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Strongyloides stercoralis induced bilateral blood stained pleural effusion in patient with recurrent Non-Hodgkin lymphoma

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Abstract. Infections and malignancies are common causes of pleural effusion. Among infectious causes, hyperinfection syndrome of *Strongyloides stercoralis* may occur in immunosuppressive patient. A 62-year-old man, known case of Non-Hodgkin lymphoma (NHL) was presented with recurrent NHL stage IV and had undergone salvage chemotherapy. Patient subsequently developed pneumonia with bilateral pleural effusion and ascites. We reported rhabditiform larvae of *S. stercoralis* in pleural fluid of both lungs without infiltration by lymphoma cells. Stool for microscopic examination also revealed rhabditiform larvae of *S. stercoralis*. This patient was a known case of NHL receiving chemotherapy resulting in immunosuppression state. Although *S. stercoralis* infection is not very common compared to other parasitic infections, it is common in immunosuppressive patients and may present with hyperinfection. Therefore, awareness of this parasite should be kept in mind in immunosuppressive patients.

INTRODUCTION

Infections and malignancies are common causes of pleural effusion and malignancy is a common cause for blood stained pleural effusion. Among the infectious causes of pleural effusion, parasitic infection with hyperinfection syndrome of *Strongyloides stercoralis* may occur in immunosuppressive patient (Meyers *et al*., 2000). We found rhabditidiform larvae of *S. stercoralis* in the cytology smear of blood stained pleural fluid in this patient with recurrent NHL who was being treated with chemotherapy and corticosteroid. Corticosteroid use is associated with a two- to three-fold increase in the risk of being infected by *S. stercoralis* (Armignacco *et al*., 1989).

Case report

A 62-year-old Malay man, a known case of NHL (Diffuse large B cell lymphoma) was admitted with recurrent cervical lymphadenopathy and difficulty in respiration. He was diagnosed as NHL (Diffuse large B cell lymphoma) from tonsil and cervical lymph node specimens' one year earlier (Figure 1) and had completed chemotherapy regimen of CHOP (Cyclophosphamide, Adriamycin, Vincristine and Prednisolone) for 6 cycles. Six months after completing chemotherapy, the patient redeveloped cervical lymphadenopathy.

On admission, patient was febrile and dyspnoiec. Bilateral crepitations on both lung fields were noted. Full blood picture revealed Hb – 10.7 g/dl, WBC – 6370/cu

mm, platelet – 252,000/ml, neutrophil – 43.2%, lymphocytes – 34.7%, monocytes – 6.4%, eosinophil – 15.4% and basophil – 03%. Repeated excisional biopsy of cervical lymph node revealed NHL (Diffuse large B cell lymphoma). Patient subsequently developed respiratory infection with pneumonia. He was diagnosed as recurrent NHL stage IV with sepsis and planned for salvage chemotherapy of R-DHAC (Rituximab + Dexamethazone, high dose Cytarabine Arabinoside, Cisplatin). Subsequently, patient developed bilateral pleural effusion and ascites. Bilateral pleural effusion became massive and pleurocentesis was done to relieve dyspnoea. Pleural fluid was sent for cytology to exclude lymphoma infiltration to pleural cavity.

Routine examination of pleural fluid was blood stained. It was centrifuged and the sediment was made into two smears, alcohol fixed smear was stained with Papanicoloau stain and air dried smear was stained with May-Grunwald Giemsa stain. Smears revealed few mesothelial cells, few lymphocytes, neutrophils and eosinophils. Levels of eosinophils were more than that of usual inflammatory smears. However, no evidence of lymphoma cell infiltration was seen. Accidentally, two coiled-shaped rhabditidiform larvae of *S. stercoralis* parasites were found in Giemsa stained smear (Figure 2). Stool for routine and microscopic examination also revealed many rhabditidiform larvae of *S. stercoralis*.

DISCUSSION

Strongyloidiasis is a nematode infection in humans with a tendency towards chronic persistent infection, special characteristic features of autoinfection, hyperinfection involving pulmonary and gastrointestinal systems, and disseminated infection involving other organs (Liu & Weller, 1993; Siddiqui & Berk, 2001; Roman *et al*., 2003; Concha *et al*., 2005).

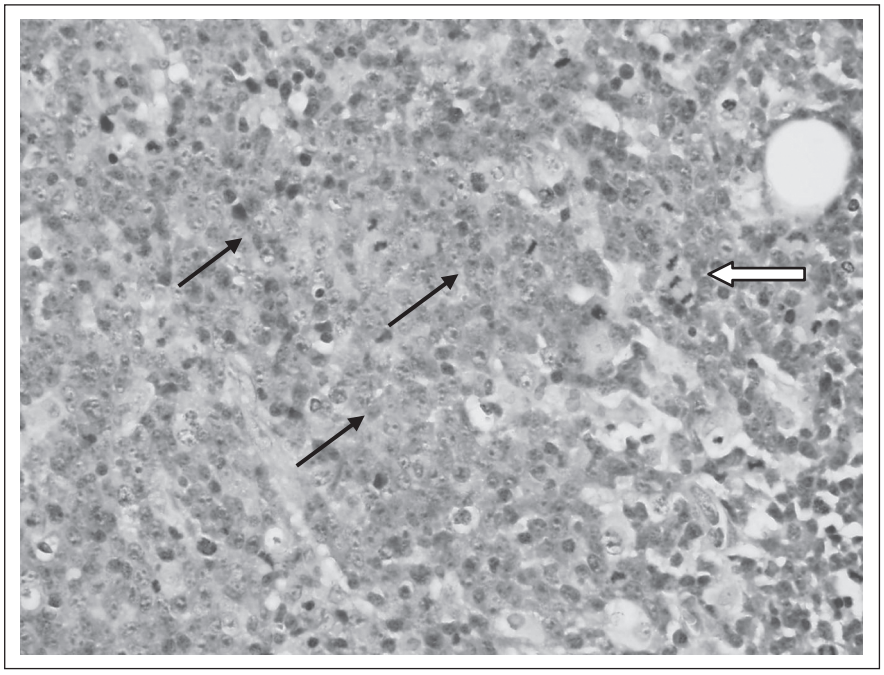

Figure 1. High power view of cervical lymph node showed Non-Hodgkin Lymphoma (Diffuse Large B Cell Lymphoma) (40 X Haematoxylin & Eosin stain). Lymphoma predispose this patient to the risk of hyperinfection with *S. stercoralis*

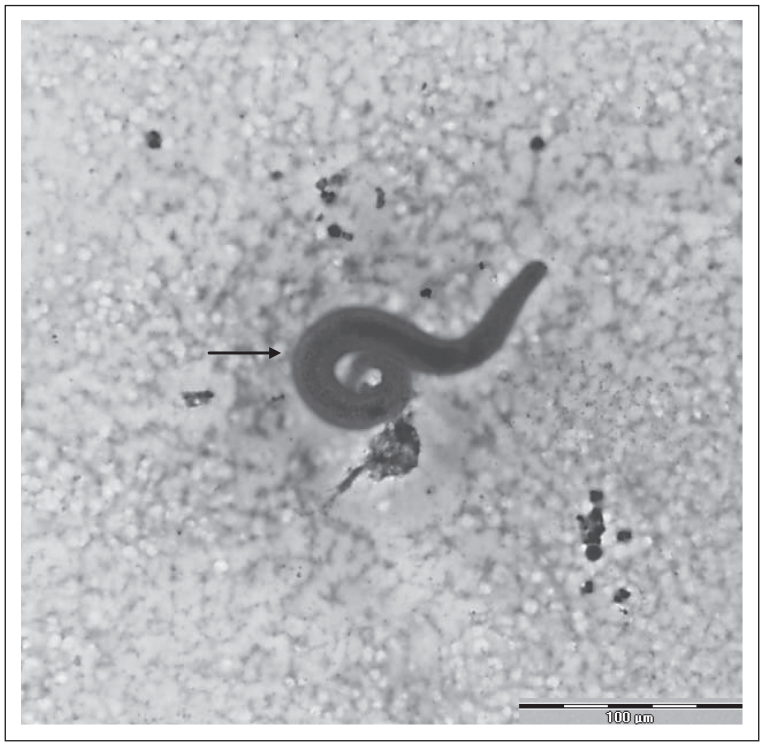

Figure 2. Giemsa stained smear of pleural fluid showed; A rhabditiform larva of *Strongyloides stercoralis* in 20X

Infection begins when filariform larvae penetrate the skin, migrate through the circulation to the capillary bed of the lung and penetrate the capillary wall to settle in the alveoli. The larvae subsequently ascend the tracheo-bronchial tree to the larynx where they are swallowed and carried to the intestines, particularly the upper small intestine. The larvae mature into adult females that deposit eggs in the intestinal mucosa. The eggs hatch into rhabditiform larvae, which are excreted in the feces and become filariform in the soil, before continuing another life cycle. In some patients, however, the larvae remain in the intestines, metamorphose into infective filariform larvae and penetrate the intestinal mucosa. These larvae traverse the lymphatics to the thoracic duct, then undergo lympho-hematogeneous dissemination to the lung and repeat the life cycle within the host for several years. Many such infected hosts showed skin lesions or gastrointestinal and/or respiratory symptoms (Meyers *et al*., 2000).

Immunocompromised hosts, particularly people on prolonged corticosteroid therapy, can have very high levels of disseminated worms due to uncontrolled autoinfection (Cotran *et al.*, 2004). Systemic strongyloidiasis occurs in association with immune suppression (intercurrent disease, HTLV-1 infection, corticosteroid treatment) and is rapidly fatal unless diagnosed and promptly treated (Edwards *et al.*, 1995). Patients who have hyperinfection syndrome usually present with fever, gastrointestinal symptoms, dyspnoea, wheezing, hemoptysis, cough, and weakness (Markell *et al.*, 1999).

This patient was a known case of NHL receiving chemotherapy with corticosteroid and dexamethazone resulting in immunosuppression. Due to immunosuppression, this patient had developed hyperinfection of *S. stercoralis*. Therefore, rhabditiform larvae from the intestine had spread via venous or lymphatic circulation, before they change into filariform larvae, to the pulmonary vasculature and penetrate

into pleural cavity giving rise to blood stained pleural effusion. Levels of eosinophils in pleural fluid are greater in numbers than the usual inflammatory smear. Eosinophilic pleural effusion revealing *S. stercoralis* is an uncommon presentation and usually seen in immunocompromised patients (Siddiqui & Berk, 2001).

Strongyloides stercoralis infection is not very common as compared to other nematode infestation such as *Ascaris lumbricoides* in this setting. According to our local data from the Department of Microbiology and Parasitology, School of Medical Sciences, Universiti Sains Malaysia, the yield of *S. stercoralis* larvae in stool specimens by microscopic examination is only 0.1 to 0.3% per year. However, detection of *Strongyloides* infestation in immunosuppressive patient has been noted in our clinical setting and it can present with hyperinfection syndrome resulting in severe complications or death. Therefore, awareness of *S. stercoralis* infection should be kept in mind in immunosuppressive patients.

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