



Eosinophilic meningitis: A case report

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ABSTRACT

Eosinophilic meningitis is defined as the presence of more than 10 eosinophils/mm³ in the cerebrospinal fluid (CSF) and/or eosinophils accounting for more than 10 percent of CSF leukocytes. Angiostrongyliasis, the most common infectious cause of eosinophilic meningitis, results from infection by the parasite nematode *Angiostrongylus cantonensis*. Here we report a case of eosinophilic meningitis due to *A. Cantonensis* in a 46 years old male patient.

INTRODUCTION

Eosinophilic meningitis is a rare clinical entity that can be useful in narrowing the differential diagnosis of central nervous system disease. It is defined by the presence of 10 or more eosinophils/ μ L in the cerebrospinal fluid (CSF)[1] *Angiostrongylus cantonensis* is the most common cause of eosinophilic meningitis among humans. Individuals become infected with this parasite by consuming contaminated raw snails, vegetables, small mollusks, or fresh water contaminated with the third-stage larvae of this parasite. The third-stage larvae of *A. cantonensis* induce eosinophilic meningitis once entrenched in the central nervous system[2]. Humans can acquire the infection by eating raw or undercooked snails or slugs infected with the parasite; they may also acquire the infection by eating raw products containing a small snail or slug. Infection can also be transmitted by ingestion of infected paratenic animals, such as crab or freshwater shrimp, or by ingestion of infected centipedes. In humans, worms migrate to the brain (or rarely, the lungs) but do not produce eggs. The incubation period of *A. Cantonensis* averages one to three weeks but has ranged from one day to greater than six weeks[3]

CASE REPORT

A 46 years old male patient was admitted in Neurology

department in our hospital with complaints of weakness of lower limbs, headache since 1 week, and neck pain since one day. He was a known case of hypertension and on regular medication. On examination, the patient was conscious, oriented, PEARL (+ ve) and blood pressure was 150/100mmHg. The echocardiographic examination shown bradycardia. Results of serum electrolyte, blood urea nitrogen, glucose, and liver function tests were within normal limits. He was alert, collaborating, and had nuchal rigidity. His sounds were normal and the abdomen showed no hepatosplenomegaly. Chest radiograph findings were normal, and computed tomography (CT) scan of the brain was unremarkable. Patient was started empirically with antibiotics and other symptomatic management.

The patient's headache persisted, and so the consultant advised for a lumbar puncture, and CSF study showed 700 cells/ μ L with eosinophilic pleocytosis. At the time of first patient interview, patient did not have any type of travel history. After getting the CSF report patient was again interviewed for a detailed history which shown that he had a history of taking partially cooked monitor lizard- liver and blood. The patient was treated with antibiotics, IV dexamethasone, a course of Albandazole (14 days), amitriptyline, antihypertensives and other supportive measures. The patient recovered after 15 days of treatment. He was then discharged at 14-days follow-up, the

general state was good, but paresthesia over the face persisted.

DISCUSSION

Eosinophilic meningitis due to *A. Cantonensis* occurs principally in Southeast Asia, particularly Thailand and Malaysia but also Southern Vietnam, as well as throughout the Pacific basin, including Indonesia, the Philippines, Taiwan, China, Japan, and several smaller Pacific islands. Due in part to ship-borne dissemination of infected rats, the parasite has also been found outside of this broad area[3]. The difference in our case from other reported cases is that this patient did not have any type of travel history and the same was the reason for delayed diagnosis.

The larvae of *A. cantonensis* are excreted in the feces of rats and picked up by intermediate or transport hosts such as snails, vegetables, slugs, and small molluscs. Humans become infected after eating these hosts or fresh produce contaminated by them. Upon ingestion, the larvae penetrate the vasculature of the gastrointestinal tract and usually migrate into the CNS via the bloodstream, causing eosinophilic meningoencephalitis. After an incubation period of 1 to 3 weeks, signs and symptoms such as headache, visual disturbance, photophobia, vomiting, nuchal rigidity, hyperesthesia, paresthesia, and fever (less common) may develop. A history of severe frontal or bitemporal headache is characteristic. Paresthesias are the most distinctive neurological finding in adults with angiostrongyliasis; symptoms may be present on the extremities, trunk, or face, and they can persist for weeks after other symptoms have resolved. Our patient also showed severe headache with paresthesia over face and nuchal rigidity.

Serologic tests along with a detailed patient interviewing were useful for differential diagnosis of CNS parasitic infections. Making a definitive diagnosis of eosinophilic meningitis due to *A. Cantonensis* is a major challenge. Patients with a history of exposure in an area of endemicity, a compatible clinical presentation, and CSF eosinophilia should be considered for the diagnosis⁴. Because larvae are rarely detected in the CSF, a definitive diagnosis is very difficult, being often based only on the identification of contaminated foods that the patients may have ingested and based on the symptoms and signs they present[5]. We believe that Eosinophilic meningitis of our patient was probably caused by *A. cantonensis*, on the basis of epidemiological and clinical data and of the response to therapy. Chotmongkol and colleagues underlined that a 2-week course of prednisolone (60 mg/day) and albendazole (15 mg/kg/day) determined a complete disappearance of headaches in 88.5% of patients[6] The same combination therapy was successful in our case as well.

CONCLUSION

Eosinophilic meningitis is a rare CNS infection. Angiostrongylus cantonensis remains the most common cause of Eosinophilic Meningitis and Albendazole administration for two weeks is considered to be the drug of choice for the eradication of the pathogen. Praziquantel is an alternative to albendazole. Corticosteroids are often combined with albendazole to control the symptoms of meningitis including severe headache.

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