Case Report

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Exceptional Continuous Tinnitus in a Vascular Loop Syndrome of the VIIIth Cranial Nerve

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Abstract

Background: A case of tinnitus possibly due to vascular loop compression of VIIIth nerve is presented.

Case presntation: A33-years old female suffers from tinnitus beginning 4 years ago. An extensive clinical and laboratory investigation did not reveal any abnormal finding except MRI signs of a vascular loop pressure of VIIIth nerve.

Discussion: The medication did not help the patient and she refused to undergo surgery. We present the arguments of the causal relationship of the findings on MRI and symptoms of the patient.

Conclusion: Tinnitus due to vascular pressure is a syndrome which should be considered in differential diagnosis in case of tinnitus.

Keywords: Tinnitus; Vascular loop syndrome; VIIIth Cranial nerve; Anterior inferior cerebellar artery (AICA).

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Background

Vascular loops have already been known as a cause of Vth, VIIth and VIIIth cranial nerve syndromes mainly in a manner of pulsatile or intermitted symptoms (i.e. trigeminal neuralgia) [1,2]. In 1984 Jannetta et al. introduced the term-disabling positional vertigo due to vascular loop pressure [3]. There are no specific features of the syndrome and because of that is not a condition acceptable to all researches [2,4].

In most cases described in the literature the syndrome consists of symptoms related to the vestibular and cochlear nerve [4].

We present a case of patient suffer from tinnitus and MRI signs of vascular loop pressure of VIIIth nerve without other symptoms of cochleovestibular function impairment.

Case Presentation

A 33 -year- old woman presented with unilateral, left ear tinnitus beginning four years ago. From her medical history she suffered from arterial hypertension treated with atenolol on the grounds of polycystic kidney disease. She also suffered from irritable bowel syndrome and had undergone surgery for ankle joint osteochondritis. Tinnitus was described as continuous during the day, worsening in quiet places or after stress or fatigue and was felt by the patient as ''whistling'' or ''waterflow''.

Neurological examination revealed only a left- beating horizontal nystagmus. Romberg and Unterberger signs were negative and no unsteadiness or posture disorder was noticed. Videonystagmography showed a spontaneous left-beating horizontal nystagmus, while caloric irrigation did not reveal any unilateral weakness or directional preponderance. The Vestibular Evoked Myogenic Potential test was also normal. Brain MRI showed a distinct vascular loop of the anterior inferior cerebellar artery (AICA) around the VIII cranial nerve, which was in contact to it within the cerebellopontine angle (CPA), outside the internal auditory canal (IAC) (**Figure 1**).

In a previous case series study, AICA vascular loops were more frequent within the CPA than inside the IAC, although the latter seem to account for auditory symptoms more often [3].

The question is if there is causative relation between the tinnitus and the vascular loop in our patient. As many authors have shown before, cranial nerve compression from vascular loop was also

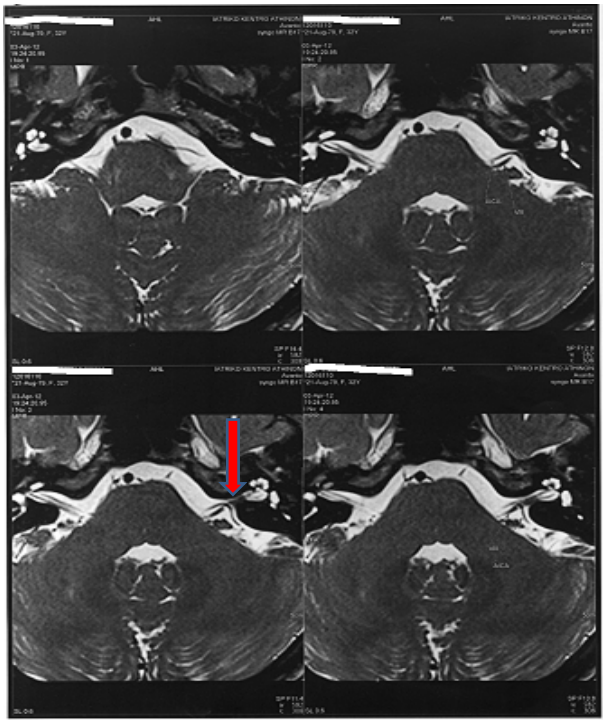

Figure 1 Axial MRI shows a vascular loop from the AICA (horizontal arrow) in contact to the VIII cranial nerve (vertical arrow) within the cerebellopontine angle and outside the internal acoustic channel .

incidentally noted in asymptomatic patients when brain MRI was performed for other reasons [5]. In addition, some researchers found that there is no clear relationship or correlation between tinnitus and the presence of a loop [6-8].

The only evidence of a causative relation is to improve the patient after surgical decompression.

Due to the minor severity and mild impact of her tinnitus in her everyday life, our patient refused to undergo surgery. As far as we know surgery is not generally suggested except in severe cases.

According to the above, in our case we have only some indirect causative suggestions in order to support our hypothesis.

The first of them comes from the fact that no other cause of the patient symptoms was identified after an extensive clinical and laboratory -including audiological -investigation as well as three year follow up.

The second of them is the fact that the tinnitus in our patient remains constant and not fatigable. Characteristics of other disorders affecting the VIII nerve such as fluctuating hearing loss are not present [9].

A third suggestion comes from treatment efforts.

The underline mechanism of the syndrome remains unclear. Subsequently a rational treatment cannot be recommended [4].

Symptoms in a loop syndrome comes from hyperactivity and hyperexcitability of the nerve due to the pressure by a vascular sector. The characteristic of loop syndrome is the positive sensory phenomena, spontaneous or evoked, due to a focal peripheral nerve injury [10].

The mechanism of production of the symptoms seems to be the generation of ectopic impulses and non-synaptic ephaptic transmission of impulses. Histopathologic evidence, however, does not support the main and causative role of demyelinization in loop syndromes involving the eighth nerve [11].

Theories implicating the mechanism of central sensitization or inflammatory process have also been suggested in literature [12].

Voltage-gated sodium (VGSC) and potassium channels play a pivotal role in action potential generation and conduction. Drugs that influence the sodium channel could benefit patient with vascular loop syndrome. Actually the most effective drug in trigeminal neuralgia is carbamazepine a VGSC blocker [13].

In case of our patient the treatment effort included administration of carbamazepine, pregabaline, amitryptiline (75 mg daily), baclofene (tolerated 15 mg daily) and benzodiazepines. The more effective drug was carbamazepine but the patient experienced mild sedation in clinical effective doses (>400 mg CR).

Finally the patient refused further medical treatment. She remains untreated taking only occasionally alprazolam which offers temporary relief by reducing the severity of tinnitus.

In our case the effectiveness of the carbamazepine could strengthen the point of view that a possible mechanism of ectopic impulse underlies.

Summarizing and relying on the above we can say that we have a series of clues that could partly support the claim that the symptoms of our patient could be attributed to the existence of a vascular loop.

As far as the prognosis of the vascular loop syndrome concerns there are no studies to deal with.

From a clinical point of view hypertension and ageing accelerate atherosclerotic changes and could result the worsening of the symptoms [14]. However, the exact cause and effect of hypertension has not been clarified, as an ectatic vessel compressing the brainstem may result in hypertension [15].

On this basis a worsening of symptoms with age would be expected. However since the natural history of the syndrome is unknown, it cannot be ruled out any change even a spontaneous remission.

Conclusion

Tinnitus due to vascular pressure is a syndrome which should be considered in differential diagnosis in every case of tinnitus even if there are not specific features of the syndrome.

Footnotes

There are no funding interests from any of the authors.

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