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SUDDEN SENSORINEURAL HEARING LOSS ASSOCIATED WITH THE PRESENCE OF A VASCULAR LOOP IN THE INTERNAL AUDITORY CANAL. CASE REPORT

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Abstract: *The vascular loop refers to a trajectory abnormality of the antero-inferior cerebellar artery or its branch in the internal auditory canal. The Chavda radiological classification identifies three types of trajectory depending on the depth of penetration of the loop at the level of the internal auditory canal. The article presents the case of a 26-year-old patient admitted to the ENT department for left ear sudden sensorineural hearing loss and dizziness. The 3D Fiesta-C axial sequence MRI shows the presence of the vascular loop inside the internal auditory canal, without exceeding half of it. The hearing loss had an unfavourable evolution, without recovery after treatment with steroids and vasodilators. Sudden sensorineural hearing loss may be a consequence of the presence of the vascular loop in the internal auditory canal. The prognosis for recovery from hearing loss is poor if the loop is at least type II.*

INTRODUCTION

The arterial vascularization of the inner ear comes from the labyrinthine artery or the artery of the internal auditory canal and is the terminal branch of the vertebrobasilar vascular system. In postmortem anatomical studies, this artery comes from the anteroinferior cerebellar artery (AICA), a branch of the basilar artery in 83% of cases, occasionally it can detach directly from the basilar artery in 14 % of cases or rarely, from the posteroinferior cerebellar artery (PICA).(1,2) In 80% of cases, the internal auditory artery is unique but there can be two arteries in 20% of cases. The common cochlear artery, a branch of the labyrinthine artery, follows the trajectory of the cochlear nerve at the level of the internal auditory canal and gives two branches at the level of the cochlea, the main cochlear artery and the cochleovestibular artery.

The vascular loop is a pathway abnormality of the anteroinferior cerebellar artery (AICA) or its branch in the internal auditory canal. From a radiological point of view, there are three types of this vascular loop, according to the Chavda classification: type I in which the AICA loop is at the edge of the meatus of the internal auditory canal, type II in which the loop insinuates in the internal auditory canal but does not occupy more than 50% of its length and type III in which the loop is located in the internal auditory canal and occupies more than 50% of its length.

MRI imaging studies use the Fiesta-C sequence in 3D mode of acquisition, with fine slices (of maximum 1 mm), for better visualization of the vascular and nerve elements in the internal auditory canal and for obtaining additional details of the contact or conflict between the blood vessel and the nerve, but also of the nerve angulation at the point of contact with the blood vessel, all of these being specific signs of vascular compression.(3) The use of equipment with strong 3T magnet offers a higher sensitivity and specificity of the method, due to the higher SNR (signal to noise ratio) which is to the benefit of visualizing small anatomical structures at stronger magnetic fields. Imaging results can be supplemented by the results of auditory brainstem response with click stimuli (click ABR), which show a prolonged I-III interval consistent with auditory neural dysfunction caused by either the contact or compression of cranial nerve by the blood vessel.

If a vascular loop is present, the auditory and vestibular symptoms are variable, and the patient can present with pulsating tinnitus, progressive sensorineural hearing loss, fluctuating hearing, sudden hearing loss, short-term recurrent paroxysmal vertigo or recurrent positional vertigo, resistant to repositioning maneuvers. The cochlear blood flow can be disturbed by the altered labyrinthine circulation. The self-regulatory circulatory system of cerebral vessels (i.e. basilar artery) becomes inefficient and may induce hypoxia which is often related to irreversible lesions of the anterior and posterior labyrinths.(4)

Regarding the treatment of the symptomatic vascular loop, there is no defined protocol. Well-documented cases of pulsating tinnitus or disabling positional vertigo may undergo vascular decompression, unlike cases of sudden sensorineural hearing loss in which this treatment option is controversial.

CASE REPORT

We present the case of a 26-year-old male patient who addressed the ENT emergency facility for a left ear sudden sensorineural hearing loss, accompanied by dizziness.

The pure tone audiometry revealed a profound sensorineural hearing loss in the left ear (figure no. 1) with an average of the auditory thresholds in air conduction (at 500, 1000 and 2000 Hz) of 93 dB HL. The acoustic impedance showed a bilateral type A tympanogram, with an absent stapedial reflex in the left ear in ipsilateral stimulation and

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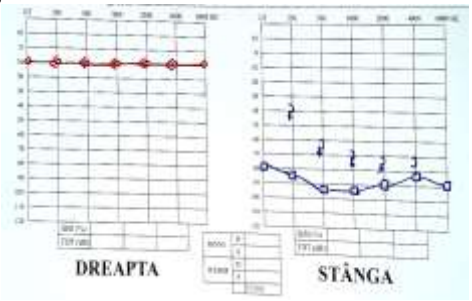
present in contralateral stimulation.

Figure no. 1. Pure tone audiogram: profound sensorineural hearing loss in the left ear



The electrophysiological examination using click ABR does not allow the measurement of the I-III interval because of profound hearing loss on high frequencies domain. During the vestibular clinical exam we noticed the presence of spontaneous horizontal rotatory right nystagmus in the right gaze. The Fukuda stepping test was abnormal, with the patient's rotation on the left side more than 35º.

The MRI imaging performed with 1,5 Tesla unit with specific 3D Fiesta-C axial sequence for the inner ear and internal auditory canal, with axial slices of 1 mm thickness, identified on the left side a Chavda II vascular loop which extends in the internal auditory canal less than 50 % of its length (yellow arrow, figure no. 2). There is also more cerebrospinal fluid in the left pontocerebellar cistern and the internal auditory canal.

Figure no. 2. Axial 3D Fiesta-C sequence MRI: vascular loop inside the left internal ear canal (yellow arrow)

During the hospitalization the patient received steroids and vasodilators for 7 days, but the audiometric evaluation did not show any improvement of his auditory thresholds. The evolution of the left peripheral vestibular syndrome was good and static compensation of the vestibulo-ocular reflex was noticed before the patient's discharge. The hearing assessment one month after the onset of the symptoms showed unchanged auditory thresholds in the left ear. Given the absence of usable hearing on the left side and the loss of stereo audition, the patient chose to wear a frequency modulated (FM) cross routing of signal system, with the microphone in the left ear and the receiver in the right ear.

DISCUSSIONS

The etiopathogenesis of sudden sensorineural hearing loss can be found in up to 10% of cases.(5,6) In the case of the vascular loop at the level of the internal auditory canal, the specified mechanism is the disturbance of the local blood flow with the development of low pressures at the cochlear circulatory system. The deeper the vascular loop, the lower the chances of recovering from hearing loss.(7)

Although there are some clinical imaging studies (8) that aimed to establish a causal link between the vascular loop and auditory and vestibular symptoms and the results do not seem to show a statistically significant correlation, these data should be interpreted with caution because they do not include a lifelong surveillance of asymptomatic patients with vascular trajectory abnormality, identified by imaging.

Regarding the case presented in this article, the sudden sensorineural hearing loss may be caused by the presence of the vascular loop in the left internal auditory canal, by inducing a disturbance of blood flow in the anterior and posterior labyrinths, also favoured by the presence of a larger volume of cerebrospinal fluid in the left pontocerebellar cistern and in the left internal auditory canal, which exerts greater pressure on the vasculonervous structures.(9)

The surgical treatment consisting of microvascular decompression has no indication in this case because it cannot restore the hearing and the patient has no complaints of pulsatile tinnitus.

CONCLUSIONS

The vascular loop is a risk factor for sudden sensorineural hearing loss. The prognosis of hearing recovery is unfavourable for those of type II and III of the Chavda classification. The surgical treatment consisting of microvascular decompression has indications in well-evaluated situations in which a potential benefit regarding the symptomatology can be obtained.

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