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Disseminated strongyloidiasis in an immunocompromised host: A case report

Nurul Suhaiza Hassanudin[1], Zubaidah Abdul Wahab[1], Khalid Ibrahim[2], Fadzilah Mohd Nor[3,4*]

[1]*Microbiology Unit, Department of Pathology, Hospital Sg. Buloh, 47100, Sg. Buloh, Selangor, Malaysia*

[2]*Director Office, Hospital Sg. Buloh, 47100, Sg. Buloh, Selangor, Malaysia*

[3]*Microbiology Discipline, Faculty of Medicine, Universiti Teknologi MARA, Sg. Buloh Campus, Jalan Hospital, 47000, Sg. Buloh, Selangor, Malaysia*

[4]*Integrative Pharmacogenomics Institute (iPROMISE), Level 7, FF3, Universiti Teknologi MARA, Puncak Alam Campus, 42300, Bandar Puncak Alam, Selangor, Malaysia*

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ABSTRACT

Infections caused by *Strongyloides stercoralis* (*S. stercoralis*) in human are generally asymptomatic, however in immunocompromised individual, hyperinfection may develop with dissemination of larvae to extra-intestinal organs. The diagnosis could be easily missed due to asymptomatic presentation and insufficient exposure towards the infection itself, which may lead to low index of suspicion as a consequence. In this report, a case of a Malaysian male with underlying diabetes mellitus, hypertension, cerebrovascular accident, bullous pemphigus and syndrome of inappropriate antidiuretic hormone secretion who initially complained of generalized body weakness and poor appetite without any history suggestive of sepsis is presented. However, he developed septicemic shock later, and *S. stercoralis* larvae was incidentally found in the tracheal aspirate that was sent to look for acid fast bacilli. Regardless of aggressive resuscitation, the patient succumbed due to pulmonary hemorrhage and acute respiratory distress syndrome. It was revealed that the current case has alarmed us via incidental finding of *S. stercoralis* larvae in the tracheal aspirate, indicating that the importance of the disease should be emphasized in certain parts of the world and population respectively.

1. Introduction

Strongyloides stercoralis (*S. stercoralis*) is a nematode belonging to the class of Secernentasida, order of Rhabditorida, family of Strongylidae and the genus of *Strongyloides*. *Strongyloides* infection is endemic in humid tropical regions including Africa, Southeast Asia, and Latin America [1,2]. *Strongyloides fuelleborni* which is mainly found in Africa and Papua New Guinea [3], is another important causative agent of human strongyloidiasis, but in a lesser frequency in contrast to *S. stercoralis*.

Strongyloidiasis begins with the penetration of a susceptible host by filariform larva (infective stage) into the skin. The larva enters the venous or lymphatic channels, and is subsequently transported to the lungs, where it migrates to the trachea. As it matures, it will then be swallowed into the gastrointestinal tract. A female parasite then lodges in the lamina propria of the duodenum and proximal jejunum where it lays egg. The eggs hatch into rhabditiform larvae, where they migrate into the intestinal lumen and go into either one of the two pathways – autoinfection or excreted to the external environment (free-living stage).

In general, strongyloidiasis can cause acute infection, autoinfection and chronic infection, hyperinfection and dissemination syndrome. However, majority of human infections are manifested themselves as chronic strongyloidiasis. The chronic state is probably maintained by a relatively low and stable number of adult worms that reside in the intestine and is survived by a means of well-regulated auto-infection [4]. Once the host immunity is compromised, the rate of autoinfection and

*Corresponding author: Fadzilah Mohd Nor, Microbiology Discipline, Faculty of Medicine, Universiti Teknologi MARA, Sg. Buloh Campus, Jalan Hospital, 47000, Sg. Buloh, Selangor, Malaysia.
Tel: +603 6126 7436
Fax: +603 6126 7073
E-mail: falah1911@gmail.com (F.M. Nor).

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population of adult worm increases, hence hyperinfection occurs. Ultimately, the larvae migrate extra-intestinally and lodge themselves into meningeal spaces, brain, liver, lymph nodes, kidney, cutaneous and subcutaneous tissues. This migration and penetration into other organs can lead to inflammation and complicate further with hemorrhage.

2. Case report

A 56-year-old man was presented with generalized body weakness and poor appetite two days prior to admission. He denied any history of fever, cough, shortness of breath, headache, and blurring of vision. There were no abdominal pain, urinary and bowel symptoms associated with the main complaints. Past medical history revealed that the patient has been diagnosed with diabetes mellitus and hypertension for more than ten years, and has suffered from cerebrovascular accident (CVA) for the past seven years. However, the patient has been able to ambulate independently. He had recently diagnosed as having bullous pemphigus and syndrome of inappropriate antidiuretic hormone secretion (SIADH) four months prior to the current admission. In relation to that, he was on multiple drugs for his underlying problems including oral prednisolone 30 mg once daily (OD) for the bullous pemphigus. He was a former government servant and had no history of recent travel.

Upon physical examination he was alert, conscious but appeared dehydrated. The vital signs were normal with a temperature of 37 °C, blood pressure of 100/70 mmHg and pulse rate of 80 beats/minute with regular rhythm. There were no oral thrush, abnormal cutaneous lesions and cervical lymphadenopathy present. A cardiovascular system and fundoscopy examinations were unremarkable. A central nervous system (CNS) examination was also normal except for lower muscle power that revealed at 4/5 for both upper and lower limbs. Respiratory examination revealed crepitation at the right lower zone bilaterally, with no dullness on percussion. Tenderness at the epigastric region was noted per abdomen. A provisional diagnosis of acute kidney injury secondary to dehydration with uncontrolled diabetes mellitus was clinically suspected.

Initial blood investigations showed hemoglobin of 7.6 g/dL, total white cell count of 10×10^9/L without eosinophilia, and normal platelet count at 200×10^9/L. Biochemically, there were evidence of hyperglycemia (16 mmol/L), impaired renal function with electrolyte imbalances; hyponatremia (115 mmol/L) and hypokalemia (2.6 mmol/L). Liver function profile was unremarkable except for low albumin level (13 g/L). The C-reactive protein (CRP) was elevated at 5.90 mg/dL (0.01–0.82 mg/dL).

Chest radiograph was done and it revealed the consolidation of the right lower zone with patchy opacity over the left lower zone of the lung. Blood for bacterial culture and sensitivity was collected on day 3 of admission. Empirical intravenous (IV) cefepime 1 g 8 hourly was commenced immediately after that while waiting for further identification of the organism. After 48 h of incubation, carbapenem resistant *Klebsiella pneumoniae* (CRE) was isolated. The patient was deescalated to IV polymyxin E 4.5 µ twice daily and imipenem 500 mg 6 hourly. Full blood picture was collected too, and features suggestive of iron deficiency anemia with neutrophilia likely due to infection are detected.

In spite of having appropriate antibiotics and improvement of his repeat renal profile, except for persistent hyponatremia, the patient developed septicemic shock on day 9 of admission. The respiratory examination revealed generalized rhonchi with prolonged expiratory phase. At the same time, tracheal aspirate was sent to look for acid fast bacilli (AFB) (immunofluorescence; Auramine O staining QBC Diagnostics, Inc.) to exclude the possibility of pulmonary tuberculosis. Upon reviewing the AFB smear, presence of apple-green fluorescent larvae (Figure 1) was noted. Following that, a wet smear (Figure 2) was performed and live helminthic larvae were seen with the esophageal lengths of approximately half of the body, while the ends were pointed with some demonstrated hooked or notched appearance. Trichrome and Giemsa staining (Figure 3) were also carried out to verify the wet smear findings. This incidental finding was informed to the clinician, thus a combination of albendazole (400 mg oral, twice daily) with ivermectin (200 µg/kg oral, daily) were administered. Unfortunately, regardless of aggressive resuscitation, the patient succumbed to illness on day 13 of admission due to pulmonary hemorrhage and acute respiratory distress syndrome (ARDS).

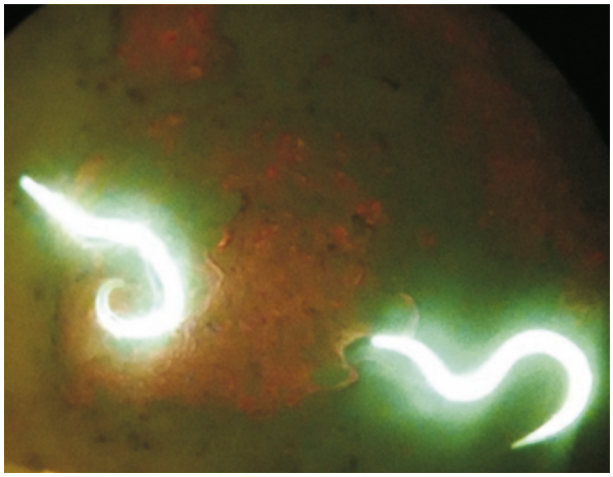

Figure 1. Presence of apple-green fluorescent larvae using Auramine-O-stain.

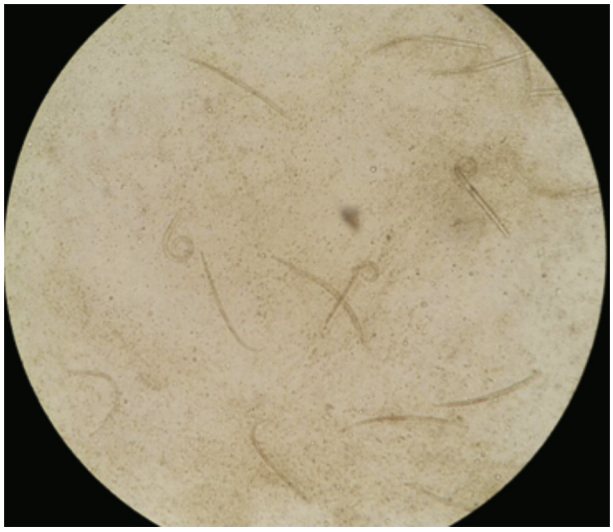

Figure 2. Larvae of *S. stercoralis* in unstained wet mount.

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