A healthy 23-year-old man who emigrated from Ethiopia at the age of 5 years was admitted with diffuse abdominal pain, anorexia, weight loss and vomiting for over six weeks. He was found to be febrile (38.9°C), but examination was non-contributory and chest X-ray was normal. Hemoglobin was 11.2 g/dl, WBC 6.4 × 10^3/µl, platelets 394 × 10^3/µl with normal urinalysis and serum chemistry. Serum albumin was 3.3 g/dl and globulins 3.8 g/dl showing increased α1, α2 and γ fractions on electrophoresis. Erythrocyte sedimentation rate (ESR) was 66 mm/h, C-reactive protein 170 mg/dl. Abdominal ultrasound was normal except for some ascitic fluid in the lower abdomen. A tap yielded clear fluid with 2.6 g/dl albumin, normal glucose, 0.8 × 10^3/µl mononuclear cells and negative microbiological stains. Cultures grew Clostridium perfringens and fever resolved with amoxicillin-clavulanate treatment. However, periumbilical pain and malaise persisted, associated with occasional diarrhea, worsening laboratory tests (serum albumin 2.2 g/dl) and cachexia [Body mass index (BMI) 17.4]. The abdomen now seemed ‘doughy’ to palpation. The purified protein derivative of tuberculin (PPD) was 30 × 17 mm. HIV test was negative. Abdominal computerised tomography scanning (CT) demonstrated a small collection of fluid in the lower abdomen; one 15 mm necrotic mesenteric lymph node and extensive involvement of the peritoneum and omentum (Figure 1). Gastroscopy, enteroscopy and distal duodenal biopsies were non-contributory.

Figure 1. The patient’s axial (A) and coronal (B) contrast-enhanced CT showing thickening of the peritoneum, peritoneal implants indenting the liver, omental cake with numerous pockets of fluid and mesenteric involvement in the form of linear soft tissue stranding, thickened crowded vessels and ascitic fluid trapped between mesenteric leaves.
An explorative laparotomy was performed revealing extensive peritoneal disease. Biopsies demonstrated necrotizing granulomatous inflammation with geographic necrosis but stains for acid-fast bacilli (AFB) were negative. Treatment with four anti-tuberculous drugs led to a gradual complete recovery. After 3 weeks, cultures of the biopsied peritoneum grew *Mycobacterium tuberculosis*.

Our patient presented with systemic illness that had two important characteristics: low serum-ascites albumin gradient (SAAG) of 0.7 that effectively rules out portal hypertension and is caused by tuberculosis or malignancy in most cases;¹ and omental and mesenteric involvement demonstrated by CT imaging (Figure 1). The differential included TB and diverse neoplastic conditions (peritoneal carcinomatosis, papillary serous carcinoma, desmoplastic small round-cell tumor and mesenchymal tumors). A histopathological diagnosis was deemed mandatory since the CT findings were not specific.² The surgical biopsy, response to treatment and cultures (in that order) clinched the diagnosis of TB peritonitis. The other two forms of abdominal TB—mesenteric lymphadenitis and ileo-cecal disease³ were not represented here, supporting hematogenous spread in this case. In most patients with abdominal TB, neither the ascitic fluid tests nor the imaging findings are specific enough for diagnosis.

Peritoneal biopsy by laparoscopy or laparotomy is the gold standard test³ and enables histological examination and culture. Typical histological findings are caseating granulomata and more specific, but less frequently found—AFB. Tuberculous peritonitis often complicates end-stage liver or kidney disease and AIDS. These high-risk patients constitute a particular diagnostic challenge. Here, reactivation of latent infection acquired in Ethiopia or recent exposure in the immigrant community leading to primary progression are both likely. Thus, our patient exemplifies the insidious non-specific presentation of abdominal tuberculosis and serves to remind clinicians of the need for a high index of suspicion and the importance of obtaining tissue as early as possible for establishing the diagnosis.

References