Ascites with Strongyloides Stercoralis in a Patient with Acute Alcoholic Pancreatitis and Liver Cirrhosis

Vasile Drug, Raluca Haliga, Qasim Akbar, Catalina Mihai, Cristina Cijevschi Prelipcean, Carol Stanciu

University of Medicine and Pharmacy “Gr. T. Popa”, Institute of Gastroenterology and Hepatology, Iasi, Romania

Abstract

Infection with Strongyloides stercoralis (S. stercoralis) is rarely reported in temperate countries. In addition, there are few reported cases of patients with ascites with S. stercoralis worldwide, usually in immunocompromised subjects. We present the case of a young patient with alcoholic liver disease and acute pancreatitis, who developed ascites with S. stercoralis. The patient had no evident immunosupression (HIV negative and absence of immunosupressive therapy). This is the first reported case where acute pancreatitis could have precipitated infection of ascitic fluid with S. stercoralis.

Key words

Strongyloides stercoralis – ascites – acute pancreatitis – liver cirrhosis.

Introduction

Strongyloides stercoralis (S. stercoralis) is a nematode common in tropical and subtropical areas infecting approximately 100 million people each year, worldwide. The prevalence of infection varies widely geographically and is commonly associated with rural areas and inadequate sanitation [1]. However, infection with S. stercoralis has been reported in developed countries such as USA and in several areas of Western Europe [2]. Some of these cases were evidently linked with traveling in endemic area [2, 3]. Sporadic cases were reported in Romania [4].

The adult form can survive and reproduce either in the soil or in the human small intestine. The life cycle of S. stercoralis in humans begins when the infective filariform larvae penetrate the skin and migrate to the lungs. Once the larvae reach the pulmonary capillary vessels, they migrate through the capillary walls into the pulmonary alveoli. The larvae are eliminated to the larynx and then are swallowed gaining access to the small bowel. The larvae develop into adult females, which lay eggs that hatch non-migratory (rhabditiform) larvae. They are passed in the stool and may also penetrate the mucosa, leading to internal auto-infection [5].

A patient infected with S. stercoralis may present various clinical syndromes [3]. In most cases, the patients are asymptomatic. The diagnosis may be revealed during a routine examination for hypereosinophylia. Some patients may be diagnosed even 15-20 years after contamination. Severe forms (hyperinfection) are reported, usually in immunosuppressed patients with potential severe outcome [5]. Dissemination may involve the gut, stomach, lung, the cerebrospinal fluid and may determine occurrence of ascites. Furthermore, larvae penetration of the intestinal wall during dissemination may result in bacteriemia due the intestinal germs. It is generally accepted that, without an aggressive treatment, hyperinfection may prove fatal [3, 5].

We present the case of a young patient with alcoholic liver disease and acute pancreatitis who developed ascites with S. stercoralis.

Case presentation

A 29 year old man, from an urban area, was admitted to our department, in June 2008 for abdominal pain, nausea, vomiting, fever, anorexia, weight loss (17 kg in the last two months) and increased stool frequency (4 stools/day). The patient was known as a heavy drinker with consecutive social difficulties but without medical problems. In May 2008 he was admitted to the surgical department for acute pancreatitis. Later, with partial improvement of symptoms, he was referred to our unit for further evaluation.

Physical examination showed pale skin and mucosa, poor nutrition (BMI 21.1 kg/m2), arterial blood pressure 110/60 mmHg, cardiac frequency 92/min, respiratory frequency 16/min. Abdominal examination revealed tenderness in the upper abdomen and hepatosplenomegaly.
Laboratory tests at admission showed increased erythrocyte sedimentation rate (100 mm/hr), leucocytosis (29,650/mm³), neutrophilia (85.4%), normochromic, normocytic anemia (Hb 8.9 g/dl), hyposideremia (serum iron 37 μg/dl), amylasemia 105 U/l and amylasuria 766 U/l, serum natrium level 125 mmol/l and CA19-9 level of 312.8 U/ml. Liver function tests revealed hepatocytolysis (AST 109 U/l), cholestasis (total bilirubin 2.75 mg/dl, conjugated bilirubin 1.73 mg/dl, alkaline phosphatase 222 U/l), a prothrombin index of 42%, serum albumin of 2.64 g/dl and gama-globulins 3.62 g/dl. The thrombocyte count, blood urea, creatinine, glycaemia, lipid level, urinary examination, LDH and CEA were within normal range. Chronic hepatitis B or C were excluded by serologic testing.

The abdominal ultrasound examination revealed minimal ascites, hepatosplenomegaly, celiac and subhepatic adenopathy. Examination of ascitic fluid was not possible due to the small quantity of ascites. Upper gastrointestinal endoscopy excluded the presence of esophageal or gastric varices.

Abdominal CT revealed multiple abdominal adenopathies, hepatosplenomegaly, but did not show any pancreatic abnormality.

Abdominal lymphoma or tuberculosis were suspected. However, diagnostic laparoscopy and lymph node biopsy was postponed due to the low prothrombin index. During hospitalization, which involved therapy with broad spectrum antibiotics, both clinical and paraclinical evolution was favorable with normalization of white blood cell count. A reduction of hepatocytolysis and cholestasis was noted. However, a moderate increase of ascites was also evident. Examination of the ascitic fluid revealed an amylase level over 100,000 UI/L and the presence of S. stercoralis (Fig. 1) and E. coli in the culture.

The coproparasitologic examination showed numerous mobile larvae of S. stercoralis (Fig. 2). This confirmed a systemic infection with S. stercoralis. In addition, plasma immunoelectrophoresis revealed marked increase of IgE (1,989 UI/ml) and IgG level (3,966 UI/ml).

We considered that ascites in this patient might have had a triple etiology: liver cirrhosis (plasma albumin/ascitic albumin gradient > 11 g/l), complication of acute pancreatitis (ascitic fluid amylase level over 100,000 UI/L) and infection with S. stercoralis and E. coli.

The patient was treated with albendazole 800 mg/day and norfloxacin 400 mg/day for 14 days, and the ascites disappeared. The control coproparasitologic examination documented the absence of S. Stercoralis.

**Discussion**

There are very few reported cases of patients with ascites infected with S. stercoralis. In 2004, Hong et al reported the case of a man who came to the United States, four years earlier from Liberia and developed ascites and subsequently was found HIV positive. In this patient, the diagnostic paracentesis showed numerous filariform larvae of S. stercoralis and stool examination confirmed the presence of both rhabditiform and filariform larvae. The authors considered the case to be the second reported in the English-language literature, after the first one reported in 1991 by Lambroza [6, 7].

In 2005, Lawate and Singh reported a case of eosinophilic ascites in a patient from India [8] and in 2006, Ramdial et al reported an autopsy case series of 5 HIV positive male patients from South Africa with mesenteric lymphadenopathy, intestinal pseudo-obstruction and ascites [9]. Typically, in S. stercoralis infected patients, if the host becomes immunocompromised, autoinfection may increase the intestinal worm burden and lead to disseminated strongyloidiasis. The diagnosis in such patients may at times be difficult because of a lower incidence of eosinophilia [3].

The adult female larvae can remain embedded in the mucosa of the small intestine for years, producing eggs that develop either in rhabditiform, noninfective larvae or filariform, infective larvae. Manifestations of dissemination occur when the filariform larvae penetrate the intestinal wall and migrate in the blood. Pulmonary involvement is common, and the central nervous system may be affected. Much less commonly described is invasion of the peritoneal cavity with peritoneal effusion [6].

We presented a patient from a temperate country where cases of infection with S. stercoralis are rare, and without past history of travel to an endemic country.
The presence of S. stercoralis in the ascitic fluid needs further discussion. The patient was repeatedly found HIV negative and was not under immunosuppressive therapy. Nevertheless, he reported chronic alcoholic abuse. We can speculate that the recent acute pancreatitis with the presence of amylase in the ascitic fluid could be the factor which contributed to the infection of the ascites. In addition, we have to underline that the plasma albumin/ascites albumin gradient was > 11g/l, documenting the presence of portal hypertension.

Another particularity of the case was that, on admission, the patient presented with high fever, leucocytosis with neutrophilia, a normal number of eosinophils in blood and ascitic fluid, and the presence of E. coli in the ascitic fluid. E. coli co-infection is common in the presence of S. stercoralis in ascites and S. stercoralis has been reported as an infection vector [3]. The therapy with Cefotaxime was initiated blindly, with normalisation of fever and leukocyte count.

The presence of abdominal adenopathy has been reported in infection with S. stercoralis [9], in acute pancreatitis and liver cirrhosis. This aspect could have lead to unnecessary laparoscopy and our wait-and-see policy has assisted the patient in escaping from the procedure. In addition, the CA 19-9 abnormal value made the case even more difficult to diagnose, but it is recognized that this tumor marker could be elevated during pancreatic inflammatory processes.

In conclusion, this is the first reported case when acute pancreatitis may have precipitated infection of ascitic fluid with Strongyloides stercoralis in a patient with alcoholic liver disease.

Acknowledgement

The authors are thankful to Dr Brandusa Copacianu and Gabriela Marinescu from Synevo Laboratories Iasi and Prof. Mariana Luca from the University of Medicine and Pharmacy Iasi for the laboratory investigation support. The authors are also thankful to Prof. Vasile Luca for advice on patient therapeutic management.

References