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Bouma, B.J.; Tytgat, K.M.A.J.; Schipper, H.G.; Kager, P.A.

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Brief report

Be aware of abdominal tuberculosis

B.J. Bouma, K.M.A.J. Tytgat, H.G. Schipper *, P.A. Kager

Department of Internal Medicine, Division of Infectious Diseases, Tropical Medicine and AIDS, Academic Medical Centre, Meibergdreef 9, 1105 AZ Amsterdam, Netherlands

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Abstract

Abdominal tuberculosis is often diagnosed in a late stage because symptoms are aspecific. Two patients with intestinal tuberculosis and tuberculous peritonitis respectively, both from endemic countries presented with long-standing fever, abdominal pain and weight loss. Acid fast bacilli were present in aspirate and biopsy specimens obtained by colonoscopy and laparoscopy respectively; PCR was positive for *M. tuberculosis* complex and later *M. tuberculosis* was cultured. Both patients responded to antituberculous therapy. In one patient AIDS was diagnosed. © 1997 Elsevier Science B.V.

Keywords: Abdominal tuberculosis; Echography; Puncture

1. Introduction

Abdominal tuberculosis is often diagnosed in a late stage with advanced disease because the clinical symptoms are aspecific. This results in serious morbidity and high mortality [1–4]. The key to the diagnosis is to think of TB and to demonstrate the organism in material obtained by aspiration of the affected organs. The epidemiology, symptomatology and the diagnostic process is discussed in two patients.

2. Case reports

Patient A, a 37-year-old Filipino man was admitted after arrival at Schiphol Airport from Lagos,

Nigeria, where he had worked as an engineer for about 10 years. For 5 months he had suffered from fever, rigors, abdominal pain, watery diarrhoea and 15 kg weight loss, but no night sweats. In Nigeria he had been treated for malaria and twice for possible infectious diseases, without improvement.

Physical examination showed a sick, shivering and dehydrated man with cold acra and oral candidiasis, 163 cm tall and weighing 45 kg. The blood pressure was 120/75 mmHg, pulse rate 120 beats/min and temperature 39°C. Palpation of the liver was painful and in the right lower abdomen a firm irregular mass was present.

Laboratory data showed an ESR of 140 mm in the first hour, haemoglobin 6.2 mmol/l (normocytic), leukocytes $12.6 \times 10^9/l$ with 96% neutrophils; alkaline phosphatase 112 U/l, gGT 174 U/l, ASAT 83 U/l, ALAT 96 U/l, albumen 29 g/l and creatinine 238 $\mu\text{mol/l}$.

An amoebic liver abscess, typhoid fever and malaria were considered although none of these di-

* Corresponding author. Tel: +31 20 5666084; fax: +31 20 6972286; e-mail: hgschipper@amc.uva.nl

agnoses fully covered his clinical picture. A chest X-ray was normal. No liver abscess was found by ultrasound but in the right lower abdomen and along the aorta enlarged lymph nodes were present. A thick blood smear, amoebic serology and parasitological examination of stools were all negative. Blood, urine and stool samples were taken for culture and examination. Metronidazole and ciprofloxacin were started without a clinical response after 2 days. Blood cultures remained negative and no pathogenic microorganisms were isolated from the stools. Mantoux skin test was inconclusive because of anergy at multitest. He had never had BCG vaccination.

In search of tuberculosis the enlarged ileocaecal lymph nodes were aspirated under ultrasound guidance, showing an aspecific inflammatory process. At colonoscopy an enlarged, polypous, ulcerating valve of Bauhin was seen (Fig. 1), with acid fast bacilli in the biopsies and *M. tuberculosis* in culture. Acid fast bacilli were also present in the lymph nodes and PCR for *Mycobacterium tuberculosis* complex herein was positive.

Because of his relationship with an African woman, the anergy and the diagnosed tuberculosis, HIV infection was considered. HIV serology was

positive and CD4 cell count was $< 10 \times 10^6/l$. Thus, the diagnosis was abdominal tuberculosis and AIDS. After institution of tuberculostatics temperature normalized promptly. Abdominal pain, diarrhoea and palpable lymph nodes resolved in a few weeks. Later antiretroviral therapy and PCP-prophylaxis were added. He returned to the Philippines and was advised to continue isoniazide, rifampicin, ethambutol and pyrazinamide for 2 months and thereafter to proceed with isoniazide and rifampicin for at least another 12 months.

Patient B was a 28-year-old Moroccan woman living in the Netherlands for 5 years. For the previous 5 months she had complained of malaise, peaking fever and night sweats and during the final 2 months of nausea, abdominal pain and diarrhoea; she had lost 8 kg body weight. She was hospitalized because of progression of her complaints.

At physical examination she was rather sick, 159 cm tall and weighing 42 kg. Blood pressure and pulse rate were normal and later temperature peaked to 40°C. Ascites and hepatosplenomegaly were present and in the right lower abdomen a mass was palpable.

Laboratory data showed an ESR of 60 mm in the first hour, haemoglobin 6.0 mmol/l (microcytic), leukocytes $11.6 \times 10^9/l$ with 4% band forms; alkaline phosphatase 125 U/l, LDH 307 U/l, albumen 37 g/l. Other liver enzymes and creatinine were normal.

Inflammatory bowel disease, lymphoma and abdominal tuberculosis were considered. A chest X-ray showed right hilar lymphadenopathy; an ultrasound demonstrated ascites, partly located with multiple thin septa, thickening of the mesentery and in the right lower abdomen adherence of intestinal loops to the abdominal wall.

In the aspirated yellow ascites fluid total protein content was 62 g/l, albumen 26 g/l, LDH 381 U/l and ZN-staining was negative. Leukocyte count was $0.9 \times 10^9/l$ with 72% lymphocytes and no malignant cells. A Mantoux test was positive (20 × 18 mm). Although usual in Morocco, she had refrained from BCG vaccination.

At laparoscopy, multiple grains were seen on the peritoneum and liver surface with many adhesions. A biopsy of the peritoneum showed a caseous granulomatous inflammation with one acid fast bacillus.

Fig. 1. Endoscopic view of the enlarged, polypous, ulcerating valve of Bauhin (B); I = ileum; C = caecum.

PCR was positive for *M. tuberculosis* complex and culturing of *M. tuberculosis* confirmed the diagnosis. ZN-staining of sputum, urine, stools and HIV serology were all negative. Therapy for tuberculosis was temporarily complicated by a hepatitis. Fever resolved in a few days, diarrhoea stopped and abdominal pain disappeared. She was treated with quadruple therapy for 2 months after which isoniazide and rifampicin were continued for another 12 months. She recovered well.

3. Discussion

Both patients were inhabitants from tuberculosis endemic countries. Neither of them had had BCG vaccination. They were suffering for months from fever, abdominal pain, diarrhoea and weight loss before the diagnosis was made. At physical examination in both patients a palpable mass was present in the right lower abdomen. Patient B presented also with ascites and hepatosplenomegaly.

In patient A intestinal tuberculosis was diagnosed after puncture of abdominal lymph nodes and biopsy during colonoscopy. In patient B the diagnosis tuberculous peritonitis became apparent during laparoscopy, when multiple peritoneal grains and many adhesions were seen. In both patients the diagnosis was made probable by ZN-staining and PCR of the material obtained by puncture or biopsy and confirmed by culturing of *M. tuberculosis*. These strains were sensitive for all four tuberculostatics with which the patients were treated. This regimen would also have been appropriate if *M. bovis* was the infectious agent, a less frequent observation in African TB. *M. bovis* infection from ingestion of contaminated milk had to be considered in our patients because they probably were infected in Africa.

In the Netherlands, immigrants (above all Moroccans, Somalians and Turks) are a risk group for tuberculosis. In 1994 the incidence in immigrants was 124 and in Dutch people 5.8 per 100 000. In about one-third of these patients tuberculosis is located extrapulmonary, e.g., abdominally [5].

Abdominal tuberculosis is subdivided into intestinal tuberculosis and tuberculous peritonitis which occur with about the same frequency (49 and 43% respectively) and in tuberculous lymphadenitis which is rare (8%). It is assumed that intestinal tuberculosis

is due mostly to swallowed respiratory secretions. Tuberculous peritonitis may result from the spread of a tuberculous focus in intestines, abdominal lymph nodes or Fallopian tube or during the course of miliary TB.

The complaints are aspecific. Abdominal pain and weight loss are predominant (62–85%), fever is less frequent (50–75%) and diarrhoea is infrequent (20%). Causal treatment is initiated often in a late stage which results in complications such as obstructive ileus, fistulas, intestinal perforation and bleeding. Mortality varies from 6 to 38% [1–4,6].

In about half of the patients with intestinal tuberculosis a palpable mass in the right lower abdomen is present. Patients with tuberculous peritonitis almost always present with ascites (97%) [1–4].

The diagnostic process of abdominal tuberculosis must be goal-directed. In patients with intestinal tuberculosis an echo-guided puncture or a biopsy during endoscopy is the procedure with the highest yield. Colonoscopy with biopsy reveals acid fast bacilli or granulomas in 30–60% [1–3]. In patients with tuberculous peritonitis, during laparoscopy peritoneal and mesenteric grains are visible in 85–95%. Biopsies will show acid-fast bacilli or caseous granulomas. In the remaining 5–15% of patients a random peritoneal biopsy shows granulomas in 80–93% [1–3]. PCR of aspirated material is helpful for early confirmation of tuberculosis and in discriminating *M. tuberculosis* complex from *M. avium* in patients with AIDS.

For radiological diagnosis of abdominal tuberculosis CT scan and ultrasound are advocated. At CT scan especially multiple pelvic, adrenal, splenic and hepatic lesions and ascites point to TB as a serious consideration in the differential diagnosis [7,8]. Recently, echography and an echo-guided biopsy of mesentery, omentum or peritoneum were shown to be very effective to diagnose tuberculous peritonitis. Localized ascites with thin septa and thickening of mesentery, omentum and peritoneum are characteristic signs of tuberculous peritonitis. When these signs are present a less invasive echo-guided puncture can replace a diagnostic laparoscopy or laparotomy. From a total of 11 patients with ascites echo-guided punctures yielded the bacteriological diagnosis in 8 of them [9].

ZN-staining of ascites is only positive in 3%;

culturing of ascites is positive in 20% [1–3]. The yield can be increased by examining the sediment of a large quantity of ascites [10,11]. Biochemical examination of ascites may be of help: high total protein content (> 25 g/l), a small serum-ascites albumen gradient (< 11 g/l) and an elevated LDH level (> 90 U/l) are suggestive of tuberculous peritonitis [3]. Ascitic lymphocytosis supports this diagnosis. High levels of adenosine deaminase in ascites may also discriminate tuberculosis from other causes of ascites. Both sensitivity and specificity of this test are high but not in patients with liver cirrhosis [12,13].

In all patients with abdominal tuberculosis, diagnosis has to be confirmed by culturing *M. tuberculosis*.

In summary, abdominal tuberculosis is an insidious disease with aspecific symptoms suggesting Crohn's disease, intestinal lymphoma, malignancy, appendicitis, *Yersinia enterocolitis* or other infections [4,14]. In the differential diagnosis it may be especially difficult to discriminate abdominal TB from malignancy. More than once, high levels of the tumour marker CA 125 have led to an erroneous diagnosis of carcinoma in these patients [15,16]. Thus the diagnosis is often made late, the treatment started in advanced disease and the prognosis poor. Once abdominal tuberculosis is considered, a goal-directed diagnostic strategy is indicated. In patients with intestinal tuberculosis an echo-guided percutaneous puncture of a palpable mass or enlarged lymph nodes or colonoscopy with biopsy is indicated. In patients with tuberculous peritonitis an echo-guided aspiration of ascites and omentum or laparoscopy with biopsy are advised. With relatively simple microbiological and microscopical examination a provisional diagnosis can be made and treatment started until confirmation by culturing. Early treatment prevents many complications. Treatment should be initiated with a four-drug regimen (isoniazide, rifampicin, ethambutol and pyrazinamide) because of possible mycobacterial resistance. After 2 months INH and rifampicin are continued for another 12 months. Treatment can be stopped if the patient responds well. AIDS patients may need a prolonged course because of impaired cellular defense.

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