中文題目：糞小桿線蟲感染導致急性呼吸窘迫症候群

英文題目：Acute Respiratory Distress Syndrome Related to Strongyloides stercoralis Hyperinfection

作　　者：吳宗勳[1#]、蔡明儒[1#]、蔡坤寶[2]、陳煌麒[3]、莊淑惠[4]、黃明賢[5,6]、黃吉志[5,7]

服務單位：高雄市立小港醫院（委託高雄醫學大學經營）[2]病理科 [3]內科 [4]護理部
高雄醫學大學附設中和紀念醫院 [1]內科部 [5]內科部胸腔內科
高雄醫學大學醫學院 [6]醫學系 [7]呼吸治療學系 # Equal contribution

摘　　要：

Case Presentation

A 72-year-old man with a history of chronic obstructive pulmonary disease, chronic gouty arthritis and chronic kidney disease had lived at home with poor self care and hygiene. He did not receive regular medical follow-up, but he took medications from local pharmacy. He came to the emergency department of our hospital for general weakness, intermittent chest tightness and diarrhea for about a week, presenting with normal vital signs and cushingoid appearance. Laboratory examination showed pyuria but no leukocytosis or elevation in cardiac enzymes. Besides, impaired liver and renal functions were also noted. He was admitted under the impression of urosepsis.

Unfortunately, his consciousness deteriorated on the fifth hospital day, and high C-reactive protein level, thrombocytopenia, and severe metabolic acidosis were noted. He was intubated for his respiratory failure. Empirical antibiotic treatment with cefpirome and metronidazole were prescribed for suspected intra-abdominal infection. He developed septic shock soon, requiring inotropic support. Because bilateral pulmonary infiltrates developed rapidly with the appearance compatible with acute respiratory distress syndrome (ARDS), antibiotic was adjusted to piperacillin/tazobactam 2 days later. Blood culture later yielded *Escherichia coli* and *Klebsiella pneumoniae*, which were both susceptible to piperacillin/tazobactam. Bronchoscopy was performed, which revealed coffee-ground discharge in posterior segment of left upper bronchi (LB2), and bronchioloalveolar lavage (BAL) cytology found filariform larvae of *Strongyloides stercoralis*. Ivermectin was administered for 2 days. Colonofiberoscopy, arranged for his diarrhea, revealed erosive lesions in transverse colon, and biopsy showed parasite-like tissue fragments with granulomatous reaction. He developed oliguric acute kidney injury with hyperkalemia and severe metabolic acidosis. Continuous veno-venous hemofiltration was started soon. However, profound shock developed few hours later, which was not reversed with full-dosed vasopressors and any aggressive treatment. He succumbed to his illness soon.

Discussion

Strongyloides stercoralis is a soil living nematode endemic in tropic and subtropic areas. It may cause hyperinfection in immunocompromised hosts with high mortality rate (up to 90%). Glucocorticoid treatment and human T-lymphotropic virus type 1 infection are the two conditions most specifically associated with hyperinfection.

There was no significant clue initially for diagnosing this patient as having *S. stercoralis* hyperinfection. Neither peripheral eosinophilia nor significant hemoptysis was noted. Bronchoscopy played a critical role to define the unexpected cause of his progressive pulmonary infiltrates. The coffee-ground discharge in the airway, which was attributed to damaged pulmonary vasculature by larvae, may provide a clue for diagnosis. The correct diagnosis was soon made by recognition of the worm in BAL cytology. For a septic patient with progressive pulmonary infiltrates, bronchoscopy may be necessary for defining the cause.

As in our case, patients with hyperinfection strongyloidiasis often develop ARDS and gram-negative septicemia. These were attributed to the migration of larvae from the gastrointestinal tract to the pulmonary system, carrying enteric bacteria on the surface of the migrating worms. Therefore, broad-spectrum antibiotic treatment in addition to antiparasitic therapy should be given.

In conclusion, hyperinfection strongyloidiasis should be considered as a cause of acute respiratory distress syndrome in immunocompromised patient, especially with the presence of chronic gastrointestinal symptoms.