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Case Report

The anterior inferior cerebellar artery abutment: an uninvited guest in sudden sensorineural hearing loss

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ABSTRACT

Sudden sensorineural hearing loss (SSNHL) is an otological emergency and prompt recognition and management has been shown to improve hearing outcomes and quality of life. A 49-year-old female presented to the ENT OPD with complaints of sudden onset decreased hearing in the right ear since 3 days. Pure tone audiometry done in an outside hospital on day 2 of patient's complaints showed right ear to have pure tone average of 90 dB (profound hearing loss) and left ear to have pure tone average of 21.66 dB (within normal limits). At presentation to the ENT OPD on day 3 of complaints, otoscopic examination revealed normal tympanic membrane. MRI inner ear and brain (plain and contrast) showed the right anterior inferior cerebellar artery to be abutting the posterior aspect of vestibulocochlear nerve. Patient received 2 doses of intratympanic injection of Dexamethasone followed by a course of oral steroids for 15 days. Serial repeat pure tone audiometry showed marked improvement of symptoms. Although most cases of SSNHL are idiopathic, a number of treatable conditions do exist. The relationship between vascular loops around the vestibulocochlear nerve and sudden sensorineural hearing loss have to be kept in mind while evaluating these patients. Prognosis for hearing recovery is based on several factors, including time since onset, degree of deafness, age, and vertigo. Although SSNHL will often spontaneously improve without treatment, directed therapy against treatable causes along with steroid therapy can aid in the improvement of symptoms.

Keywords: Otological emergency, Sensorineural hearing loss, Anterior inferior cerebellar artery abutment

INTRODUCTION

Sudden sensorineural hearing loss (SSNHL) is an otological emergency for which a definitive aetiology and treatment remains controversial, but prompt recognition and management have been shown to improve hearing outcomes and quality of life.[1] There is a wide age distribution with an average of 50-60 years and no sex preference.[1] Several factors have been postulated as central to the aetiology of idiopathic sudden sensorineural hearing loss (ISSNHL). The aetiology is identified in less than 5-10% of cases.[1] Possible causes include labyrinthine viral infection, vascular insult, intracochlear membrane rupture and autoimmune inner-ear disease. Vascular loops in the cerebellopontine angle may interfere with Cranial Nerve VIII and cause otological symptoms, such as vertigo, tinnitus and/or sensorineural hearing loss.[2] One such vascular loop, that is the anterior inferior cerebellar artery (AICA), which was seen in a patient with sudden sensorineural hearing loss, will be discussed in this case report. The prevalence of AICA loops within the internal auditory canal has been reported to be between 13-40% in post-mortem dissections and 14-34% by magnetic resonance imaging (MRI).[3] Steroid therapy is widely used as the standard treatment for SSNHL.[1] The most frequently administered intratympanic steroids are dexamethasone and solumedrol (methyl prednisolone sodium succinate).[1]

CASE REPORT

A 49-year-old female presented to the ENT OPD with complaints of decreased hearing in the right ear since 3 days which was sudden in onset, gradually progressive with decreased hearing to normal conversations as well. There was no history of ear trauma, ear pain or ear discharge. Pure tone audiometry done in a local hospital on day 2 of patient's complaints showed right ear to have pure tone average of 90 dB (profound hearing loss) and left ear to have pure tone average of 21.66 dB (within normal limits) (Figure 1).

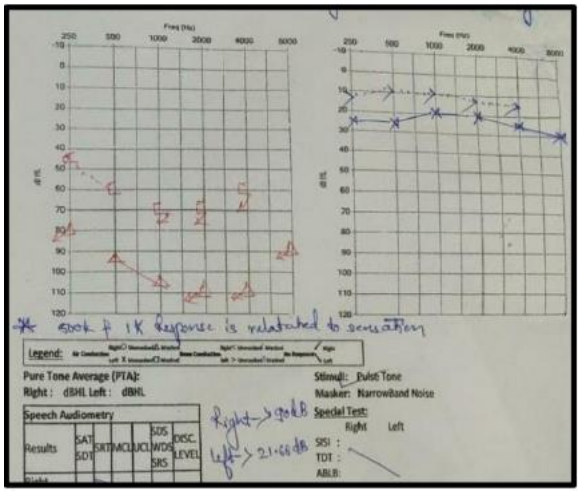

Figure 1: PTA on day 2 of complaints.

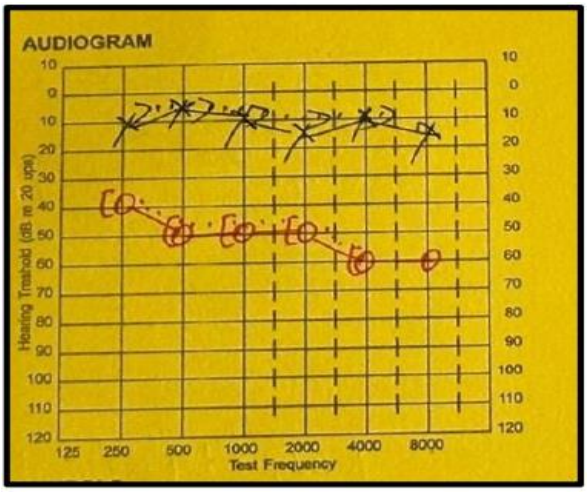

Figure 2: PTA after intratympanic steroid.

At presentation to the ENT OPD on day 3 of complaints, otoscopic examination showed a normal tympanic membrane on both sides. Intratympanic injection of dexamethasone 4 mg was given in the right ear under local anaesthesia on day 3 and 5 of patient's complaints. She was simultaneously started on tablet prednisolone 20 mg, thrice a day for 3 days, which was tapered and stopped over a period of the next 12 days. Serial repeat pure tone audiometry suggested a pure tone average of 50 decibels in the right ear and 15 decibels in the left ear (Figure 2). Over her course of stay in the hospital of 6 days, she was also administered injection vitamin B12 once daily. On follow up visits, patient showed marked improvement of symptoms. Patient was also counselled for carbogen therapy.

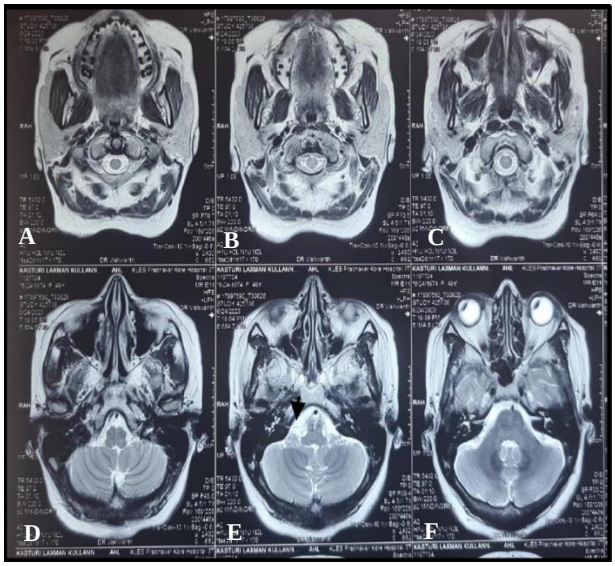

Figure 3 (A-F): MRI brain and inner ear suggestive of right AICA abutting the posterior aspect of vestibulocochlear nerve.

DISCUSSION

The anterior inferior cerebellar artery arises from the basilar artery and courses variably posterolaterally in the Cerebellopontine angle or along the underside of the cerebellum supplying the anterior cerebellum including the flocculus, middle cerebellar peduncle, and inferolateral pons. It gives rise to the labyrinthine (internal auditory) artery, which supplies the cochlea and vestibular system. A terminal artery, the labyrinthine artery is the sole blood supply of the labyrinth and an area of the brainstem and cerebellum. There is high variability in the course of the AICA within the CPA.[3]

A study done by Chadha et al concluded that Vascular loops in contact with CN VIII are a normal variant and subjects with unilateral hearing loss were twice as likely to have these vascular loops in the symptomatic ear.[4] Subjects with pulsatile tinnitus were about 80 times more likely to have a vascular loop in contact with CN VIII than patients with non-pulsatile tinnitus. This strongly supports the view that vascular loops in the internal auditory canal could generate arterial pulse synchronous tinnitus by transmission of vibrations, presumably through CN VIII into the cochlea. Therefore, it seems reasonable that in patients with pulsatile tinnitus, diagnostic work-up should include MRI scanning of the internal auditory canal. If a vascular loop is found in contact with the affected CN VIII, and no other cause for pulsatile tinnitus is identified, surgical intervention can be

considered in selected cases. These techniques include decompressing the internal auditory canal, or placing a silicon or Teflon cushion between the 'compressing artery' and CN VIII.[4] Before this type of procedure should be widely recommended for this indication, a prospective study is required to assess efficacy (and safety).[4] In a study done by Moosa et al it was concluded that microvascular decompression (MVD) of the vestibulocochlear nerve can be effective in selected patients who exhibit pulsatile tinnitus or disabling positional vertigo in the setting of a vascular loop within the ipsilateral IAC, but available evidence at this time does not support MVD for SSNHL.[3]

Steroid therapy is widely used as the standard treatment for SSNHL. The evidence for steroids comes from Wilson et al who found that steroids had a significant effect on the recovery of hearing in patients with hearing loss of 40-90 dB.[5] Also, a study done by Battaglia et al suggested that Idiopathic sudden sensorineural hearing loss patients treated with intratympanic dexamethasone + high dose prednisone taper (combination therapy) have a higher likelihood of hearing recovery than those treated with high dose prednisone taper alone.[6] Keeping this in consideration, we decided to start the patient on oral steroids on the same day of administration of intratympanic (IT) steroids. The most frequently administered intratympanic steroids are dexamethasone and solumedrol (methyl prednisolone sodium succinate). The research shows a wide variation in the concentration given. Most studies quote doses of 10-24 mg/ml dexamethasone and 30-40 mg/ml solumedrol. Higher concentrations may have better outcomes.[1]

In a study done by Yasheer et al it was found that IT injection of dexamethasone at a dose of 10 mg/ml was associated with better hearing outcomes compared with 4 mg/ml for the treatment of ISSNHL.[7] In a study done by Thomas et al it was concluded the superiority of IT Dexamethasone at 24 mg/mL for the treatment of ISSNHL, with significantly better recovery of PTA compared with 10 mg/ml.[8] Animal data have shown that higher concentrations of IT steroids result in greater accumulation within the cochlea as well as a longer duration of presence of the drug within the inner ear.[8] The main advantage of IT treatment is the reduction in systemic corticosteroid side effects. IT steroids very rarely cause changes in serum glucose levels in patients with diabetes. They may also be given to patients with cataracts, myasthenia gravis and glaucoma. Four factors as mentioned below, have been shown to affect recovery and have to be noted while treating patients with sudden sensorineural hearing loss: time since onset-the earlier the presentation the better the prognosis, age-there is a worse prognosis in over 60 years of age, vertigo-a poor prognostic indicator, audiogram-patients with profound hearing loss and a downward-sloping audiogram have a poorer prognosis.[1]

Fetterman et al suggested that if therapy with one or more treatment types was started within 1 month of sudden hearing loss, there was a ~50% rate of improvement vs. ~25% if started later than 1 month. The amount of improvement seen on sequential audiograms may reflect the position of the subject on his/her own spontaneous improvement curve.[9] Harada et al suggested that the severity of the initial hearing loss is used as an indication of more severe damage to the cochlea. A severe initial loss theoretically lowers the potential of recovery of hearing. Profound hearing loss at 2 kHz and mild hearing loss at 4 kHz were associated with high rates of cure. Up-sloping audiograms have been reported to have a better prognosis for recovery. The correlation between high-tone hearing loss and vertigo was explained by the anatomical proximity of the basal turn of the cochlea and vestibule. Cochlear lesions may cause vestibular damage by endolymphatic alterations via ductus reunions. Vertigo may be an indicator of the extent and severity of the injury. Time from onset of hearing loss to presentation also appears to be significant in prognosis. Most studies have demonstrated that presentation after 7-10 days results in a poor outcome.[10]

In a study equating the efficacy of oral versus intratympanic steroid therapy indicated equal efficacy comparing both the modalities.[11] In a study demonstrating the efficacy of oral steroid therapy in SNHL it was observed that approximately one third of patients had full recovery in hearing, one third had partial recovery, and approximately one third did not recover from hearing loss. In addition, the hearing level remained relatively stable following 2 months of treatment. More than 2 months of oral steroid therapy for ISSHL is not recommended because no additional benefits were evidenced.[12] In a similair study by O'Brien et al SNHL caused by a vascular AICA loop around vestibulocochlear nerve no obvious compression of the vestibulocochlear nerve indicates that the symptoms may have been caused by hypoxia or micro compression. The treatment with steroids improved the patient's tinnitus and hearing loss except in the higher frequencies. Although the first-line treatment for sudden SNHL is steroids to reduce inflammation, it is plausible in this case that steroid treatment may have resolved the acute cause of the patient's symptoms which unmasked the primary cause of the underlying SNHL caused by the AICA loop.[13] With such uncertainty regarding not only the association of vascular loops with SNHL/tinnitus but also the exact pathophysiology involved, there remains no standardized scaling system to guide treatment when vascular loops are suspected. A lack of uniform grading systems for vascular loops can also cause confusion.[13]

CONCLUSION

Although most cases of SSNHL are idiopathic, a number of treatable conditions do exist. The relationship between vascular loops around the vestibulocochlear nerve and sudden sensorineural hearing loss have to be kept in mind

while evaluating these patients. Surgical management is significant in cases which have a pulsatile tinnitus in association with sudden SNHL. Prognosis for hearing recovery is based on several factors, including time since onset, degree of deafness, age, and vertigo. Although SSNHL will often spontaneously improve without treatment, directed therapy against treatable causes along with steroid therapy can aid in the improvement of symptoms.

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