Brain Stem Glioma Complicated by *Strongyloides stercoralis*

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ABSTRACT

The simultaneous occurrence of brain stem glioma and disseminated *Strongyloides stercoralis* infestation in an Ecuadorian male is reported. The unusual finding of *Strongyloides* larvae in the potassium hydroxide preparation for fungi and in the Gram stain for bacteria from a sputum during hemoptysis fortuitously established the diagnosis of disseminated strongyloidiasis. Of interest was the demonstration of various stages of *Strongyloides* in the sputum during treatment. *Strongyloides* infestation must be assiduously searched for in susceptible patients who are, or who are to be, immunosuppressed.

Introduction

Disseminated strongyloidiasis is being recognized with increasing frequency in patients who have impaired immunity, particularly cell-mediated immunity. Diagnosis by sputum examination has been reported rarely. This complication is reported in a patient who was treated with steroids for a brain stem glioma.

Case Report

A 32-year-old Ecuadorian engineering student presented to the emergency room with acute urinary retention. He gave a four months history of blurred and double vision, reduced hearing in the left ear, weakness of the left arm and leg, unsteady gait, and urinary hesitancy. He admitted to chronic abuse of alcohol. An evaluation in Ecuador had revealed normal cerebrospinal fluid, a technetium cisternogram consistent with convexity block, and parenchymal calcifications on computerized tomographic (CT) scan of the head. No ova or parasites were seen in the stool. Cysticercosis was diagnosed and a shunt recommended, but the patient came to the United States for a second opinion.

Physical examination revealed: horizontal and vertical nystagmus; weakness of the Vth, VIIth, IXth, and Xth cranial nerves on the right, and of the Vth, VIIth, and VIIIth cranial nerves on the left; spastic quadriparesis with greater involvement of the left side; dysmetria of the left upper extremity; and gait ataxia. There was a flaccid neurogenic bladder on cystometrogram.

The eosinophil count was 110 per cu mm; stool examination for ova and parasites was negative; no calcifications were seen on soft tissue x-rays of the neck or thigh or on CT scan of the head; and complement fixation tests for cysticercosis were negative. The diagnosis of cysticercosis was abandoned. Brain stem auditory evoked potentials, CT scan, posterior fossa angiography, and pneumoencephalography were consistent with an intrinsic lesion of the brain stem. The patient refused biopsy and, hence, dexamethasone and brain stem irradiation was started on the 26th hospital day for a presumed astrocytoma.

The cerebrospinal fluid findings on admission were opening pressure 310 mm of water, a protein concentration of 178 mg per dl, a glucose concentration of 84 mg per dl, and 162 leukocytes,—of which...
92 percent were mononucleated. Complete microbiological studies were negative. Since the patient gave a history of headache and malaise following the technetium-labelled albumin cisternogram in Ecuador, the since the cerebrospinal fluid examination in Ecuador was reported to have been negative, the cerebrospinal fluid abnormalities on admission were attributed to a chemical meningitis. On the 24th hospital day, cerebrospinal fluid obtained at the time of pneumoencephalography revealed no cells, a protein concentration of 55 mg per dl, and a glucose concentration of 67 mg per dl.

On the 48th hospital day, a red maculopapular rash was noted on the anterior chest wall, and the patient complained of intermittent abdominal pain.

On the 57th hospital day, the patient, whose neurological deficits had progressed only slightly, became febrile and lethargic, and had hemoptysis. Bilateral infiltrates were seen on chest x-ray. Sputum examinations for fungi and for bacteria were requested. The potassium hydroxide preparation for fungus revealed filariform larvae with the characteristic notched tail of *Strongyloides stercoralis*. Filariform larvae were also detected on the Gram stained preparation (figure 1). A stool specimen revealed numerous rhabditiform larvae but no filariform larvae (figure 2). Exactly 11.25 g of thiabendazole were administered over a period of four days. A temporary decrease of larvae in the sputum was followed by a heavy sputum parasite load of all stages: rhabditiform larvae and adult female worms (figure 3). Five days after treatment was begun, the patient became comatose, developed grand mal seizures, became hypotensive and anuric, and expired.

Autopsy revealed a pontine glioma confined to the brain stem and not producing obstruction of the ventricular system or significant increase in the volume of the posterior fossa. The meninges were cloudy. *S. stercoralis* ova, filariform larvae, and rhabditiform larvae were seen in the pharynx and in bronchi. Adults and larvae were seen in the duodenum. The remainder of the gastrointestinal tract was free of parasites.

**Comment**

There is good evidence for disseminated *S. stercoralis* infestation in this patient. He came from an endemic area and he was immunosuppressed. The initial negative stool specimen in Ecuador can be explained by the fact that very few larvae are shed in the chronic state, and multiple fresh stool examinations may be necessary to make the diagnosis. Non-specific abdominal pain and all degrees of intestinal hypomotility may occur at the onset of dissemination owing to invasion of the intestinal mucosa by filariform larvae carrying intestinal flora with them. This patient had neither diarrhea nor diarrhea alternating with constipation, as has been described. The striking red maculopapular rash on the anterior chest wall was not biopsied, but a similar rash has been reported once previously. It may be due to the migration of filariform larvae through cutaneous capillaries, and its recognition may allow the diagnosis of dissemination to be made in its early stages. At autopsy two weeks after the rash was noted, it was petechial. The presence of filariform and rhabditiform larvae, ova,
FIGURE 2. Rhabditiform larvae (second stage larvae) of *Strongyloides stercoralis* from stool. Wet preparation, × 50.

and adults in the lung documented the process of auto-infestation.

The neurological deterioration was consistent with disseminated strongyloidiasis. A few patients have been reported in whom free and degenerating larvae were seen throughout the cerebral hemispheres, cerebellum, spinal cord, meninges, and skull. Larvae occupied capillaries, and microinfarcts were observed. Larvae could not be demonstrated in the central nervous system, and meningitis may result. Bacteria are carried into the central nervous system, and meningitis may result. The patient died of septic shock, the usual mode of exodus.

Treatment for strongyloidiasis was reviewed by Meltzer et al in 1979. Oral administration of thiabendazole, 25 mg per kg, BID for two days, may be insufficient for disseminated disease. In one animal study, thiabendazole did not penetrate the blood brain barrier and could not be detected in cerebral tissues. The clinical effectiveness of other agents, mebendazole, levamisole, pyrvinium pamoate, and diethylcarbamazine, is unknown. Nor has the question of whether or not to discontinue treatment with steroids been settled.

A vigorous search for Strongyloides should include a duodenal aspiration, if necessary, and should be performed in all patients who are, or who are to be, immunosuppressed, and in whom a reasonable probability of infestation exists.

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References