Recovery of *Angiostrongylus cantonensis* from Cerebrospinal Fluid of a Child with Eosinophilic Meningitis

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Viable *Angiostrongylus cantonensis* was recovered from the cerebrospinal fluid of a 17-month-old boy with eosinophilic meningitis. Neurological findings were minimal, and the child had an uneventful recovery.

Eosinophilic pleocytosis due to invasion of the central nervous system by helminths is an uncommon observation in the United States. However, this is not an uncommon finding in meningitis cases seen on certain South Pacific islands and in areas of Southeast Asia (2, 12, 14, 19). Most of the eosinophilic meningitis in these areas is thought to be due to infection of the central nervous system by the metastrongylid lungworm of rats, *Angiostrongylus cantonensis* (12, 15, 20). Relatively few reports in the world literature have documented eosinophilic meningitis due to this parasite by actually demonstrating the worm in the central nervous system, particularly in nonfatal cases. This report describes a young visitor to Hawaii with an eosinophilic pleocytosis from whose cerebrospinal fluid (CSF) *A. cantonensis* was recovered.

Case report. D. A., a 17-month-old boy, was admitted to Maui Memorial Hospital because of irritability, vomiting, and personality change (consisting of hitting and biting his mother), followed by lethargy occurring over a 6-day period. Physical examination was unremarkable, including no fever or evidence of meningeal irritation. A lumbar puncture was performed. The CSF was clear; however, what appeared to be living thread-like worms were observed in the fluid. The cell count of the CSF was 230 leukocytes per ml. The cell differential was reported as 90% lymphocytes and 10% neutrophils. The CSF glucose level was 65 mg/100 ml. Culture of the CSF revealed no growth of bacteria after 72 h. The peripheral leukocyte count was 16,700/ml, with 60% neutrophils, 36% lymphocytes, 1% monocytes, and 3% eosinophils.

The patient was transferred to Kauaikeolani Children's Hospital in Honolulu. The physical and neurological examination was again normal, except for a slight bilateral internal strabismus. A repeat lumbar puncture was performed about

72 h after the initial one. The opening pressure was 130 mm of water. The CSF was hazy, and the cell count was 708 leukocytes per ml and 396 erythrocytes per ml. A Wright stain of the cells in the spinal fluid revealed 53% lymphocytes, 1% polymorphonuclear leukocytes, 1% monocytes, and 45% eosinophils. The CSF protein was 37 mg/100 ml, and glucose was 57 mg/100 ml. The CSF culture for bacteria was again negative after 72 h. A repeat peripheral leukocyte was 28,000/ml, with a differential of 34% neutrophils, 49% lymphocytes, 4% monocytes, and 13% eosinophils. A stool examination for ova and parasites was negative.

The hospital course was unremarkable except for a low-grade fever. The patient was given supportive and symptomatic care and recovered completely, including the minimal internal strabismus. He was discharged on hospital day 4. A 3-year follow-up examination revealed normal growth and development; the patient presently has no apparent sequelae of his illness.

The parasites observed in the spinal fluid obtained on Maui were identified as four female and one male young adult *A. cantonensis*.

Human *A. cantonensis* infection associated with an eosinophilic pleocytosis was first reported on Taiwan in 1945 (3). That case was fatal. It was not until 17 years later that human infection was again reported, when young adult parasites were observed in the brain of a patient with fatal eosinophilic meningoencephalitis (13). Since that time evidence has been accumulating that *A. cantonensis* is a common cause of eosinophilic meningitis in certain areas of the world (12, 15, 20). In the United States, cases thought to be due to this nematode are uncommon and have been reported only from the Hawaiian Islands (5, 8, 13).

Actually recovering viable *A. cantonensis* from the CSF appears to be a relatively uncommon event, and only a few reports have been able to document *A. cantonensis* in the central

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nervous system associated with an eosinophilic pleocytosis (4, 7, 19). Finding the parasite in the CSF of our patient is the first report of recovering viable parasites from an eosinophilic meningitis patient from the United States.

The life cycle of *A. cantonensis* involves the domestic rat as a definitive host, with snails and slugs serving as the intermediate host (17). Human infection is acquired by consumption of a molluscan intermediate host or a transport host which has not been properly cooked and contains viable infective third-stage larvae. When consumed by their normal rat host, these larvae naturally invade the central nervous system and apparently have a similar propensity to do so when a human becomes an accidental host. All the possible sources of human infection have not been identified, and some infections are suspected of having been acquired by eating raw and poorly cooked transport hosts, such as freshwater shrimp, aquatic and amphibian crabs, and certain marine fish (15). They apparently become contaminated with infectious larvae by eating the infected molluscan intermediate host. The Hawaiian Islands are an endemic area for *A. cantonensis*, and garden snails and slugs commonly contain infective larvae (17). Consequently, they represent a potential source of danger for young children, who may indiscriminately put objects in their mouths. Although snails and slugs were common in the home environment of our patient, he was not noted to handle or eat them. The source of the infection of this patient has not been established.

Minimal or no fever in the presence of a significant CSF pleocytosis, as observed in our patient, is not an unusual finding in this disease (5). This patient also had the characteristic uneventful course and spontaneous recovery typical of eosinophilic meningitis. There is no specific therapy for patients with eosinophilic meningitis. Parenteral penicillin, tetracycline, and prednisone were not observed to have any therapeutic benefit over an untreated group of patients (12). In general, this disease is thought to be self-limited, although recent reports have described serious sequelae and even death due to *A. cantonensis* infection (12, 16, 19). A 3-year follow-up on our patient revealed no apparent sequelae from the infection.

Making a definite diagnosis of eosinophilic pleocytosis due to *A. cantonensis* is usually difficult. A serological test is currently not available to specifically detect an infection due to this parasite. Recovery of the parasites from the CSF would document the cause of the disease, but this is a relatively rare occurrence. Therefore, the diagnosis of eosinophilic meningitis due to *A. cantonensis* is usually a clinical one, requiring

the presence of eosinophils in the CSF and a compatible epidemiological history and clinical course. A possible problem in diagnosis, as was observed with the initial analysis of the CSF of this patient, is the documentation of eosinophils in the spinal fluid. Eosinophils are not easily distinguished from neutrophilic leukocytes in a counting chamber unless specifically looked for. Staining of CSF cells with Wright or Giemsa stains and looking for eosinophils is the preferable method.

Simply finding eosinophils in the CSF does not establish the diagnosis of eosinophilic meningitis due to *A. cantonensis*. However, finding an eosinophilic pleocytosis usually does suggest invasion of the central nervous system by a helminthic parasite. The parasitic agents most commonly implicated are the cysticercus of the pork tapeworm *Taenia solium* (10), the lung fluke *Paragonimus westermani* (9, 11), and the nematode *Gnathostoma spinigerum* (6). The only well-documented nonhelminthic infectious cause of an eosinophilic pleocytosis is *Coccidioides immitis* (1, 18).

The increase in world travel has resulted in the occurrence of a number of human infections with which many United States physicians might be unfamiliar. This case report illustrates a distinct pathological finding, eosinophilic pleocytosis, for which there are only a limited number of known causes. By being aware of the possible causes, physicians will have a better idea of what epidemiological information to inquire about and what to expect during the course of the disease, and they can have a more rational approach to therapy.

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