



Case Report

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Vestibular Paroxysmia Associated with Recurrent Episodes of Pulsatile Tinnitus: A Case Report and Review of the Literature

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Abstract

Vestibular paroxysmia is defined as paroxysms of vertigo that could be presented with paroxysmal pulsatile tinnitus. It is usually caused by neurovascular compression of the vestibulocochlear nerve. The condition is responsive to carbamazepine. Refractory cases are treated with microvascular decompression. We present a case of vestibular paroxysmia of 27 years old patient that was her main presentation is tinnitus in addition to vertigo. She was treated with microvascular decompression (MVD).

Keywords

Vestibular paroxysmia, Tinnitus, Pulsatile, Vascular compression, Vestibulo-cochlear nerve compression

Introduction

Vestibulocochlear nerve compression could present as episodic attacks of vertigo or pulsatile tinnitus caused by vascular compression of the vestibulocochlear nerve [1,2]. The pulsatile nature of the episodes is caused by the nature of the vascular compression [3]. Vestibular Paroxysmia is a rare disease, believed to be the cause of 4% of all dizziness conditions. The anterior inferior cerebellar artery (AICA) is thought to be the cause of 75% of vestibular paroxysmia cases.

Although seen less frequently, other blood vessels have been reported as a cause of this rare entity including: posterior inferior cerebellar artery (PICA) in 5% of the cases, the vertebral artery with 10% occurrence and the veins in 10% of the cases [4,5].

Fast sodium channel blockers such as Carbamazepine and Oxcarbazepine could be the main treatment in these patients, as they prove their efficacy in decreasing attacks' frequency. In addition, they decrease the intensity of the attack [3].

The surgical treatment should be offered in the refractory cases, or those who has side effects related to the drugs [6].

Herein, we report a rare case of a 27-year-old female patient who presented with pulsatile tinnitus, diagnosed as a case of vestibular paroxysmia, and treated with microvascular surgical decompression.

Case Report

A 27-year-old female presented to our clinic with a 14 years history of episodic pulsatile tinnitus. The tinnitus was left sided, that is continuous, increases in quite ambient, with stress and upon waking up in the morning. It decreases with the use of headphones and with compressing the left side of the neck. Upon further questioning, the patient reports 6 episodes of vertigo over the last 3 years. Each episode lasted 2 minutes, it was not related to any certain provocation. The patient did not report headaches and does not have a family history of migraine. There is no history of seizures, or family history of epilepsy. The patient had been evaluated multiple times by ENT specialists. She had undergone different investigations with no apparent diagnosis.

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In neurological examination, the patient was conscious, oriented, and alert. Cranial nerves examination was intact with no apparent abnormality. She had normal motor, sensory and cerebellar examination. The patient had negative Dix-Hallpike test, Romberg test, Hoffman's test, and Babinski sign.

Brain magnetic resonance imaging (MRI) was performed and showed left aberrant anterior inferior cerebellar artery, compressing the left vestibule-cochlear nerve and the left facial nerve.

(Figure 1) and (Figure 2) preoperative MRI showing aberrant AICA artery compressing the vestibulocochlear and the facial nerve.

For this reason and based on the above findings the patient underwent left retro-mastoid, retro-sigmoid craniotomy for microvascular decompression of the AICA was done (Figure 3) and (Figure 4). No intraoperative complications have been reported. The patient was doing well after the surgery, with intact cranial nerves, no motor or sensory deficits. The tinnitus significantly improved in the first day, and this proves that aberrant AICA was the cause of her symptoms.

The patient was discharged, after 4 days of surgery, on Carbamazepine, dexamethasone, analgesics and PPI.

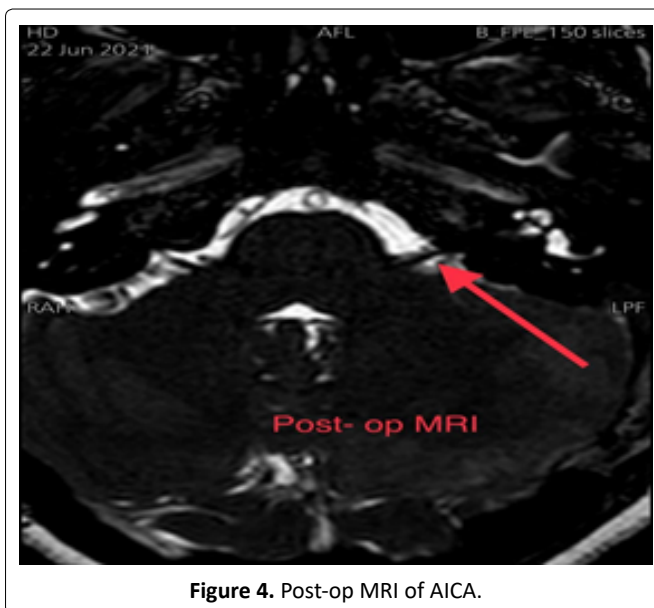


Figure 4. Post-op MRI of AICA.

On follow up, the tinnitus improved with no reported complications after the surgery.

Discussion

The patient presented with paroxysms of tinnitus and vertigo. Based on negative Dix-Hillpike maneuver and the results of MRA, benign positional vertigo and vertebra-basilar insufficiency were excluded. Clinical picture was useful to exclude Meniere's disease and perilymphatic fistula. Due to the absence of headaches vestibular migraine were not taken into consideration. Our patient follows the criteria for probable diagnosis of vestibular paroxysmia, as she had 6 episodes of vertigo, lasting two minutes, not provoked and not contributed to any other diagnosis [7]. But this case was special, as the main complaint of the patient was related to tinnitus, and vertigo was not even mentioned by the patient, until we have asked her thoroughly about it. The presentation of tinnitus made it more difficult to investigate and to interpret.

Pulsatile tinnitus has a range of differential diagnosis, including glomus tympanicum, dural AV fistulas and carotid artery abnormalities, all of them were excluded based on the imaging findings and clinical presentation [8].

The subjective nature of tinnitus makes it difficult to interpret. It was evident in the past years that tinnitus could be caused by neurovascular compression of the 8th cranial nerve, or arachnoiditis at the cerebellopontine angle [9,10]. The literature argues about the possibility of tinnitus with compression, due its vague nature. This was also evident in the fact that only 44-70% of patients responded to MVD [11-14].

It is worthy to note that tinnitus presentation is rare in VIII compression, and until the moment of publishing this case report, there are no criteria related to tinnitus as a presentation [15]. Prior studies have suggested different presentations of the tinnitus associated with the compression, as some described it to be pulsatile in nature, and others report continuous tinnitus. It is evident, that tinnitus could present both with pulsatile or continuous nature [16-20].

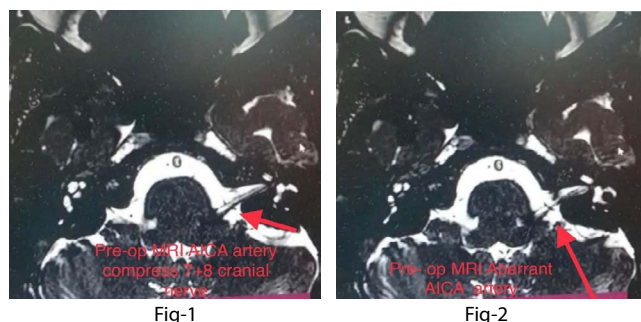


Figure 1 and 2: Preoperative MRI showing aberrant AICA artery compressing the vestibulocochlear and the facial nerve.

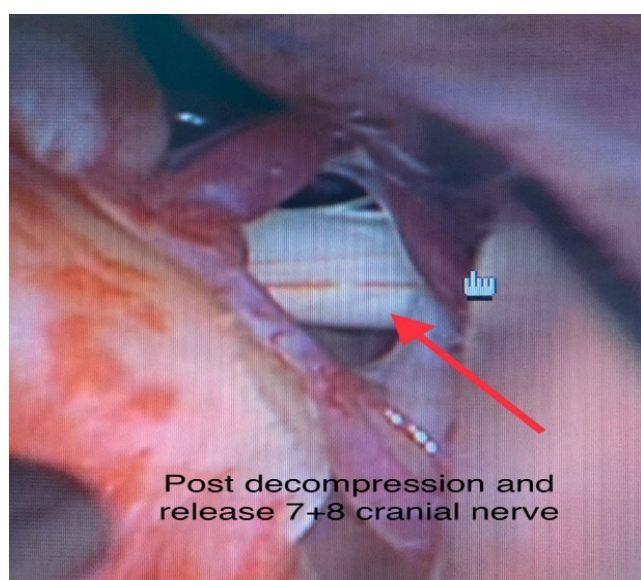


Figure 3. Intraoperative view of decompression and release

Neurovascular compression usually affects the intracisternal part of the nerve, with a range of 0-10.2 mm distance from the internal acoustic porus [16,21]. This part is vulnerable to compression due its covering; this is the site of transition from central myelin to Schwann cell. The vestibulocochlear nerve is the largest nerve to have the central myelin portion [22].

The radiological evidence of neurovascular compression is considered sensitive but not specific feature of the condition [23].

Microvascular decompression is a well-established for multiple conditions like trigeminal neuralgia. Yet it the role of decompression in vestibulocochlear nerve is still controversial. The surgical decompression success rate to relive tinnitus has low percentage. This is explained due to subjective nature of the presentation of tinnitus and the complexity of the surgery. Moreover, it has been postulated that the compression changes centrally located nuclei, and that would not be relieved with surgery. The complexity of the surgery has few reasons, the vascular compression implied on the cochlear nerve could be found along the whole nerve. Furthermore, there is always a risk of injury to the AICA during mobilization [24,25].

Multiple factors have been elucidated to predict the outcome of MVD. Young females, presentation unilateral tinnitus of short duration and those with unaffected hearing would have better outcomes after the surgery [25-29].

Literature Review

Bibliography research is made with PubMed using the keywords "Vestibular paroxysmia". The research result was 93 articles. We included in this literature review 29 articles. Studies were published from 1994 to 2021.

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