

Angiostrongylus eosinophilic meningitis associated with sensorineural hearing loss

VERAJIT CHOTMONGKOL, M.D., KWANCHANOK YIMTAE, M.D.*, PEWPAN M. INTAPAN, M.D.†

Abstract

A 59-year-old woman who presented with chronic headache, neck stiffness and left-sided hearing loss is reported. The diagnosis of *angiostrongylus* eosinophilic meningitis was made. The patient improved after treatment with prednisolone, including hearing. *Angiostrongylus* eosinophilic meningitis associated with sensorineural hearing loss has not previously been reported.

Key words: Angiostrongylus; Meningitis; Hearing Loss, Sensorineural

Introduction

Angiostrongylus cantonensis is the most common infectious cause of eosinophilic meningitis. Acute to subacute severe headache with non-focal neurological findings, with the exception of occasional involvement of the cranial nerve, are the most common presenting symptoms.^{1–4} Unilateral or bilateral involvements of the VIth and VIIth cranial nerves were associated with this disease. We report a case of eosinophilic meningitis associated with sensorineural hearing loss which, to our knowledge, has not previously been reported.

Case report

A 59-year-old woman Thai farmer was admitted to Srinagarind Hospital in October 2002 because of headache and hearing loss for one month. She had been in good health. One month previously she experienced generalized headache and left-sided hearing loss. She was treated at local clinics and a local hospital without improvement. One day prior to admission she had severe headache with vomiting. She gave a significant history of having eaten raw Pila snails one week before this illness. On physical examination she was alert and afebrile. She had a stiff neck and bilateral papilloedema. Both external auditory canals and tympanic membranes were normal in appearance. Rinne's test was positive bilaterally, with Weber's localizing to the right ear. There was no nystagmus. Audiometry showed right mild sensorineural hearing loss and left severe, downslowing sensorineural hearing loss. The peripheral white blood cell (WBC) count was 7000 cells/mm³, with 65 per cent polymorphonuclear cells, 21 per cent lymphocytes, six per cent monocytes and six per cent eosinophils. Serum glucose, BUN and creatinine, electrolytes, liver function tests, VDRL and TPHA, urinary analysis, chest X-ray and CT scan of the brain were within normal limits. Cerebrospinal fluid (CSF) analysis showed an initial opening pressure of 300 mmH₂O. The WBC count was 273 cells/mm³, with 39 per cent polymorphonuclear cells, four per cent lympho-

cytes and 54 per cent eosinophils. The protein level was 158 mg/dl and glucose level was 54 mg/dl (simultaneous serum glucose level of 111 mg/dl). Stains and cultures of the CSF for bacteria, acid-fast bacilli and fungi and cryptococcal antigen were negative. Serologic analysis of the serum for antibody against the 29 kDa specific antigen of *A. cantonensis* was present on immunoblotting.

Eosinophilic meningitis was diagnosed. The patient was treated with prednisolone 60 mg/day in three divided doses for two weeks, and two tablets of acetaminophen were given every four to six hours to relieve the headache if it persisted or recurred. The headache had disappeared by the second day of treatment, and the hearing loss gradually improved. Repeat audiometry two months later showed right mild sensorineural hearing loss and normal hearing in the left ear. The auditory brainstem response revealed a normal response on the right ear, but absence on the left despite normal hearing.

Discussion

In humans, infection by *A. cantonensis* is caused by eating third-stage larvae in raw or inadequately cooked intermediate hosts, such as snails and slugs, or transport hosts such as freshwater prawns, frogs, and the yellow tree monitor. When third-stage larvae are ingested they penetrate the blood vessels of the intestinal tract and are carried to the meninges, where they usually soon die. A presumptive diagnosis may be made in patients who have symptoms of meningitis with CSF eosinophilia and a history of consumption of raw snails. A number of serological tests have been used to support the diagnosis of angiostrongyliasis. Recently, we found that specific antigenic band of 29 kDa antigen from young adult female worms may serve as a reliable marker for the diagnosis of human angiostrongyliasis.⁵ The role of antihelmintics is still inconclusive. Supportive treatment, such as analgesic drugs and repeat lumbar puncture, is recommended. We recently demonstrated that a two week course of prednisolone was beneficial in relieving headache, shortened

From the Departments of Medicine, Otolaryngology* and Parasitology†, Faculty of Medicine, Khon Kaen University, Khon Kaen 40002, Thailand.

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the median time until resolution of the headache, and reduced the need for repeat lumbar puncture.⁴

- **This paper is a case report of sensorineural hearing loss associated with meningitic infection with *Angiostrongylus***
- **The patient improved after steroid therapy**
- **It is the author's contention that the association between this infection and sensorineural deafness has not been previously reported, but no search strategy is included to validate this claim**

In our patient angiostrongyliasis was definitely diagnosed from the clinical manifestations, CSF abnormalities and serologic findings. The auditory brainstem response suggested a retrocochlear lesion, in which case the sensorineural hearing loss would be secondary to cranial neuropathy, and the location causing cranial neuropathy should be within the subarachnoid space. The mechanism of this disease causing sensorineural hearing loss is unknown, but is likely to be due to an inflammatory response to foreign bodies and the possible toxicity of worm excretory products. From this presentation, eosinophilic meningitis caused by *A. cantonensis* adds a new diagnosis to the differential diagnosis of sensorineural hearing loss associated with meningitis.

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Address for correspondence:
Verajit Chotmongkol, M.D.,
Department of Medicine,
Faculty of Medicine,
Khon Kaen University,
Khon Kaen, Thailand 40002.

Fax: 066-043-202476