is not a reliable measure, but it does question the value of imaging every patient presenting with pulsatile tinnitus.

Conclusion

The rate of intracranial aneurysms within this group of symptomatic patients who all presented with pulsatile tinnitus is comparable to the rate reported in the normal population. Clinically relevant abnormalities have also been demonstrated on the contralateral side to the patients' symptoms. We propose that vascular pathologies could be incidental findings rather than the underlying cause of pulsatile tinnitus; however, we accept that a firm conclusion cannot be drawn from comparing findings obtained from a small sample of patients with results of epidemiological studies of the normal population.

Further research is required in order to stratify an investigation protocol to improve the rate of positively correlating clinical findings in the pulsatile tinnitus population.

The NICE recently produced a recommendation to provide imaging (magnetic resonance angiography in the first instance) to evaluate pulsatile tinnitus. We recommend an audit of this protocol after a period of time to assess its value in identifying causative pathology.

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Competing interests. None declared

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