
We report a case of respiratory failure caused by *Strongyloides stercoralis* in a patient with a renal transplant; the respiratory failure showed dramatic response to therapy with thiabendazole. The clinical aspects of infestation with *S. stercoralis* in the immunocompromised host are discussed, and features are demonstrated which may have significant implications concerning primary treatment and prophylaxis.

*Strongyloides stercoralis* is a helminth that can cause severe infestation in the immunosuppressed and malnourished host.1 We report a case of infestation with *S. stercoralis* with severe, diffuse pulmonary involvement causing marked hypoxemia in a patient with a renal transplant. Although severe pulmonary infestation with this parasite has previously been reported in the immunocompromised host,2,3 this case displays some unique features, including antemortem demonstration of the organism in pulmonary tissue, a dramatic response to therapy with thiabendazole, later requiring administration of pyrvinium pamoate to eradicate the organism, and the presence of an additional pulmonary infection.

### CASE REPORT

A 47-year-old Puerto Rican male recipient of a renal transplant was admitted to the University of Illinois Hospital on Feb 11, 1979, with chief complaints of malaise, abdominal pain, and diarrhea. Renal failure secondary to hypertension had been noted three years earlier. In August 1978, a guaiac test of stools was noted to be positive for blood on a screening physical examination done in preparation for a renal transplant. The stools also revealed larval forms of *S. stercoralis*, and the patient was treated with thiabendazole (25 mg/kg of body weight) daily for four days. One month after therapy, stools and duodenal aspirate were negative for organisms.

In November 1978, transplantation with a cadaveric kidney was successfully performed, and the patient received prednisone and azathioprine. He was readmitted one month later because of acute rejection, which was treated with...
anticoagulation, irradiation, and continued immunosuppression.

On Feb 11, 1979, the patient was seen with a two-day history of malaise, abdominal pain, and diarrhea. His temperature was 37.8°C (100.1°F), the blood pressure was 120/70 mm Hg, the pulse rate was 120 beats per minute, and the respiration rate was 20/min. The findings on examination of the lungs, heart, and abdomen were normal. A guaiac test of the stools was positive for blood. Laboratory data included a hematocrit reading of 30 percent, a white blood cell count of 4,600/cu mm, with no eosinophils, and a serum creatinine level of 0.9 mg/100 ml. A chest roentgenogram showed no infiltrates, and analysis of arterial blood gas levels revealed a pH of 7.56, an arterial carbon dioxide tension (PaCO₂) of 36 mm Hg, and an arterial oxygen pressure (PaO₂) of 49 mm Hg.

Lower gastrointestinal bleeding developed, and subsequent colonoscopic examination showed diffusely edematous and friable mucosa. Specimens of stool, gastric aspirate, and sputum revealed rhabdoid and filariform larvae diagnostic of S. stercoralis.

Dyspnea on slight exertion was noted on the second day of hospitalization, and the chest roentgenogram showed a diffuse nodular infiltrate (Fig. 1). Therapy with thiabendazole (25 mg/kg), ticarcillin, and gentamicin was begun. On the third day of hospitalization, bronchoscopic examination was performed, and both bronchial brushings and transbronchial biopsy revealed larvae of S. stercoralis (Fig. 2). There was no evidence for other organisms.

Over the next five days the malaise and diarrhea resolved, the chest roentgenogram showed complete resolution of the infiltrates, and arterial oxygenation greatly improved (pH, 7.48; PaCO₂, 27 mm Hg; and PaO₂, 78 mm Hg). By the 11th day of hospitalization, larvae were absent from stool and sputum; and therapy with antibiotics, including thiabendazole, was discontinued. Later on the 11th day of hospitalization, the patient developed an upper gastrointestinal hemorrhage which required laparotomy and oversewing of a duodenal ulcer. Two days following surgery, cultures of blood were positive for Enterobacter cloacae, and therapy with amikacin was given for seven days.

On the 15th day of hospitalization, larval forms of S. stercoralis were again found in the gastric fluid and sputum, and therapy with thiabendazole was resumed for four days. Larvae in the sputum persisted, and administration of pyrvinium pamoate (25 mg daily) was begun and continued for six days. Thereafter, organisms were absent from the stool and the sputum.

On the 20th day of hospitalization, the patient developed a fever and a diffuse patchy infiltrate in the chest. Titers for cytomegalovirus during the convalescent phase were reported to show a fourfold increase compared to the acute phase of the illness. Because of severe respiratory distress and hypoxemia, the patient was intubated. Bacteremias with Bacteroides fragilis and E. cloacae was discovered, and laparotomy done on the 27th day of hospitalization revealed the source to be an abscess of the rectus sheath. On the 31st day of hospitalization, bronchoscopic examination and transbronchial biopsy were repeated but yielded no diagnosis. Severe hypoxemia persisted, and the patient died on the 36th day of hospitalization.

Autopsy revealed acute and chronic bronchopneumonia, with focal organization; and postmortem viral cultures of the lung grew cytomegalovirus. There was no histologic or cultural evidence of other organisms in the lung or other tissues.
DISCUSSION

*Strongyloides stercoralis* is an intestinal nematode which has a life cycle enabling it to perpetuate itself in the human host. The filariform larvae are able to penetrate the skin, enter the blood stream, and then migrate to the lungs. There, the larvae travel up the tracheobronchial tree to the glottis, are swallowed, and reach the small intestine, where they develop into rhabdoid larvae that are capable of causing two types of severe clinical infestation, hyperinfection and disseminated disease. Hyperinfection is an augmentation of the usual life cycle in which large numbers of the infective filariform larvae are produced. The case presented is an example of hyperinfection in that there was no demonstrable dissemination or involvement of organs outside of the normal life cycle.

Infestation with *S. stercoralis* in the immunosuppressed host has been noted to involve the lungs. Diffuse pulmonary infiltrates and acute respiratory failure have been described, but pulmonary involvement is generally mild. Antemortem diagnosis has been made by examination of sputum, transtracheal aspiration, and cytologic examination of bronchial washings obtained by bronchoscopic examination. The patient described in this report demonstrates that *S. stercoralis* is capable of causing severe respiratory infection with marked hypoxemia. The presentation with diarrhea and numerous organisms in the feces, gastric aspirate, and sputum strongly suggested the diagnosis, but a bronchoscopic procedure was done to evaluate the patient for an additional pathogen. In the process, organisms were demonstrated in tissue by transbronchial biopsy (Fig 2b). To our knowledge, this represents the first case in which *S. stercoralis* has been demonstrated histopathologically in pulmonary tissue ante mortem.

The efficacy of thiabendazole in acute pulmonary infection due to *S. stercoralis* is clearly demonstrated in this patient by the marked clinical improvement with therapy. Thiabendazole is considered the drug of choice because, unlike other common anthelminthic drugs, it is absorbed well and acts within the tissues. While Scowden et al have recently demonstrated the efficacy of this drug, most previous series and reports of severe, diffuse pulmonary involvement have demonstrated a rather poor response to therapy with thiabendazole. The dramatic response in our patient would appear to confirm its role as the primary drug in treating hyperinfection with *S. stercoralis*; however, in spite of the resolution of clinical infection and the initial clearing of the organism, we again noted infestation that persisted in spite of therapy with thiabendazole. Therapy with pyrvinium pamoate, a nonabsorbable anthelminthic drug, eliminated the organism from stool and sputum. This drug has been shown to be effective in nonsystemic disease, and its use in this patient demonstrates its effectiveness as an adjunct to thiabendazole in systemic infection.

For prophylaxis against systemic strongyloidiasis, recent reports have suggested careful analysis of stool and upper small-bowel aspirate for the detection of organisms in all patients who are to undergo immunosuppression. In the present case, pretransplant infestation was appropriately identified and treated, and follow-up specimens of duodenal aspirate and stool were negative. Nevertheless, recurrent infestation with *S. stercoralis* with diffuse pulmonary involvement occurred. Therefore, the presently recommended prophylactic regimen of follow-up examination and short-term treatment may be inadequate to prevent recurrent infection. Prophylaxis with thiabendazole (25 mg/kg daily for two days each month) or a less toxic nonabsorbable drug like pyrvinium pamoate may be indicated on a continuous basis to effectively prevent the development of active infection in a patient with recent infestation if he is to undergo immunosuppression. Perhaps such a regimen would benefit the immunosuppressed host with a history of infestation with *S. stercoralis* in the remote past or even in those from endemic areas. Although the incidence of infestation with *S. stercoralis* is low, a prospective randomized trial examining prophylactic regimens conducted in a center where there is a population with endemic infestation may be indicated.

The case presented also demonstrates the presence of concurrent and sequential infections, which is a relatively common feature in the immunocompromised host. Gram-negative septicemia and a second diffuse pulmonary infection, presumably due to cytomegalovirus, were found. This confirms the need for a vigorous diagnostic approach to suspected infection in the immunocompromised host, including invasive procedures for biopsy, even when one infection has been identified and is under treatment.

REFERENCES