Strongyloides Stercoralis Infection With Bloody Pericardial Effusion in a Non-Imunosuppressed Patient

Cha-Po Lai, MD, PhD; Yung-Hsiang Hsu, MD;*  
Ji-Hung Wang, MD; Chi-Mo Lin, MD, PhD

A 63-year-old Taiwan aboriginal male was admitted with exertional dyspnea, appetite loss and general fatigue. Echocardiography revealed moderate pericardial effusion and histological examination of the pericardiocentesis sample revealed an eosinophil-dominated bloody exudate. The larvae of Strongyloides stercoralis were detected in the pericardial specimen. After treatment with anti-nematodal agents, the eosinophilia decreased from 26% to 1% and the patient’s symptoms improved. This is a rare case of Strongyloides-induced bloody pericardial effusion in a non-immunosuppressed patient. (*Circ J 2002; 66: 613–614)

Key Words: Eosinophilia; Hyperinfection; Non-immunosuppression; Pericardial effusion; Strongyloidiasis

Bloody pericardial effusion is most commonly caused by tumor, tuberculosis, iatrogenically or post myocardial infarction! Infection by the nematode, Strongyloides stercoralis (S. stercoralis), whose primary hosts are human and some nonhuman primates; is generally a benign cutaneous and/or gastrointestinal infection, but hyperinfection has been observed with increasing frequency in patients with impaired immunity.3–5 We report a case of Strongyloides-induced bloody pericardial effusion in a non-immunosuppressed patient.

Case Report

A 63-year-old Taiwan aboriginal male was admitted to hospital in August for the evaluation and treatment of his exertional dyspnea, appetite loss and general fatigue. He had been a hunter for about 30 years. The exertional dyspnea and chest discomfort had been apparent since March of that year. Ischemic heart disease associated with heart failure had been suspected and treated at another hospital, but the symptoms did not improve. Appetite loss, dysphagia and abdominal distension gradually appeared from June of the same year, and liver cirrhosis with ascites was suspected by the local physician.

The patient was 165 cm in height and weighed 66 kg; the pulse rate of 80 beats/min was regular and blood pressure was 130/98 mmHg. There was a revealed pan-systolic murmur (2/6) at the apex and moist rales were heard on both sides of the lower back. ECG revealed sinus rhythm, normal axis derivation and no Q wave. Chest radiography revealed cardiomegaly with bilateral pleural effusion.

Blood biochemistry revealed normal liver and kidney data, except for hyperuricemia with 12.3 mg/dl of uric acid. Eosinophilia (26%) was present in otherwise cell counts (white blood cell [WBC] 5,600 cells/mm³, red blood cell [RBC] 4.18×10¹²cells/mm³, hemoglobin [Hb] 11.8 g/dl, hematocrit [Ht] 35.6% and thrombocyte 231×10³ cells/mm³) in the peripheral venous blood. Rhabditiform larva of S. stercoralis was found in the fecal smear (Fig 2A).

Abdominal ultrasonography revealed moderate ascites with mild hepatomegaly and a normal spleen. Echocardiography revealed moderate pericardial effusion, left ventricular hypertrophy with normal left ventricular function (ejection fraction = 56%) and moderate mitral regurgitation (Fig 1).

Pericardiocentesis collected approximately 200 ml of bloody pericardial effusion, which contained 3,600 WBC/mm³ with 29% eosinophils, Hb 5.5 g/dl and Ht 18.5%. Biochemistry examination showed that the pericardial effusion was exudate with glucose 83 mg/dl, lactate dehydrogenase (LDH) 578 IU/L, total protein 5.5 g/dl and...
albunin 2.7 g/dl. Filariform larva was found (Fig 2B), but not acid fast-stained bacteria or malignant cells. Because the pericardial effusion refilled on the 5th day after pericardiotomy, pericardiocentesis was performed and histopathological examination also detected larvae in the pericardial specimen (Fig 2C,D). There were neither larvae nor ova in the sputum, nor were focal lesions found in the brain, lung or liver on computed tomography scans.

Large doses of antinematodal agents (mebendazole 900 mg/day and pyrantel pamoate 1,425 mg/day) were administered orally twice weekly for 6 weeks. The symptoms and ascites improved and peripheral venous blood cell counts revealed that the eosinophilia decreased to 1.0% in 6,700 cells/mm³ of WBC. Six consecutive weekly stools without larvae were produced and follow-up echocardiography did not reveal pericardial effusion 1 month after the patient was discharged.

Discussion

Strongyloidiasis is ubiquitous in tropical and subtropical climates, especially in warm wet regions such as Taiwan. 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 tracheobronchial 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, 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. The present patient showed skin lesions or respiratory symptoms. The present patient showed heart failure symptoms, including ascites, which can be secondary to pericardial effusion. Finally, larvae were documented in the pericardial effusion and pericardial specimen.

A hyperinfection syndrome of Strongyloidiasis may develop in immunosuppressed patients and is characterized by the dissemination of infective larvae to other organ systems, resulting in hepatitis, cerebritis, pneumonitis or cardiomyopathy. There can be more than 80% mortality if infection is combined with Gram-negative bacterial septicaemia! Hyperinfection with cerebral involvement has been reported in a non-immunosuppressed, healthy patient! The present patient was also in an immunosuppressed condition, except that he had taken corticosteroid intermittently for his gouty arthritis for approximately 20 years, with average doses of 30 mg prednisolone every 2 months. The intermittent steroid treatment may be associated with the development of pericardial Strongyloidiasis although he was not ever on long-term steroid therapy.

Transcutaneous infection is the main route of Strongyloidiasis. Nolan et al reported, however, that oral transfer of adult S. stercoralis produced autoinfection in gerbils and larvae of S. stercoralis have been found in the feces of wild flying squirrels at Taipei Zoo (unpublished data). The present patient used to be a hunter and for about 20 years followed his tribe’s custom of eating the raw guts of flying squirrel, so the most likely route of infection in this patient would be oral.

We are the first to document, to our knowledge, S. stercoralis infection with pericardial involvement in a patient, although others have reported clinically suspected Strongyloidiasis cardiomyopathy. Nolan et al reported, however, that oral transfer of parasitic adult worms produces infection in mice and infection with subsequent autoinfection in gerbils. Int J Parasitol 1999; 29: 1047–1051.


