Section: Miscellaneous

Case Report

TINNITUS: A RARE PRESENTATION IN A LACUNAR STROKE

Pranathi Chanti[1], Nivetha M[2], Abdul Malik[3], Priya Mahato[4], Shweta N. Dandekar[5], Anusha S. Aladahalli[6], Dishakkshi Singh[6], Vishal Venugopal[7]

[1]Osmania Medical College, Hyderabad, India
[2]Stanley Medical College, ChennaI, India
[3]Shadan Institution of Medical Sciences, Hyderabad, India
[4]GMERS Medical College Gotri, Vadodara, India
[5]Rajiv Gandhi Medical College and Chhatrapati Shivaji Maharaj Hospital, Mumbai, India
[6]JJM Medical College, Davangere, India
[7]Bhaarath Medical College & Hospital, Chennai, India

Received : 09/04/2023
Received in revised form : 25/05/2023
Accepted : 08/06/2023

Keywords:
Endotracheal tube, Alkalized lidocaine, Cuff-related complications.

Corresponding Author:
Dr. C. Sridevi,
Email: t.sridevi32@gmail.com

DOI: 10.47009/jamp.2023.5.3.344

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2023; 5 (3); 1722-1726

Abstract

Lacunar stroke results when a single penetrating artery is occluded and accounts for one-fourth of cerebral strokes. It typically affects deep structures of the brain involved in motor control, sensation, and coordination. Despite their classical effects, they can sporadically damage other regions as well, such as auditory pathways. Tinnitus, an unusual ringing in the ears, is more frequently linked to impairments in the auditory circuits in the brain. Nonetheless, tinnitus is a rare presentation of lacunar stroke. There haven't been many descriptions of it in the literature. We describe a case of chronic lacunar infarct in a 47-year-old male presenting with tinnitus. Patient has undergone evaluation and started on medications with regular two-month follow-ups. The motive of this report is to create awareness that stroke patients can present with tinnitus and assess tinnitus as an independent risk factor for stroke.

INTRODUCTION

Tinnitus refers to the subjective perception of sound within the vicinity of the cranial region in the absence of any discernible external acoustic stimuli. It is a prevalent condition worldwide, affecting around 15-20% of the population. However, in 1–3% of cases, it can significantly impact an individual's quality of life.[1] Various risk factors have been found to be associated with tinnitus, such as obesity, smoking, alcohol consumption, previous head injuries, hypertension, sleep disturbances, and certain medications. Research has also found a correlation between tinnitus and the use of diuretics as well as low systolic blood pressure in patients receiving antihypertensive therapy. Patients with severe cases of tinnitus may experience sleep problems, anxiety, and depression. There is a possibility of shared pathophysiological mechanisms between tinnitus and ischemic cerebrovascular disease (ICVD), such as arterial stiffening. Arterial stiffening can affect the microcirculation of the cochlea, leading to impairment. Research has found a significant association between a higher common carotid artery stiffness index and the severity of tinnitus and.[2,3] Additionally, arterial stiffness is linked to a higher incidence of stroke. The American Academy of Otolaryngology-Head and Neck Surgery Foundation (AAO-HNSF) has recognized the significant impact of tinnitus on individuals and has recently released its first-ever comprehensive clinical guidelines for managing patients with tinnitus.[4] These guidelines are evidence-based and provide a framework for clinicians to follow when assessing and treating patients with persistent tinnitus. The guidelines include recommendations for diagnostic workup as well as suggested treatment options, including education and counseling. Despite numerous proposed causes and complex pathogenic mechanisms, there is currently no definitive treatment available for tinnitus. There are no drugs approved by the Food and Drug Administration (FDA) or European Medicines Agency (EMEA) for tinnitus, and none have been found in the literature to provide a replicable, long-term reduction of tinnitus impact beyond placebo effects. The current approach to treatment involves managing underlying conditions and symptomatically suppressing the perception of tinnitus.[5] The primary objective of treatment is to improve quality of life rather than achieve a complete cure. A variety of treatment regimens are available, but none have shown complete efficacy.[6] The global prevalence of tinnitus has been found to be 14.4% in adults, and the highest prevalence was noted in South America. The pooled prevalence of severe tinnitus among

ISSN (O): 2687-5365; ISSN (P): 2753-6556

adults was 2.3%. There is widespread inconsistency in defining and reporting tinnitus, leading to variability in prevalence estimates among studies. The most common definition of tinnitus used while measuring prevalence was tinnitus lasting for more than 5 minutes.[7,8]

CASE PRESENTATION

A 47-year-old male came to the hospital with chief complaints of ringing in both ears for 4 months. The ringing sensation was insidious in onset, persistent throughout the day, and present equally in both ears. It was associated with headaches and sleep disturbances. The ringing was low-pitched, and the patient describes the loudness as being 7 on a scale of 10. The quality of the sound was pure tone. The patient used calming music and earplugs to mask these sounds throughout the course of the day. The patient reported an intermittent headache that was unilateral, of a pulsating type, and not associated with an aura. The patient complained that he was unable to sleep at night or during the morning hours because of a ringing sensation, due to which he was able to sleep only 4 hours on average. The symptom was not associated with hearing change, was not exacerbated by loud noise, and was not associated with pain with exposure to loud noise. There was no history of exposure to loud noise, previous episodes of ringing sensation, trauma to the head and neck, otalgia, otorrhea, vertigo, dizziness, nystagmus, diplopia, or changes in gait. There was no history suggestive of stroke or stroke-like episodes in the past. The ringing was not synchronous with the heartbeat or with respiration. There was no jaw pain, grinding of teeth during sleep, or clicking sensation in the jaw. There was no history of psychiatric admissions or medications in the past. There was no history of similar complaints in the family. The patient does not report the presence of any significant stressors prior to the symptom onset, but the current symptomatology is causing him stress as well as an inability to attend to his work appropriately. The patient was not on drugs known to cause ototoxicity or on any illicit drugs. Significant medical history includes treatment for diabetes mellitus since 12 years and hypertension since 1 year, both well controlled with medication as evident by regular follow-up. The patient is also taking medications for headaches and insomnia. On physical examination, the patient was a healthy male with a normal BMI. The patient was awake, alert, and oriented to time, place, and person. Cranial nerve examination was found to be normal, motor and sensory system examination was unremarkable. Reflexes and cerebellar examination were normal, and fundoscopic examination showed no abnormalities. Cardiovascular, respiratory, and musculoskeletal examinations were all within normal limits. Ear examination showed a normal external ear, an external auditory canal with some earwax, and a normal tympanic membrane. Rinne's test was performed, and the air conduction was greater than bone conduction. Weber's test was performed, and it was lateralized to both ears equally. An audiogram was performed, and it was normal according to age. Relevant investigations were performed: a plain CT scan of the brain showed symmetrical periventricular ischemia in bilateral frontal lobes; the rest of the cerebral parenchyma was normal and showed normal gray and white matter differentiation; the fourth ventricle was normal; the cerebellum and brainstem were normal; and there was evidence of a shift in midline structures. An MRI of the brain showed a chronic lacunar infarct and a prominent perivascular space in the left lentiform nucleus, along with mild changes of chronic ischemia in cerebral white matter. The posterior fossa structures were normal, the ventricular system was normal, the extracerebral spaces were normal, and no shift of midline structures was seen. An MR angiogram (Figure 3) and an MR venogram (Figure 4) were performed and were found to be normal. ECG, echocardiography, and color doppler were normal.

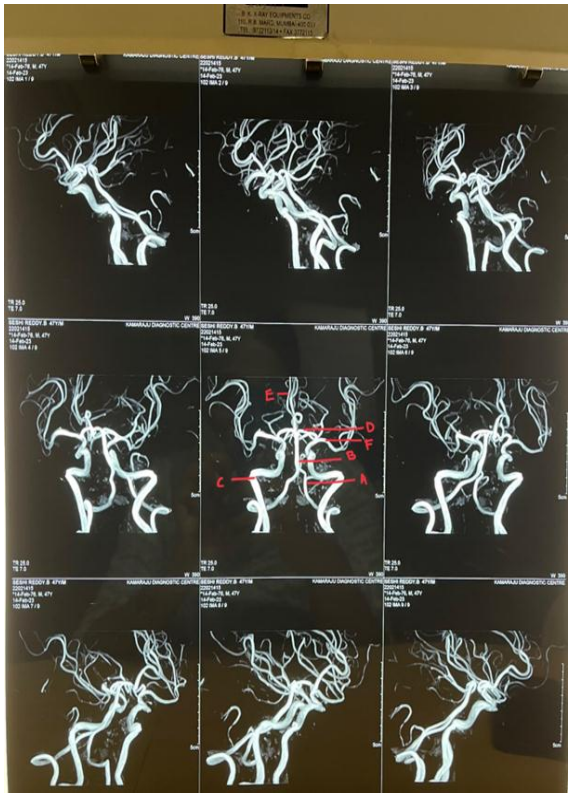

Figure 1: MR Angiography of Circle of Willis showed Internal carotid arteries, Middle cerebral arteries, Anterior cerebral arteries, Vertebral arteries, basilar arteries and posterior cerebral arteries are patent, there is no arteriovenous malformations or aneurysms present.

A- Left Vertebral Artery
B-Basilar Artery
C-Right Internal Carotid Artery
D-Left Posterior Cerebral Artery

ISSN (O): 2687-5365; ISSN (P): 2753-6556

E-Right Anterior Cerebral Artery
F-Left Middle Cerebral Artery

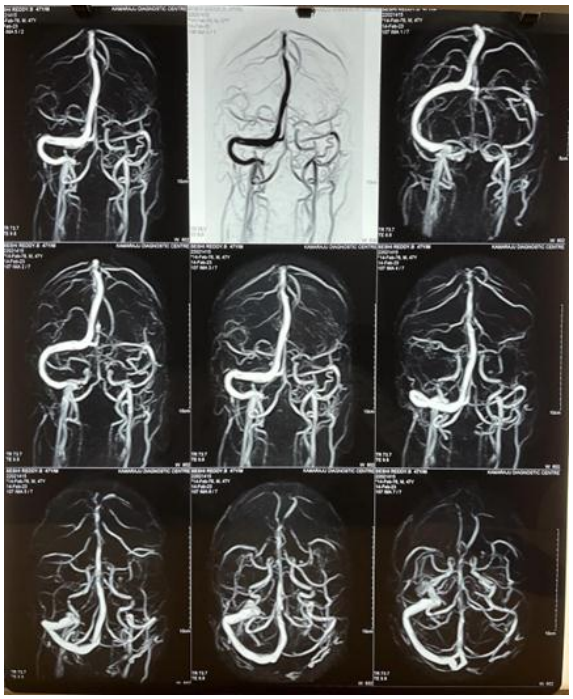

Figure 2: MR venogram was performed and it showed superior sagittal,straight, transverse and sigmoid sinuses are patent. Superior sagittal sinus and straight sinus are draining into the right transverse sinus. Deep cerebral veins are patent.

DISCUSSION

The pathophysiology of tinnitus is currently an issue that is poorly understood and receives very little scientific attention. However, the most common risk factors for tinnitus include prolonged exposure to sound, an injury to the head and neck, or an infection. The most recent hypotheses have moved toward a more central approach to tinnitus, in which the central nervous system (CNS) is regarded as a cause or generator of the condition, with uncontrolled neuronal feedback loops from the auditory brainstem and thalamus to the primary auditory cortex and vice versa.[1] Since the vertebrobasilar circulation is primarily responsible for inner ear symptoms such as vertigo and hearing loss, a stroke in this particular circulatory system could theoretically cause tinnitus in these patients.

However, in our particular case, a 47-year-old man presented with a buzzing sensation that had been going on for four months after an episode of stroke. While this is a fairly unusual symptom, it has been previously recorded in the literature. In our particular case, we have reason to believe that this is not the cause, despite the fact that, as stated before, we consider this to be one of the primary causes of tinnitus.[9]

Previous studies have identified numerous risk factors for tinnitus. These risk factors include but are not limited to: 1) older age (>60 years); 2) male sex; 3) arterial hypertension; 4) lacunar type of stroke; 5) presence of multiple ischemic foci; 6) presence of bilateral ischemic foci; 7) changes in blood cell count; and 8) disturbances of agglutination. A lacunar infarct, the existence of multiple ischemic foci, hypertension, and the fact that the patient was of male sex all contribute to this particular instance having a higher probability of acquiring tinnitus.[10]

When compared to vestibular symptoms, a stroke that affects a patient's ability to hear is extremely uncommon in the vast majority of patients, as was indicated in the previous research.[11] This could be due to multiple factors, one of which is that the vestibular nervous system is more widespread than the auditory system and hence more likely to be affected by a stroke.[12] In cases of hearing loss, lesions tend to be bilateral; consequently, these patients frequently exhibit severe stroke symptoms that prevent them from leading a normal life. The auditory pathway is frequently spared by the most common types of strokes as the major parts of the auditory pathway, such as the cochlear nucleus, inferior colliculus, and medial geniculate body, have multiple sources of blood supply. Another factor is that the auditory system is well represented in the CNS system and has a strong bilateral representation above the level of the cochlear nuclei.[13] Tinnitus has been identified as a novel risk factor for stroke, despite the fact that our patient experienced stroke before developing tinnitus. Those with tinnitus between the ages of 20 and 40 have an increased risk of ischemic cerebrovascular disease. Patients older than 40 have no increased risk of developing a stroke. Since our patient is over the age of 40, we can infer that his tinnitus is unlikely to play a role in a subsequent episode of stroke; therefore, treating it actively may not provide much benefit in preventing future ICVD.[14] Thiazide diuretics are the most common antihypertensives that are known to produce tinnitus; nevertheless, calcium channel blockers, ace inhibitors, and potassium sparing diuretics have also been associated with increased tinnitus.[15] Although the pathophysiology of both is linked, there has been no proof regarding the amelioration of tinnitus with stroke medications. As in the case of our patient, he had been on Tab Ecosporin AV 150 for three months and has shown no signs of improvement with his tinnitus. According to a study, low-dose gabapentin has been shown to alleviate tinnitus in a stroke patient. Low-dose gabapentin was administered for a pre-existing postherpetic thoracic neuralgia, but the tinnitus improved markedly and returned after drug discontinuation. The hearing function was unaltered. The patient's results demonstrated that a subgroup of tinnitus patients with secondary contributing factors, such as stroke, could benefit from low-dose gabapentin.[16]

ISSN (O): 2687-5365; ISSN (P): 2753-6556

The following therapeutic approaches have been shown to improve the prognosis of patients suffering from tinnitus:

Cognitive-behavioral therapy (CBT) is a type of psychotherapy that uses techniques like cognitive restructuring and relaxation to modify a patient's tinnitus-related thoughts and behaviors. Patients are encouraged to maintain a journal and complete "homework" in order to develop coping skills.[17]

Tinnitus retraining therapy (TRT) is based on the idea that abnormal neuronal activity in the auditory system is responsible for tinnitus. The goal is to habituate the auditory system to the tinnitus signals, making them less noticeable or bothersome. TRT involves individual counseling to educate patients about the auditory system, the causes of tinnitus, and the benefits of TRT.[18]

Sound therapy is also an integral part of TRT.

Masking involves the use of wearable devices similar to hearing aids that emit low-level white noise, such as a high-pitched hiss, to reduce the perception of tinnitus. It can also produce residual inhibition, which is a temporary reduction in the perception of tinnitus that continues for a short time after the masker is turned off.[19]

Biofeedback and stress management techniques can be beneficial for such patients. Tinnitus can be stressful, and stress can make tinnitus worse. Biofeedback is a relaxation technique that allows patients to gain control over their stress by changing their physiological responses. Electrodes placed on the skin send information about bodily processes such as pulse rate, skin temperature, and muscle tension to a computer, which displays the data on a monitor. Patients learn to modify these processes by changing their thoughts and emotions, leading to a reduction in the body's stress response.[20]

There are currently no FDA-approved medications for the treatment of tinnitus, and no drug, supplement, or herb has been shown to be more effective than a placebo in clinical trials. This includes ginkgo biloba, which was marketed for this purpose on occasion. While some patients may believe that acupuncture is beneficial for tinnitus,[21] others may disagree. In our case, the patient was put on CBT twice a week for the last month and has exhibited modest improvement. Although the results of the same over a longer period of time have not yet been determined. Masking was advised, but the patient declined to go with it further due to a lack of resources.

Even though in our case the tinnitus developed after the stroke, tinnitus has been proven to frequently be a predictor of stroke. Patients have been observed to have hearing loss and tinnitus 1-2 days before an episode of stroke, and this may prove to be a useful indicator for stroke.[22]

CONCLUSION

A comprehensive literature review reveals a consistent association between tinnitus and chronic lacunar stroke. Tinnitus in a lacunar stroke is a relatively rare manifestation that has a considerable impact on the patient's quality of life. Studies examining the temporal relationship between tinnitus and chronic lacunar infarction indicate that the two conditions may occur at separate times, with tinnitus occurring either before or after the stroke. It can result from unregulated neural feedback between the auditory brainstem, thalamus, and primary auditory cortex. The findings of this report imply that tinnitus may be a risk factor for stroke on its own, unlike other well-known risk factors like high blood pressure, diabetes, and smoking. As a result, it should be looked at as a potential warning sign and a significant risk factor for lacunar stroke. Tinnitus is substantially alleviated when the underlying condition causing it is correctly diagnosed and treated.

REFERENCES

1. Atik A. Pathophysiology and treatment of tinnitus: an elusive disease. Indian J Otolaryngol Head Neck Surg. 2014 Jan;66(Suppl 1):1-5. doi: 10.1007/s12070-011-0374-8. Epub 2011 Dec 15. PMID: 24533352; PMCID: PMC3918281.
2. Bayraktar C, Taşolar S: Relationship between increased carotid artery stiffness and idiopathic subjective tinnitus. Eur Arch Otorhinolaryngol. 2017;274(5): 2125-30. 10.1007/s00405-016-4440-4. . [PubMed] [Google Scholar]:10.1007/s00405-016-4440-4
3. van Sloten TT, Sedaghat S, Laurent S, et al.: Carotid stiffness is associated with incident stroke: a systematic review and individual participant data meta-analysis. J Am Coll Cardiol. 2015:2116-25. 10.1016/j.jacc.2015.08.888
4. Tunkel DE, Bauer CA, Sun GH, et al.: Clinical Practice Guideline: Tinnitus. Otolaryngology-Head and Neck Surgery. 2014, 151:1-40. 10.1177/0194599814545325
5. Dobie R (2004: In: Snow J (ed) Tinnitus: theory and management. Decker, Hamilton, 10.3766/jaaa.18.3.7
6. Dobie RA: A review of randomized clinical trials in tinnitus. Laryngoscope. 19991098, 1202:1211. 10.1097/00005537-199908000-00004
7. Jarach CM, Lugo A, Scala M, et al.: Global Prevalence and Incidence of Tinnitus: A Systematic Review and Meta-analysis. JAMA Neurol. 2022, 1:888-900. 10.1001/jamaneurol.2022.2189
8. McCormack A, Edmondson-Jones M, Somerset S, Hall D: A systematic review of the reporting of tinnitus prevalence and severity. Hear Res. 2016, 337:70-9. 10.1016/j.heares.2016.05.009
9. Kim JS, Lee H: Inner ear dysfunction due to vertebrobasilar ischemic stroke. Semin Neurol. 2009, 29:534-40. 10.1055/s-0029-1241037
10. Przewoźny T, Gasecki D, Narozny W, Nyka W: Risk factors of sensorineural hearing loss in patients with ischemic stroke. Otol Neurotol. 2008, 29:745-50. 10.1097/MAO.0b013e318181336c
11. Bamiou DE. Hearing disorders in stroke. Handb Clin Neurol. 2015;129:633-47. doi: 10.1016/B978-0-444-62630-1.00035-4. PMID: 25726294.
12. Kim HA, Lee H. Recent Advances in Understanding Audiovestibular Loss of a Vascular Cause. J Stroke. 2017 Jan;19(1):61-66. doi: 10.5853/jos.2016.00857. Epub 2016 Dec 12. PMID: 28030893; PMCID: PMC5307938.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

13. Häusler R, Levine RA: Auditory dysfunction in stroke. Acta Otolaryngol. 2000, 120:689-703. 10.1080/000164800750000207
14. Huang YS, Koo M, Chen JC, Hwang JH: The association between tinnitus and the risk of ischemic cerebrovascular disease in young and middle-aged patients: A secondary case-control analysis of a nationwide, population-based health claims database. PLoS One. 2017, 2:0187474. 10.1371/journal.pone.0187474
15. Figueiredo RR, Azevedo AA, Penido NO: Positive Association between Tinnitus and Arterial Hypertension. Front Neurol. 2016, 5:171-10. 10.3389/fneur.2016.00171
16. Chen WH, Yin HL: Successful amelioration of tinnitus in a stroke patient by low-dose gabapentin. J Stroke Cerebrovasc Dis. 2012, 21:907-1. 10.1016/j.jstrokecerebrovasdis.2011.07.014
17. Fuller T, Cima R, Langguth B, Mazurek B, Vlaeyen JW, Hoare DJ. Cognitive behavioural therapy for tinnitus. Cochrane Database Syst Rev. 2020 Jan 8;1(1):CD012614. doi: 10.1002/14651858.CD012614.pub2. PMID: 31912887; PMCID: PMC6956618.
18. Han BI, Lee HW, Kim TY, Lim JS, Shin KS. Tinnitus: characteristics, causes, mechanisms, and treatments. J Clin Neurol. 2009 Mar;5(1):11-9. doi: 10.3988/jcn.2009.5.1.11. Epub 2009 Mar 31. PMID: 19513328; PMCID: PMC2686891.
19. Hobson J, Chisholm E, El Refaie A. Sound therapy (masking) in the management of tinnitus in adults. Cochrane Database Syst Rev. 2012 Nov 14;11(11):CD006371. doi: 10.1002/14651858.CD006371.pub3. PMID: 23152235; PMCID: PMC7390392.
20. Carmen R, Svihovec D. Relaxation-biofeedback in the treatment of tinnitus. Am J Otol. 1984 Jul;5(5):376-81. PMID: 6383065.
21. Bhatt JM, Lin HW, Bhattacharyya N: Prevalence, Severity, Exposures, and Treatment Patterns of Tinnitus in the United States. JAMA Otolaryngol Head Neck Surg. 2016, 1:959-965. 10.1001/jamaoto.2016.1700
22. Lee H, Sohn SI, Jung DK, et al.: Sudden deafness and anterior inferior cerebellar artery infarction. Stroke. 2002, 33:2807-12. 10.1161/01.str.0000038692.17290.24

ISSN (O): 2687-5365; ISSN (P): 2753-6556