Isolated Contralateral Sudden Sensorineural Hearing Loss: An Unusual Manifestation of Pontine Infarct

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SUMMARY – *Unilateral, acute onset sensorineural hearing loss (“sudden sensorineural hearing loss” [SSNHL]) as an isolated event without other associated neurological deficits usually results from a lesion of the cochlea. Lesions in the ascending central auditory pathways cranial to the cochlear nucleus seldom result in unilateral hearing loss due to decussation of the central auditory pathways at multiple levels. We describe a patient with a tiny acute infarct in the right pons resulting in isolated acute onset left-sided SSHNL, without any other associated acute neurological deficits.*

Introduction

The auditory pathway has early decussation (immediately after the cochlear nucleus), as well as multiple levels of decussation. Due to the redundancy and decussations of auditory fibres in its central nervous system course, unilateral insult of the central pathways of auditory system rarely results in deafness in either ear. To the best of our knowledge, there are only three reported cases of contralateral hearing loss resulting from infarct of the lateral lemniscus. These patients also had additional acute neurological deficits. In our case, the patient only had acute onset of isolated left-sided hearing loss, without any other signs or symptoms of acute stroke, making it unique.

Case Report

A 58-year-old woman with acute respiratory failure developed left-sided acute hearing loss during her stay in the intensive care unit. Her medical history included diabetes, chronic renal disease, peripheral vascular disease, coronary artery disease, cardiac failure, and prior lacunar infarct in the left pons. Clinical examination showed normal ear canals and middle ear spaces. Weber examination lateralized to

the right, and the patient could not hear the tuning fork by air conduction on the left on Rinne testing. Pure tone audiometry (Figure 1) revealed hearing within normal limits on the right with exception of mild hearing loss at 250, 6000, and 8000 Hertz (Hz). There was no response in the left ear to the limits of the equipment with air conduction. Bone conduction could not be done on the left due to patient fatigue. The findings of the hearing tests and audiometry were consistent with left sensorineural hearing loss. The hearing loss was not associated with tinnitus, vertigo, aural fullness, otalgia, otorrhoea or trauma. There was no significant change in the residual baseline right-sided motor weakness from the prior left pontine infarct. The remainder of the central nervous system examination was unremarkable. MRI of the brain showed a tiny acute infarct in the right mid pons, in addition to a tiny old infarct on the left side of the pons slightly more anterior in location (Figure 2A,B). The findings were consistent with left-sided SSNHL due to an infarct in the right (contralateral) pons. The cochlear nerve was not well demonstrated as high resolution imaging was not done. There was no diffusion abnormality along the course of left cochlear nerve. MR/CT angiography was suggested to evaluate the vasculature, but was not performed.

Figure 1 Audiometry showing profound hearing loss on the left with no response at the limits of the audiometer with air conduction. X, unmasked air conduction, left; O, unmasked air conduction, right; <, unmasked bone conduction, right.

Figure 2 A) Axial diffusion-weighted image showing a punctate hyperintense infarct in the pons on the right side (long arrow). An old lacunar infarct in the left pons, slightly more anterior, appears hypointense (short arrow). B) Axial diffusion coefficient image showing restricted diffusion in the acute punctate infarct in the pons on the right side (long arrow). An old lacunar infarct in the left pons appears hyperintense (short arrow).

Discussion

Theories of possible aetiologies for SSNHL include labyrinthine (cochlear) vascular compromise, viral infection, vestibular schwannoma

(VS), and intracochlear membrane rupture¹. Unilateral SSNHL is a well-known association of AICA territory infarct and is commonly due to ischaemic injury of the cochlea, the peripheral end-organ, thus occurring on the ipsilat-

Figure 3 Graphic showing the major pathways of the central auditory system from the ventral and dorsal cochlear nuclei and superior olivary nuclei to the auditory cortex. Note the extensive decussations and bilateral projections starting with the second order neurons in the brainstem. This bilateral redundancy is the primary reason for the extreme rarity of unilateral acute SSNHL in brain stem infarcts.

Table 1 Literature review of contralateral sudden sensorineural hearing loss from brainstem infarct.

<i>Author</i>	<i>Age, Gender</i>	<i>Hearing loss</i>	<i>Associated neurological findings</i>	<i>MRI findings</i>
Doyle et al. ³	63 yrs, male	Sudden right sensorineural hearing loss	Right facial palsy, nystagmus, unsteady gait	Infarct of left upper pons, dorsomedial aspect
Murakami et al. ⁴	64 yrs, male	Sudden right sensorineural hearing loss	Initially: none Later: diplopia, vertigo, nausea, bilateral lateral gaze nystagmus, left cerebellar ataxia, right hemiparesis, Horner's and bilateral cortical blindness	Initial MRI 12 hours after onset- infarct in superolateral pons on the left and left cerebellum Follow up MRI after 2 weeks: Interval enlargement of left cerebellar infarct, new infarct in right cerebellum, dorsal pons, and bilateral occipital lobes
Lee et al. ⁵	60 yrs, male	Sudden right sensorineural hearing loss	Limb dysmetria, gait ataxia, facial palsy, gaze palsy	Infarct of left lower midbrain and upper dorsolateral pontine tegmentum

eral side of associated AICA territory infarcts². The auditory pathway has early decussation (immediately after the cochlear nucleus), as well as multiple levels of decussation (Figure 3). A critical concept is that due to the redundancy and decussations of the auditory fibres in their central nervous system course, unilateral insult of the central pathways of auditory system rarely results in deafness in either ear. Nevertheless, our patient suffered SSNHL in one ear seemingly from an acute infarct in the contralateral pons. To the best of our knowledge, there are only three reported cases of contralateral hearing loss resulting from infarct of the lateral lemniscus³⁻⁵. These patients also had additional acute neurological deficits [Table 1]. In our case, the patient only had acute onset of isolated left-sided hearing loss, without any other signs or symptoms of acute stroke, making it unique. We hypothesize that in our pa-

tient, unlike the usual pattern of decussation with some fibres crossing over and some fibres ascending on the same side, the majority of the ascending auditory fibres would have crossed over to the contralateral olivary complex or lateral lemniscus, resulting in contralateral hearing loss from the acute pontine infarct. The possibility that the patient suffered two insults -an identifiable acute infarct in the right side of pons and an additional insult to the left cochlea - which could not be identified on the MRI appears unlikely, given the lack of any acute neurological deficits other than SSNHL.

In spite of the bilateral redundancy of the auditory pathway, a tiny infarct in the ascending auditory pathway rarely results in contralateral hearing loss and should be interpreted as the likely cause of contralateral SSNHL, particularly when there are no other imaging or clinical findings suggesting alternate causes.

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