causing perceptual disinhibition (Figure 2-A) (3). Peduncular hallucinosis is most frequently caused by vascular lesions of the brainstem [8], but as cases are rare, most data on this topic is based on case reports and small case series where other causes such as infections, tumours and multiple sclerosis have been mentioned (9-12). Most patients with brainstem hallucinations have lesions that involve the auditory structures, located in the pontine tegmentum. These lesions involve sensory structures and are supposed to be responsible for cortical deafferentation and subsequent activation of cortical auditory structures (13).

Auditory hallucinations can be a hallmark of either cortical or subcortical brain damage. The underlying lesions responsible for their occurrence can be located at any level of the auditory pathway. While the most common type of paracusis in brainstem stroke is tinnitus, more complex types of hallucinations, as was the case of our patient, can be observed (14,15). Auditory hallucinations can be classified as being of verbal and non-verbal quality. In some cases it can be difficult to correctly classify the hearing impairment in the acute phase of the illness due to brainstem related accompanying signs (most often severe dysarthria), hyperacusis and auditive hallucinations being mistakenly interpreted as tinnitus (1). Verbal hallucinations have been classically associated with temporal lobe lesions, but brainstem stroke can also lead to such manifestations.

Multiple hallucinations have been reported in brain lesions that did not involve the classical sensory pathways, suggesting an indirect connexion to remote brain centres. A study on this issue used lesion network mapping to analyse the maps of resting functional imaging in normal subjects and subjects with brain lesions and new-onset hallucinations, with the aim of identifying the common anatomical basis. It was thus observed that for most of the patients with auditory hallucinations lesions were connected to the dentate nucleus of the cerebellum. Beside the role of the dentate nucleus, a standard system for both visual and auditory phenomena which involved the cerebellar vermis has also been described. It remains to be seen if disruption of cortical – cerebellar circuits is also responsible for auditory hallucinations in patients with pontine lesions (Figure 2-B) (16).

Auditory dysfunction is a rare manifestation of focal brainstem haemorrhage and limited data regarding the prevalence of hearing abnormalities associated with brainstem lesions is available (17,18). Numerous fibres of the acoustic pathway pass through the brainstem, but as there is a bilateral representation above the medullary-pontine junction, brainstem stroke often presents with vestibular symptoms and only rarely with hearing disturbances. Most neurons of the auditory pathway above the cochlear nuclei have a binaural quality meant to accomplish the complex coding needed for the space localisation of sounds. This type of space-sensitive perception can be achieved by two prompting inputs described as: interaural time difference and interaural level difference. Moreover, in line with this function, the nuclei along the auditory pathway receive prominently excitatory inputs from the contralateral ear (19).

The ventral cochlear nucleus is extensively connected to both inferior colliculi and olivary complex and the contralateral cochlear nuclei (15). Connections from the ventral cochlear nucleus travel to the

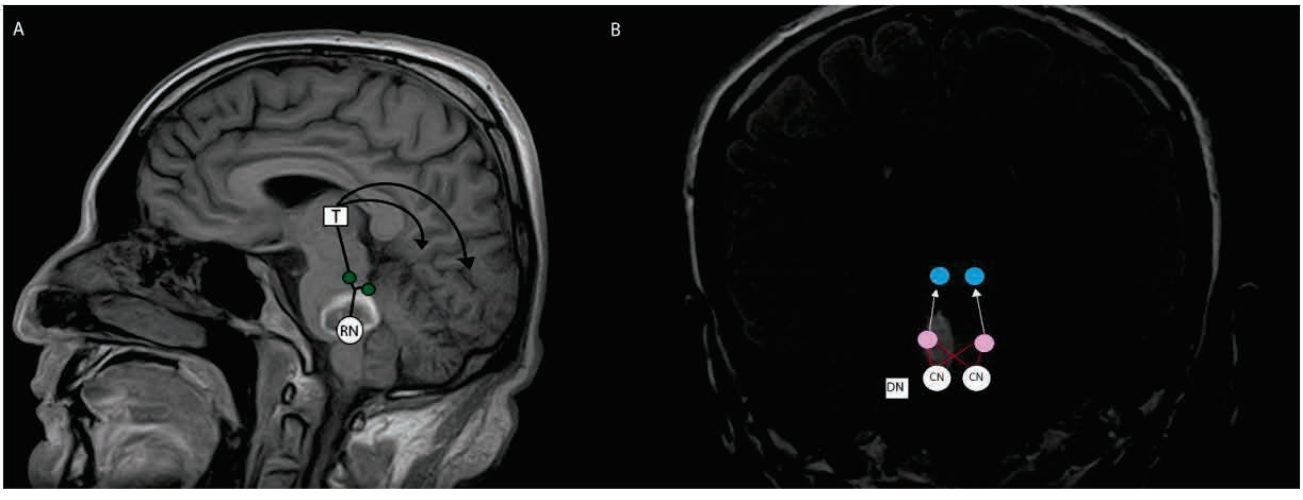

FIGURE 2. A: T1 sequence, sagittal section, showing the main pathway causing neurotransmitter imbalance in peduncular hallucinosis (RN – dorsal raphe nucleus; T – thalamus; green dots – pedunculopontine and laterodorsal tegmental nuclei). **B:** T2 FLAIR sequence, coronal section, showing main pathways involved in central auditory dysfunction caused by brainstem lesions (white dots – CN – ventral cohlear nuclei; pink dots – superior olivary complex; blue circle – inferior colliculi; DN – dentate nucleus)

trapezoid body, a structure which encompasses numerous auditory nuclei that integrate cochlear information. Some of these fibres synapse in the contralateral superior olivary complex, while others only pass through the trapezoid body to join the lateral lemniscus to the inferior colliculi (4). A subdivision of the trapezoid body, the medial nucleus of the trapezoid body, is particularly important for mediating inhibitory signals to the contralateral superior olivary complex, which plays a key role in interaural differencing. The superior olivary complex thus receives inhibitory output from the contralateral cochlear nucleus via the medial nucleus of the trapezoid body and excitatory signalling from the ipsilateral ventral cochlear nucleus (19,20).

Impairment of any of these pathways in brainstem lesions involving the pons, as was the case in our patient, can be the cause of central hearing impairment (Figure 2-B) (15). Our patient presented with bilateral, asymmetric hypoacusis, which we considered secondary to his pontine haemorrhage. What was further particular in this case was the association of paradoxical hyperacusis. In previously reported cases, the most likely cause was the injury of the auditory fibres projecting to the inferior colliculi, above the superior olivary complex and the lateral lemniscus (21,22). We assumed that the haemorrhage also led to a dysfunction of the right trapezoid body and the right superior olivary complex, which represent the first center of bilateral auditory integration in the brainstem (23). In support of this hypothesis, the follow-up MRI of the patient showed subtle signs of hypertrophic secondary olivary degeneration. However, involvement of the ventral acoustic striae that decussate in the trapezoid body could equally explain the bilateral hearing loss (23). A phenomenon of hypersensitization secondary to the damage of auditory sensory fibres most likely occurred in addition to the loss of inhibitory output from the right medial nucleus of the trapezoid body, contributing to the paradoxical left hyperacusis (22,24).

CONCLUSIONS

Peduncular hallucinosis is a rare syndrome usually caused by lesions in the upper brainstem. Associations of auditory hallucinations with hypoacusis and paradoxical hyperacusis, as was the case of our patient, has been rarely reported in literature. Further brain networking mapping studies are definitely needed to enrich our current understanding of such complex auditory impairments.

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