CLINICAL REPORTS

Meningococcal meningitis presenting with bilateral deafness and ataxia

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Summary

A 50-year-old man presented with bilateral deafness and ataxia of sudden onset and without constitutional symptoms or signs of meningeal irritation. He was subsequently proved to have meningococcal meningitis, and the deafness and ataxia resolved following appropriate antibiotic therapy.

KEY WORDS: meningococcal meningitis, deafness, ataxia.

Introduction

The classical signs and symptoms of meningococcal meningitis are fever, malaise, headache, nuchal rigidity, nausea, vomiting, various degrees of disorientation and mental depression leading to coma (Arenstein, 1978). Rare presentations which have been reported are: acute cerebellar ataxia (Yabek, 1973), subarachnoid haemorrhage (Huskisson and Hart, 1969) and brain abscess (Daniels, 1963). We have recently seen a man who presented with bilateral deafness and ataxia of sudden onset who was subsequently proved to have meningococcal meningitis.

Case report

A 50-year-old man was admitted with a history of rapidly diminishing auditory acuity over 3 days. On the day of admission he became completely deaf bilaterally and was unable to walk unassisted.

On examination, he was well oriented, afebrile and there was no neck stiffness. Neurological assessment revealed bilateral total sensory deafness and severely ataxic gait. Laboratory investigations revealed an elevated erythrocyte sedimentation rate (50 mm/hr, Westergren); full blood count, serum electrolytes, urea, glucose, vitamin B₁₂, liver function tests, coagulation profile, serology for syphilis and blood urine culture were normal or negative. An electrocardiogram and radiograms of the skull, sinuses and chest were normal. On the basis of our initial

examination and investigations we were unable to formulate a definitive diagnosis.

Three days later, the patient developed pyrexia (38.5°C) and became drowsy and confused with nuchal rigidity. A purpuric rash was noted over the entire body. Lumbar puncture yielded turbid cerebrospinal fluid (CSF) containing protein 6.76 g/l, glucose 1 mmol/l (blood glucose 8.7 mmol/l), and lactate 4.84 mmol/l (normal range 1.28-3.60 mmol/l). On microscopy there were neutrophils 5.2×10^9 /l, lymphocytes 0.8×10^9 /l and red cells 0.265×10^9 /l. Counter current immunoelectrophoresis of the CSF detected the presence of antigen specific for *Neisseria meningitidis* type B which was subsequently cultured. Blood, urine and throat swabs cultures remained negative.

Treatment was initiated with penicillin G, 2 million units given every 2 hr by intravenous injection. Following 1 week of treatment the patient was afebrile and oriented. Hearing was normal bilaterally, although the gait was still ataxic. Two weeks later, the patient was able to walk unassisted and was discharged with a mild residual unsteadiness.

Discussion

Inflammatory reaction and fibrosis of the meninges along the roots of the cranial nerves are thought to be the cause of the cranial nerve palsies which occasionally complicate meningococcal meningitis (Merritt, 1973). In addition damage to the auditory nerve may occur suddenly, producing an auditory defect that is usually permanent (Merritt, 1973). Since there was no ear disease or other signs of cerebellar damage in our patient, it appears that his presentation with nerve deafness and ataxia must be attributed to early involvement of the cochlear and vestibular components of the VIIIth nerves in the inflammatory process. In the absence of constitutional symptoms or other signs of meningeal inflammation, this presentation was highly

misleading, and it was only when such signs did appear that the diagnosis was made.

The basis for a selective involvement of the VIIIth nerves remains unknown, however the reversibility of the lesion precludes a mechanism such as thrombosis of the nutrient arteries. It is possible that damage may have occurred through an allergic reaction; such a mechanism has been invoked to explain the facial paralysis that occurs not infrequently after the meningeal reaction has subsided (Merritt, 1973). Whatever the precise mechanisms may be, sudden onset of nerve deafness, with or without signs of vestibular involvement, should alert the clinician to the possibility of meningitis even in the absence of other signs and symptoms.

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