Case Report

Acute Respiratory Distress Syndrome due to *Strongyloides stercoralis* in Non-Hodgkin's Lymphoma

Ajit Vigg, S. Mantri, Vijay Anand P. Reddy[1] and V. Biyani[1]

Departments of Chest Diseases and Oncology, Apollo Hospitals, Hyderabad, India

ABSTRACT

Strongyloides stercoralis is a nematode endemic in tropical and subtropical regions. In immunocompetent subjects, pulmonary disease caused by the parasite is unremarkable but the same can be life threatening in immunocompromised subjects. Though described in literature it is rarely seen in Indian subjects. We report a patient with ARDS due to *Strongyloides stercoralis* complicating non-Hodgkin's lymphoma with neutropenia. **[Indian J Chest Dis Allied Sci 2006; 48: 67-69]**

Key words: ARDS, *Strongyloides stercoralis,* Hyperinfection, Non-Hodgkin's lymphoma.

INTRODUCTION

Strongyloides stercoralis is a soil living nematode, endemic in tropical and sub-tropical regions as well as in Southern United States and parts of Europe. Sixty million people are estimated to be infected worldwide. It is mainly encountered in areas where sanitary facilities are poor or in moist environments, such as in mines or tunnels. This nematode is capable of auto-infection. It causes minimal clinical manifestations in an immunocompetent host. In an immunocompromised host, hyperinfection may be associated with gram-negative infection and multiorgan involvement can occur. In immunocompetent subjects, pulmonary disease is unremarkable; however *S. stercoralis* infection though rare can be life threatening in immuno-compromised subjects[1]. It is associated with neoplastic diseases[2], such as Hodgkin's disease and other lymphomas[3-5], leukemias, non-malignant conditions treated with corticosteroids,[6] *e.g.* organ transplantation, in severe malnutrition and alcoholism. Even though infection is common in tropical regions, hyperinfection with *S. stercoralis* leading to ARDS is rare[7]. We report one such patient of non-Hodgkin's lymphoma with chemotherapy-induced neutropenia with hyper-infection, ARDS and multiorgan failure.

CASE REPORT

A 65-year-old man was diagnosed as a case of non-Hodgkin's lymphoma stage IIA diffuse large cell type. Six days after the second cycle of chemotherapy with cyclophosphamide 75 gm / m^2, adriamycin 50 mg / m^2, vincristine 1.4 mg / m^2, prednisolone 80 mg / day (5 days), the patient developed febrile neutropenia with a total white cell count of 800 / mm^3. Absolute neutrophil count was 560 / mm^3. He was given granulocyte colony stimulating factor (G-CSF) and broad-spectrum antibiotics comprising of I.V. piperacillin and tazobactam, and amikacin. Stool examination was essentially normal. Following recovery of leukocyte counts, he developed productive cough, progressive dyspnoea and high-grade fever. Clinically, he was tachypnoeic with cyanosis. Examination of chest revealed bilateral diffuse coarse crackles. Sputum culture showed growth of *Staphylococcus aureus*. Blood culture was sterile. He was started on teicoplanin 400 mg once a day according to the sensitivity pattern. The respiratory failure worsened for which he required mechanical ventilation. Roentgenogram of chest showed pulmonary infiltrates and bilateral alveolar shadows consistent with ARDS (Figure 1). Endotracheal tube aspirate for gram stain, fungal stain and culture showed numerous larvae of *S. stercoralis* (Figure 2). Fungal stain was negative while the endotracheal tube aspirate on culture grew *Staphylococcus aureus.* He also had a maculopapular rash on both thighs and over the abdominal wall. Skin biopsy showed larvae of *S. stercoralis*[3] (Figure 3). Patient was treated with ivermectin (6 mg per day) for three days and albendazole (400 mg per day) for seven days.

Patient's general condition deteriorated and he developed sepsis with septic shock and disseminated intravascular coagulopathy requiring inotropic support. Patient did not improve and succumbed to his illness on 10th day of hospitalisation.

[Received: November 1, 2004; accepted: December 29, 2004]

Correspondence and reprint requests: Dr Ajit Vigg, Head, Department of Chest Diseases, Apollo Hospitals, Jubilee Hills, Hyderabad-500033; India; Tele.: 91-040-23607777, Extn 3040; Fax: 91-040-23608050; E-mail: drajitvigg@yahoo.com.

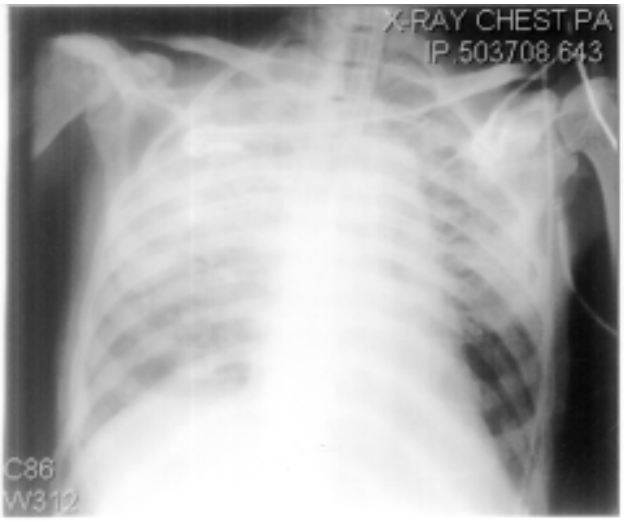

Figure 1. Chest roentgenogram showing bilateral alveolar opacities consistent with ARDS.

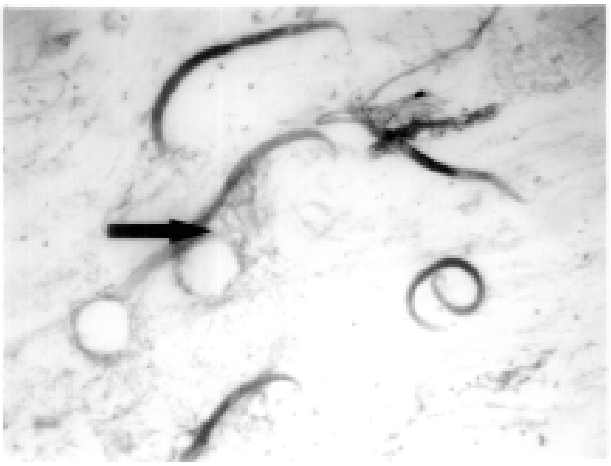

Figure 2. Endotracheal secretions showing larvae of *S. stercoralis* (H & E × 40).

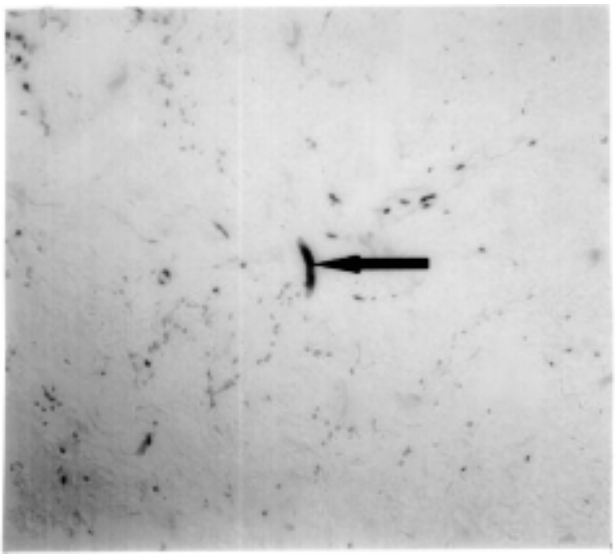

Figure 3. Histopathology of skin showing dermis with larva of *S. stercoralis* (H & E × 40).

DISCUSSION

The ova of the female *S. stercoralis* hatch into rhabditiform (non migratory) larvae that are capable of maturing into non infectious adults or moulding into filariform (infective) larvae. Initial invasion occurs when the patient's skin is exposed to contaminated soil for faeces. The filariform larvae penetrate the dermis and migrate through the venous system to the lungs, ascend to the trachea, are swallowed into the digestive tract and infect the small intestinal mucosa. Some larvae, however, re-enter the blood stream through the bowel wall and migrate through lungs bypassing the soil cycle. This ability for autoinfection implies that infestation can be life long and extremely heavy. Massive autoinfection leads to disseminated strongyloidiasis, the so called hyperinfection syndrome, which can result in severe pulmonary disease[8,9] with associated septicemia.

Usually symptoms of pulmonary strongyloidiasis include cough, dyspnoea, wheezing and haemoptysis; peripheral eosinophilia is common. However, in immunocompromised subject, the absolute eosinophil counts can be low as was in the present case. In most cases, hyperinfection in immunocompromised hosts with overwhelming infestation with involvement of lungs, colon, liver and other organs is frequently associated with ARDS[10]. In one series[9], the incidence of ARDS was 45 percent. The mechanism of development of ARDS in such a condition is said to be lung injury caused by direct damage by parasites or endotoxin mediated injury from associated bacterial sepsis[10]. Additionally, intense inflammatory reaction triggered by intrapulmonary destruction of the large number of larvae following administration of antihelminthic agents can also lead to ARDS[11]. Patients with HTLV-1 seropositivity have been shown to be more likely to develop hyperinfective strongyloidiasis[12,13]. This is postulated to be due to even lower IgE levels in these patients than seen in HTLV-1 negative patients. In the present case, development of ARDS was possibly due to a combination of heavy parasitic load and superadded bacterial infection of *Staphylococcus aureus.*

In conclusion, hyperinfection with disseminated strongyloidiasis should be considered in ARDS developing in an immunocompromised host.

ACKNOWLEDGEMENTS

We would like to thank the management of Apollo Hospital for all the necessary co-operation and Department of Pathology for their support.

REFERENCES

1. Genta RM. Global prevalence of strongyloidosis: critical review with epidemiologic insights in to the prevention of disseminated disease. *J Infect Dis* 1989; 11: 755-67.

2. Powell RM, Moss JP, Nagar D. Strongyloidiasis in immunosuppressed hosts. *Arch Intern Med* 1980; 140: 1061-3.
3. Aydin H, Doppl W, Battmann A. Opportunistic *Strongyloides stercoralis* hyperinfection in lymphoma patients undergoing chemotherapy or radiotherapy: report of a case and review of literature. *Acta Oncol* 1994; 33: 78-80.
4. Genta RM, Miles P, Fields R. Opportunistic *Strongyloides stercoralis* infection in lymphoma patients: report of a case and review of literature. *Cancer* 1989; 63: 1407-11.
5. Sandland JT, Ranglmann W, Flynn PM. *Strongyloides stercoralis* infection mimicking relapse in a child with small non cleared cell lymphoma. *Am J Clin Oncol* 1997; 20: 215-6.
6. Cruz T, Reboucas G, Rocha H. Fatal strongyloidiasis in patients receiving corticosteroids. *N Engl J Med* 1996; 275: 1093-6.
7. Jindal SK, Aggarwal AN, Gupta D. Adult respiratory distress syndrome in the tropics. *Clin Chest Med* 2002; 23: 445-55.
8. Woodring JH, Halfhill H, Reed JC. Pulmonary strongyloidiasis: clinical and imaging features. *Am J Roentgenol* 1994; 162: 573-42.
9. Woodring JH, Halfhill H, Bergev R, Reed JC, Moser N. Clinical and imaging features of pulmonary strongyloidiasis. *South Med J* 1996; 89: 10-19.
10. Cook GA, Rodriguez H, Silva H, Rodriguez-tube B, Bohorquez de Rodriguez H. Adult respiratory distress secondary to strongyloidiasis. *Chest* 1987; 92: 1115-6.
11. Thompson (Jr), Berger R. Fatal adult respiratory distress syndrome following successful treatment of pulmonary strongyloidiasis. *Chest* 1991; 99: 772-4.
12. Adedayo O, Grell G, Belcot P. Hyperinfective strongyloidiasis in the medical ward: review of 27 cases. *South Med J* 2002; 95: 711-6.
13. Gotuzzo E, Terashima A, Alvarex H, *et al. Strongyloides stercoralis* hyperinfection associated with human T-cell lymphotropic virus type-1 infection in Peru. *Am J Trop Med Hyg* 1999; 60: 146-9.