

Strongyloides stercoralis and peritoneal tuberculosis in a migrant from Ghana

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ABSTRACT: The gastrointestinal tract is reported to be the sixth most common extrapulmonary site of *Mycobacterium tuberculosis* infection and chest X-ray shows concomitant lesions in 25-30% of cases. Any location from mouth to anus can be involved but, the ileocecal area is the most typical site of gastrointestinal tuberculosis, while tuberculous peritonitis is relatively rare. We describe a case of gastrointestinal and peritoneal tuberculosis in a man coming from Ghana.

— **Key words:** *Strongyloides stercoralis*, Peritoneal tuberculosis, *Mycobacterium tuberculosis*.

INTRODUCTION

Mycobacterium tuberculosis gastrointestinal involvement may be either primary or secondary, from ingestion of the organism or from a pulmonary source. *Tuberculous peritonitis* occurs in less than 1% of cases¹⁻¹⁰ and results from spread of adjacent tuberculous disease as an abdominal lymph node, intestinal focus, Fallopian tube or during miliary tuberculosis.

Symptoms of fever, abdominal pain, weight loss are common; ascites usually result from peritoneal lymphatic obstruction and a lack of peritoneal fluid reabsorption, rarely from portal hypertension².

Acid fast smear of peritoneal fluid is rarely positive, the culture is positive in 25% of cases³; diagnostic laparoscopy is usually necessary for definitive diagnosis.

CASE REPORT

A 31 year old man, originally from Ghana and resident in Calabria since three years, was admitted for a history of 30 days of fever, weight loss (10 kg), dysuria, diarrhoea. He was transferred from another hospital with the suspicion of schistosomiasis.

He was asthenic, with dehydrated pale mucous membranes and globular abdomen; on physical examination the

patient was febrile with normal pulse and blood pressure, cardiopulmonary examination was unremarkable, abdomen was distended and painful with mild hepatomegaly, inguinal adenopathy, painful hands and feet joints.

WBC were 2500 mmc, 64% of neutrophils, 28% of lymphocytes, CD4+ T-cells were 42% and CD8 + T-cells were 12%; Hb 11.8 g%; CRP 16 mg/dl; total protein 6.5 g/dl with albumin 2.43 g/dl; renal and urine examination were normal as well as coagulation tests.

The serology for HIV, HBV, HCV, Cytomegalovirus, VDRL and TPHA were negative.

Antibodies and Schistosome eggs in faeces and urine were negative.

The stool examination showed numerous mobile larvae of *Strongyloides stercoralis*, as such, the patient was treated with Albendazole 400 mg po bid for 8 days.

Mantoux intradermal reaction and *QuantiFERON-TB* Gold test were positive.

Ultrasonographic examination revealed considerable ascites, thickening loops and multiple mesenteric lymph nodes.

A chest X-ray documented circumscribed parenchymal consolidation in the basal right lung, with a slight pleural effusion. A thoracic CT scan showed calcified nodules in the left basal and in the left upper lobe. Direct microscopy of acid-fast bacillus in the sputum was negative.

Paracentesis and analysis of ascitic fluid was performed: pH 8.5, 1200 white blood cells, albumin level of 27 g/L, serum-ascites albumin gradient (SAAG) of 11 g/L; fluid cytology didn't reveal filariform larvae of *S. stercoralis*; Gram stain and Ziehl-Neelsen staining in the specimen were negative; the cytology revealed typical mesothelial elements and numerous leukocytes (mainly lymphocytes).

The exploratory laparoscopy described a thickened and hyperemic peritoneum with whitish or yellowish granular nodules (5 mm) and peritoneal biopsy revealed chronic granulomatous infection with giant cells.

The seventeenth day of hospitalization, antituberculosis treatment with four drugs was initiated, on the basis of body weight: rifampicin 450 mg po q24h, isoniazid 300 mg po q24h, pyrazinamide 1000 mg po q24h and ethambutol 1000 mg po q24h, plus vitamin B6 300 mg po q24h.

The results of culture exam of ascitic fluid were positive for *Mycobacterium tuberculosis*, after the treatment had already started.

DISCUSSION

Tuberculosis infection may involve any part of the gastrointestinal tract from a primary ingestion of the organism or secondary to other source.

The peritoneal involvement is due to different mechanisms: hematogenous spread of bacilli from a primary pulmonary site, swallowing sputum in the case of active lung disease, spread from secondary foci such as the peritoneum, ruptured retroperitoneal and mesenteric lymph nodes, from lesion in adjacent organs or by direct contamination⁴.

There are three different clinical forms of peritoneal tuberculosis: wet-ascitic, fibrotic-fixed, and dry-plastic form. The clinical differences among the three types are not always distinct with the exception of the clinical presentation of abdominal distension, which is not present in the dry-plastic form, the less common form⁵.

The most common clinical features are fever and abdominal pain, weight loss is more common in secondary gastrointestinal tuberculosis. Only one third of patients have diarrhea; although ulceration and mucous diarrhea are common with secondary form, hemorrhage and the presence of blood in the stool are uncommon, maybe due to the obliterative endarteritis.

ATB is a diagnostic challenge, especially in the absence of lung involvement; concomitant lesions at the chest X-ray are present in 25-30% of cases, the pleural effusion may be the only finding in the course of peritoneal TB⁶.

The characteristic colonoscopic appearances of TB are ulceration and polypoid lesions⁷, these exams can help the

diagnosis, mainly the differential diagnosis. The confirmation comes from the study of ascites fluid and biopsies.

The peritoneal fluid is exudative, usually containing 500 to 2000 cells, lymphocytes typically predominate, protein content higher than 30 g/L, SAAG < 11 g/L (low specificity); an increased adenosine deaminase level have high sensitivity and specificity [8], cytological study must always be performed to look for neoplastic cells, the direct observation of acid-fast bacillus has a sensitivity of less than 10%, the evidence of granulomas with caseous necrosis on histological examination of peritoneal biopsy and the isolation of *M. tuberculosis* in culture of ascitic fluid or in biopsies confirm the diagnosis.

The non-specific clinical presentation often results in diagnostic delay, with an average of 3 months; we made diagnosis and we started a specific treatment after 17 days. We were initially searching for *Strongyloides stercoralis*, an unusual cause of ascites, but we found *Mycobacterium tuberculosis*.

CONFLICT OF INTERESTS:

The Authors declare that they have no conflict of interests.

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