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Tuberculous meningitis presenting as sensorineural hearing loss

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Abstract
We report a 60-year-old male who presented to the Otorhinolaryngology department with an acute unilateral sensorineural hearing loss associated with fever and night sweats. The diagnosis of tuberculous meningitis was made. Unilateral sensorineural hearing loss as a presenting symptom of tuberculous meningitis has not been previously reported.

Key words: Hearing Loss, Sensorineural; Tuberculosis, Meningeal

Case report

A 60-year-old male of Asian origin presented as an emergency at the Otorhinolaryngology department with a two-day history of left-sided hearing loss, vertigo, double vision and neck stiffness. He had been complaining of fever and night sweats for the preceding two weeks, and indeed had had similar symptoms six months previously following a trip to Pakistan. He denied any tuberculosis contacts. On examination he appeared unwell, but was afebrile and non-tachycardic. Both external auditory canals and tympanic membranes showed normal appearances. Rhinne's test was positive bilaterally with Weber's localising to the right ear. There was no nystagmus and cranial nerve examination demonstrated a bilateral sixth nerve and a partial third nerve palsy. The white cell count was 4.8 g/dl and the erythrocyte sedimentation rate was 27. A chest X-ray showed miliary shadowing and focal consolidation in the right upper zone. Pure tone audiometry showed a hearing loss in the left ear of 70 dB with a sensorineural pattern. A medical review was sought and a presumptive diagnosis of tuberculous meningitis made.

Computerised tomography (CT) of the head showed mild hydrocephalus and cerebro-spinal fluid obtained by lumbar puncture revealed a protein content of 4.8 g/l and a white cell count of 41 with a monocytic picture; and was positive for acid-fast bacilli.

The patient started an anti-tuberculous regime of ethambutol, rifampicin and pyridoxine. He became increasingly drowsy three days later and was transferred to a local neurosurgical centre for observation. He returned to the medical ward two weeks later. No cause for this transient deterioration was found. His clinical picture markedly improved and the ocular palsy resolved, but his hearing in the affected ear six months later showed no significant improvement either clinically or on pure tone audiometry.

Discussion

Tuberculosis is the world's leading cause of death from a single infection. The incidence of tuberculosis has been increasing in the United Kingdom, with 7000 new cases each year.[1] One of the explanations for this increase is the association of tuberculosis and human immunodeficiency virus.[2] The initial infection with *Mycobacterium tuberculosis* is commonly subpleural, largely affected the mid to upper lung zones. Primary tuberculosis is usually symptomless but *Mycobacterium tuberculosis* that reaches the draining hilar lymph nodes may be seeded into the bloodstream, leading to widespread infection.[1]

Acute sensorineural deafness is considered an otological emergency, often requiring admission, and most cases are idiopathic in origin.[3] Treatment may be medical, surgical or a combination depending on the cause. Medical treatment includes the use of vasodilators, steroids and vitamins; the mechanisms of action of these treatments are not fully understood and there is a difference of opinion as to their benefits.[3]

Tuberculous meningitis may be asymptomatic or may present with signs of raised intracranial pressure and ocular palsies, but acute unilateral sensorineural hearing loss has not been reported as a presenting feature. Diagnosis is by a high index of suspicion combined with blood and serological markers; and is confirmed by lumbar puncture with examination of cerebro-spinal fluid, which shows white cells and reduced glucose, and is positive for acid-fast bacilli as in our case.[4] CT of the brain in these patients may show hydrocephalus, as in our case, parenchymal involvement, cerebral infarct and focal or diffuse cerebral oedema.[5] Infection of the middle ear associated with miliary tuberculosis and leading to acute sensorineural deafness has been reported; in this case the hearing loss returned to normal.[6] In our case there has been no such improvement in the patient's hearing loss to date.

The mechanism of tuberculous meningitis causing sensorineural hearing loss is unclear but may result from haematogenous, lymphatic, perineural or direct spread to the inner ear structures, as in bacterial meningitis.[7] Cranial nerve involvement may be explained by the accompanying arachnoiditis and adhesions.[8] As with bacterial otitis media, the possibility of tuberculous infection spreading

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to the cerebral meninges directly from the middle ear through necrotising bone or via the inner ear cannot be excluded. The latter could be explained by a tuberculous otitis media spreading by direct invasion through the oval or round windows into the internal auditory meatus and cochlea.[7] In our case the patient showed signs of meningitic involvement such as neck stiffness and cranial nerve involvement, just before the sudden sensorineural hearing loss. However, there were no signs or symptoms suggestive of otitis media.

The main treatment of tuberculous meningitis is with anti-tubercular chemotherapeutic agents as per local guidelines; intensive monitoring in a high dependency area; and urgent surgical treatment of complications such as cerebral abscesses or osteomyelitis. The prognosis has been reported to correlate with evidence of focal weakness, the Glasgow Coma Scale score, age, cranial nerve palsy and the degree of hydrocephalus at presentation.[9]

In this case a patient with no previous history of ear problems developed an acute sensorineural deafness secondary to tuberculous meningitis. This presentation has not been reported before and adds a new diagnosis to our differential of acute sensorineural hearing loss.

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