Case Reports

Eosinophilic Meningitis

PHILIP R. FISCHER, MD
Salt Lake City

CEREBROSPINAL FLUID EOSINOPHILIA may be caused by a variety of agents including parasites, fungi, neoplasms and central nervous system foreign bodies.[1] Worldwide, the rat lungworm *Angiostrongylus cantonensis* is the most common cause of what is typically known as eosinophilic meningitis. Diagnosis of central nervous system infestation with this parasite in the continental United States, however, has not previously been reported. In this paper I describe a case of eosinophilic meningitis presumed to be due to *A cantonensis* that was diagnosed in Utah, review the current knowledge of human disease caused by this parasite and report two symptoms not commonly associated with this illness.

Report of a Case

A 16-year-old boy presented to the Primary Children's Medical Center, University of Utah Medical Center, Salt Lake City, because of headache, malaise, myalgia and migratory arthralgia for two weeks. For the two months before admission, the patient had been working as a field hand in Hawaii and was in good health. Two weeks before admission, headache, muscle and joint discomfort, fatigue and subjective fever developed. His temperature was 37°C (98.6°F) when seen by a local physician who suspected a viral syndrome. Symptoms increased with painful and possibly swollen ankles, knees, hands and wrists. His headache became severe. After flying to Salt Lake City from Honolulu, the patient sought medical attention.

Detailed questioning elicited that the patient, on a dare, had ingested a live land snail four weeks before admission. His medical history was remarkable only for infectious mononucleosis six months before admission.

On physical examination the patient was mildly lethargic. His temperature was 37.4°C (99.3°F), the heart rate was 100 per minute and the blood pressure was 138/82 mm of mercury. There were mild papilledema, nuchal rigidity and mild diffuse abdominal tenderness without any palpable abnormality. The hands and feet were hyperesthetic. The examination otherwise showed no abnormalities.

The leukocyte count was 12,400 per μl with 57% polymorphonuclear cells, 31% lymphocytes, 7% monocytes and 5% eosinophils. A computed tomographic scan of the head showed normal findings. Lumbar puncture showed an opening pressure of 400 mm of water with cerebrospinal fluid that had 69 mg per dl protein, 50 mg per dl glucose, 10 erythrocytes per μl and 452 leukocytes per μl (3% polymorphonuclear cells, 63% lymphocytes, 9% monocytes and 25% eosinophils). Tests of electrolytes, renal function, liver enzymes and creatinine phosphokinase level all gave normal values. Bacterial cultures of the blood and cerebrospinal fluid were negative, and no ova or parasites were found in the stool or cerebrospinal fluid specimen. Radiographs of the ankle and chest were normal.

The patient was treated with aspirin and acetaminophen. The headache subsided some, but papilledema persisted throughout his hospital stay. His deep joint pains diminished, but his superficial skin pain and sensitivity increased after four days in hospital. Diplopia with leftward gaze, blurred vision, left facial weakness and transient hypertension to 164/80 mm of mercury developed. Then, stable with decreasing discomfort, he was discharged on the eighth hospital day.

Most of the patient's symptoms and physical findings resolved during the first few weeks following discharge. Nonetheless, some mild paresthesias are still present six months later. He has also had a pronounced bilateral decrease in visual acuity with evidence of optic atrophy.

Discussion

Angiostrongylus cantonensis is a roundworm that most commonly uses the rat as a definitive host. Snails and slugs act as intermediate hosts and carry the larvae. Larvae ingested by rats migrate to the central nervous system and finally to the rat lung where mature adult worms reproduce. Humans are an accidental final host and apparently do not allow continued reproduction.[2]

Angiostrongylus is found throughout the Pacific and southeast Asian regions of the world. Infection occurs year-round but is most common in the summer months. It has been reported in persons of all ages but, in some areas, most cases occur in children. Larvae may invade a human host either by ingestion of an intermediate host—as presumed in our patient—or following superficial skin contact with the larvae.[3]

There is no definitive diagnostic test for *A cantonensis*. On occasion, worms have been recovered from spinal fluid,[4] brain tissue[5] and the anterior chamber of the eye.[6] Usually adult worms or larvae cannot be re-

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From the Department of Pediatrics, University of Utah Medical Center, Salt Lake City.

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Reprint requests to Philip R. Fischer, MD, Department of Pediatrics, University of Utah Medical Center, 50 North Medical Drive, Salt Lake City, UT 84112.

covered and the diagnosis must be based on a typical clinical presentation, spinal fluid eosinophilia and travel to or contact with an area known to host the organism.

The typical clinical course[2,7,8] begins a few days to four weeks after contact with the larvae. Headache is the most common presenting symptom and may be accompanied by signs of meningeal irritation. Fever is usually absent or low grade. Neurologic abnormalities are not uncommon and include papilledema, visual impairment, sixth and seventh cranial nerve weakness, paresthesias and altered mental state. Peripheral eosinophilia is common, and elevated cerebrospinal fluid leukocyte counts, with many eosinophils (average of 40%), are usual. The clinical course is usually self-limited, with symptoms resolving in one to five weeks. Facial paralysis is often the most persistent symptom.

There is no known specific therapy, and supportive measures are the indicated treatment. Antibiotics and steroids are ineffective.[7] The illness is usually benign, and the prognosis for gradual and full improvement within one to two months is good.[7]

This patient ingested a possible intermediate host from an area endemic for *Angiostrongylus*. His course of headache, central nervous system involvement, spinal fluid eosinophilia and some gradual resolution is characteristic of *A cantonensis* infestation. His myalgia and arthralgia, however, were unusual. Laboratory tests in search of evidence for myositis, active mononucleosis and rheumatic fever gave normal findings, and it was concluded that the myalgia and arthralgia were caused by his parasitic disease. The duration of visual impairment seen in this patient is also unusual. Possible pathogenetic mechanisms of altered vision include optic neuritis and intracranial adhesions and are discussed elsewhere.[7]

In conclusion, this patient shows characteristic findings of the eosinophilic meningitis caused by *Angiostrongylus cantonensis* along with newly associated symptoms. With frequent travel to and from endemic areas, it behooves physicians all over the world to be aware of this disease.

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Effect of Respiratory Alkalosis in Tricyclic Antidepressant Overdose

HOWARD A. BESSEN, MD
JAMES T. NIEMANN, MD
RICHARD J. HASKELL, MD
ROBERT J. ROTHSTEIN, MD
Torrance, California

OVERDOSAGE WITH TRICYCLIC ANTIDEPRESSANTS is a common clinical problem. Patients may present with signs and symptoms of peripheral (tachycardia, mydriasis, dry mouth, ileus) or central (agitation, delirium, seizures, coma) cholinergic blockade.[1,2] The most life-threatening consequence of overdosage from these drugs may include a variety of cardiac conduction and rhythm disturbances.[2-8] Treatment of tricyclic antidepressant-induced cardiotoxicity with physostigmine and various antiarrhythmic drugs has yielded conflicting results.[2,7-9] Resolution of cardiac conduction and rhythm disturbances following systemic alkalinization has been reported in experimental animals poisoned with tricyclic antidepressants[9,10] and in human subjects.[10-13] Alkalinization has generally been accomplished with sodium bicarbonate and occasionally with hyperventilation. Previous clinical reports attesting to the beneficial effects of systemic alkalinization, however, have involved subjects who were acidemic before treatment, and it has been argued that their improvement was the result of correction of the accompanying acidemia and not the alkalinization per se.[14]

The following case report suggests that tricyclic antidepressant-induced alterations in cardiac conduction can be significantly affected by hyperventilation and respiratory alkalosis in the absence of pretreatment acidemia.

Report of a Case

A 30-year-old woman was admitted to the emergency department after being found unconscious by her family. The patient had a history of depression and attempted suicide and was receiving outpatient psychiatric care. The patient's family was aware that she was taking medications, but was uncertain of the kind or amount.

Initially her temperature was 41.0°C (105.8°F), pulse 128 per minute and regular, respirations 20 and blood pressure 90/60 mm of mercury. The patient was comatose and unresponsive to verbal or tactile (painful) stimuli. On neurologic examination she had no oculocephalic, oculovestibular, corneal or gag reflex. Both pupils measured 4 mm and were weakly reactive to light. The extremities were flaccid and areflexic. The

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From the Departments of Emergency Medicine (Drs Bessen, Niemann and Rothstein) and Medicine (Dr Haskell), Harbor-UCLA Medical Center, Torrance, California, and UCLA School of Medicine, Los Angeles.

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Reprint requests to Howard A. Bessen, MD, Department of Emergency Medicine, Harbor-UCLA Medical Center, 1000 West Carson Street, Torrance, CA 90509.