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CASE REPORT

Acute tinnitus and hearing loss as the initial symptom of multiple sclerosis in a child

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Summary Acute hearing loss with or without tinnitus has been reported in a number of adult series of multiple sclerosis (MS), but is considered a rare phenomenon. It generally occurs during disease exacerbations, rather than as an isolated finding or presenting feature. We present the case of an 11-year-old girl in whom persistent tinnitus and reversible hearing loss were the sole manifestation of MS at initial presentation.

1. Introduction

Acute hearing loss with or without tinnitus is rare in adults with multiple sclerosis (MS), and when present, often occurs during disease exacerbations and in association with other neurological deficits rather than as a sole symptom. In children, data is scarce and although monosymptomatic presentations of MS with brainstem symptoms have been described [1], tinnitus with acute sensorineural hearing loss (SNHL) has not been previously reported in this population.

We report the case of an 11-year-old girl with MS whose only symptom at initial presentation was unilateral non pulsatile tinnitus, and audiological testing showed an ipsilateral high frequency SNHL.

2. Case report

An 11-year-old girl presented to her local otolaryngologist with a 6 weeks history of sudden onset, unilateral, non-pulsatile, non fluctuating, subjective tinnitus which she described as a ''constant buzzing in the right ear''. This was associated with hearing loss. There was no associated vertigo, otalgia, or headache, and no history of recent trauma or infection. Past medical history was unremarkable and family history was only relevant for diabetes mellitus and thyroid disease. At this time, clinical examination was said to be normal and the only audiological test performed was a pure tone

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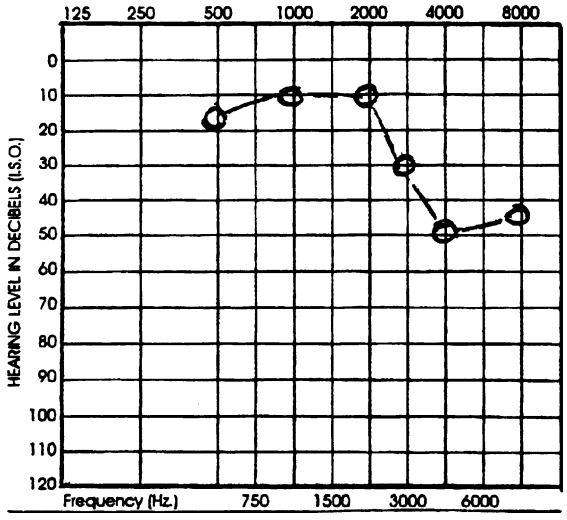

Fig. 1 Air and bone conduction thresholds in right ear of patient, were the same at initial presentation to her local otolaryngologist, 6 weeks after commencement of symptoms.

audiogram which showed moderate high-frequency sensorineural hearing loss on the right and normal hearing on the left (Fig. 1). Temporal bone CT was normal. No further management was advised.

A month later, the patient presented to the Otolaryngology Department, Royal Children's Hospital, Melbourne because of persisting tinnitus. Her clinical examination was once again normal but pure tone audiogram showed normal hearing bilaterally, with thresholds at 10 db or better. An MRI was performed and although no overt structural abnormality was identified, the study was not satisfactory due to large areas of signal dropout caused by the patient's metallic dental braces.

Two months later the patient underwent repeat MRI scan following removal of the dental braces, and this showed areas of demyelination with multiple hyperintense lesions on T2 weighted and FLAIR images within the periventricular white matter adjacent to the left trigone, the right side of the body of the lateral ventricle, the left frontal white matter and over the rostrum of the corpus callosum; the latter resembling the appearance of Dawson's fingers (Fig. 2). The patient was then referred to the Neurology Department for further assessment. There were no symptoms other than tinnitus and formal neurological exam was entirely normal.

MRI scan performed a further 3 months later demonstrated a new area of T2 hyperintensity in the right frontal white matter without evidence of post-gadolinium enhancement and no change in the previously noted lesions.

Over the next 12 months, the patient presented on two separate occasions with episodes of limb paraesthesias that resolved spontaneously after a 2—3 days period and one episode of unilateral 6th nerve palsy that resolved after a 3 days course of intravenous methylprednisolone. The MRI scans performed during these episodes showed partial resolution of old lesions but no evidence of new white matter abnormalities. CSF analysis showed no cells, normal protein and positive oligoclonal bands.

On last review, 2 years after initial presentation, her tinnitus persisted and, although her neurological

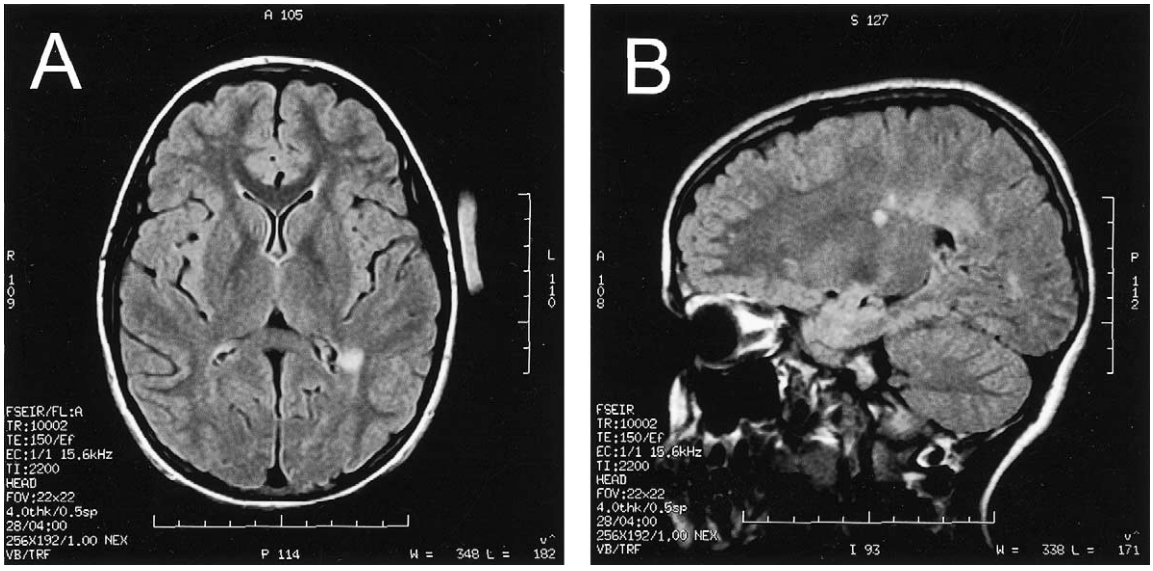

Fig. 2 (A) Axial FLAIR image showing a focal area of hyperintensity in the left posterior periventricular white matter. (B) Sagittal FLAIR image demonstrating multiple hyperintensities (Dawson's fingers) at the calloso—septal interface.