Case Report

Abdominal tuberculosis presenting as ascites in an older indigenous woman: a case report

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Introduction: Indigenous people tend to have higher incidence of Mycobacterium tuberculosis infection (TB). The diagnosis of TB is a challenge among older patients because of its non-specific presentation, especially for cases of extrapulmonary TB.

Case presentation: A 77-year-old indigenous woman presented with ascites secondary to abdominal TB. A computed tomography scan of the abdomen showed free fluid. The patient had a positive tuberculin test, and the ascitic fluid was an exudate with an adenosine deaminase activity test value of 80.76 IU l⁻¹. The mesenteric lymph biopsy showed a central area with caseating granulomas and peripheral giant cells, suggestive of TB commitment at the mesenteric lymph node level. The diagnosis was confirmed by a positive TB ascitic fluid culture. Anti-TB treatment improved the patient’s symptoms.

Conclusion: In patients with ascites as the primary symptom, abdominal TB should be suspected, especially among those belonging to special population groups such as indigenous or older people.

Keywords: abdominal tuberculosis; ascites; indigenous; older patients.
Abdominal TB mimics several diseases, affecting abdominal organs such as the peritoneum, intestine and mesenteric lymph nodes. It also behaves clinically like many abdominal diseases that are often ignored, leading to a significant impact on morbidity and mortality due to lack of early diagnosis and treatment (Sharma & Bhatia, 2004; Uzunkoy et al., 2004; Tan et al., 2009). Here, we report a case in an older indigenous woman.

Case report

A 77-year-old indigenous woman presented with symptoms of weakness, fatigue, decreased appetite and unintended weight loss for 6 months. After this, she had a progressive increase in size of her abdomen with generalized colicky abdominal pain associated with bloating but without fever. She reported exposure to TB in her community, as well as ingestion of unpasteurized milk.

On physical examination, the patient had a blood pressure of 110/70 mmHg, a pulse of 65, and temperature of 36.8 °C. An ophthalmoscope examination was unremarkable. She was alert, hydrated and without neurological deficits or cardiopulmonary disorders. The abdomen was prominent on inspection without collateral circulation but with a positive ascitic wave. There was generalized deep pain on palpation without signs of peritoneal irritation. No masses, organomegaly or hernias were present. Her lower extremities were without oedema. The patient was admitted for hospitalization due to ascites. Initial blood results showed the following: haemoglobin 11.0 g dl⁻¹, haematocrit 33.0 %, leukocytes 6000 mm⁻³, albumin 3.1 g per 100 ml, and carcinoembryonic antigen 3.0 mU ml⁻¹. Hepatic biochemistry and enzymes were normal.

In addition, anti-nuclear and anti-smooth muscle antibodies, hepatitis B and C virus, CA-125 serum antigen and HIV ELISA were all negative. A tuberculin test was positive at 16 mm. A chest X-ray showed a calcified lymph node in the lower lobe of the left lung. The sputum bacilloscopy was negative on three occasions, which ruled out active lesions in the lung and pleura. Endoscopic and imaging studies were performed. An upper gastrointestinal tract endoscopy showed chronic atrophic gastritis, and a colono-scopy showed diverticular disease of the sigmoid colon. An abdominal and pelvic computed tomography (CT) scan showed free fluid in the subphrenic area and parietocolic sulci and mesenteric fat thickening, without retroperitoneal adenopathies. The liver was decreased in size with evidence of a simple cyst in the left lobe. The spleen, kidneys and genitals were normal (Fig. 1).

With the results of the abdominal CT, we proceeded to examine the ascitic fluid through ultrasound-guided paracentesis. The ascitic fluid showed a turbid appearance, with pH 8.0, and glucose 80 mg dl⁻¹, proteins 5.5 g dl⁻¹, albumin 3.0 g dl⁻¹, amylase 30 U l⁻¹, lactate dehydrogenase (LDH) 440 IU l⁻¹, alkaline phosphatase 35 IU l⁻¹, negative for erythrocytes, leukocytes 1200 mm³ (neutrophils 20 %, lymphocytes 80 %) and adenosine deaminase test 80.76 IU l⁻¹. A Gram stain was negative for bacteria, and cytology was negative for malignancy. In summary, the ascitic fluid analysis showed an exudate with a low serum ascites albumin gradient of 0.1 (serum albumin of 3.1 to ascitic fluid albumin of 3.0) and a high ADA test result.

A laparoscopic biopsy of the mesenteric ganglion showed a central area with caseating granulomas and peripheral giant cells, without malignant cells, and was suggestive of TB commitment at the mesenteric lymph node level (Fig. 2). The culture from ascites was positive for M. tuberculosis after 4 weeks.

Fig. 1. CT scan of the abdomen showing free fluid in the abdominal cavity and located in the subphrenic area, and parietocolic sulci and mesenteric fat thickening, without retroperitoneal adenopathies.

Fig. 2. Histology of the mesenteric ganglion with haematoxylin and eosin staining showing a central area with caseating granulomas and peripheral giant cells, without malignant cells.
Discussion

Ascites is a symptom that may be the first sign of a systemic or intra-abdominal disease. It is defined as the presence of serous fluid between the visceral and parietal peritoneum. Aetiologies for ascites include cirrhosis in 75% of cases, cancer in 10% of cases, heart failure in 5% of cases and other miscellaneous causes in 10% of cases (Runyon, 1994; Hou & Sanyal, 2009). In the case reported here, the medical history, clinical data and chest X-ray allowed some common causes of ascites and portal hypertension to be ruled out, including alcohol or viral cirrhosis, heart failure and kidney disease (e.g. nephrotic syndrome). Other less common causes of ascites that were ruled out included cancer, inflammatory or immunological processes such as systemic lupus erythematosus, and autoimmune liver disease (Runyon, 1994; Sharma & Bhatia, 2004; Uzunkoy et al., 2004; Tan et al., 2009; Hou & Sanyal, 2009; Chou et al., 2010).

Because our patient belonged to an indigenous population group, we needed to rule out ascites due to TB (Belo et al., 2013; Hernández Sarmiento et al., 2013). A chest X-ray did not show any active pathology, but a lesion (calcified lymph node) suggested a previous primary TB infection. Chest radiographs demonstrate pulmonary involvement in only 25% of cases (Sharma & Bhatia, 2004), and for this reason the absence of radiographic findings did not completely rule out a possible extrapulmonary TB infection (Tan et al., 2009). A positive tuberculin test suggested the initial TB diagnosis.

An abdominal CT showed fluid collection in the abdominal cavity and subphrenic area. Different authors have described abdominal CT as useful for the differential diagnosis of ascites when peritoneal TB prevalence ranges between 30 and 90% (Vázquez Muñoz et al., 2003). CT is a valuable diagnostic tool for finding lesions in the omentum, mesentery, peritoneum and mesenteric lymph nodes suggestive of peritoneal tuberculosis, yet these images may be difficult to distinguish from ovarian carcinoma or peritoneal carcinomatosis (Vázquez Muñoz et al., 2003; Tan et al., 2009; Chalya et al., 2013). In our patient, the abdominal CT showed abundant ascites in all areas of the peritoneum, with some depletion of mesenteric fat, but did not show omental lesions as seen in other abdominal TB cases reported (Tan et al., 2009; Chalya et al., 2013). Another potential aetiology for ascites in this case corresponds to an ovarian tumour process; however, our patient had a negative serum CA-125 antigen, a CT that ruled out adnexal lesions and peritoneal fluid cytology that was negative for malignancy (Tan et al., 2009; Chalya et al., 2013).

Given the ascitic fluid characteristics described above, and having calculated a low serum ascites albumin gradient index gradient (<1.1 g dl⁻¹), we were able to rule out carcinomatosis of peritoneal, pancreatic or biliary origin, other diseases such as nephrotic syndrome, and collagen vascular disease. This left peritoneal TB as a reasonable possibility (Runyon, 1994; Sharma & Bhatia, 2004; Uzunkoy et al., 2004; Tan et al., 2009).

In relation to the cellularity of the peritoneal fluid, our patient had greater than 1000 counts, indicating an exudate. The differential cell count with predominance of lymphocytes was characteristic of malignancies or collagen diseases, which were discarded, as well as TB. Similarly, elevated LDH levels are associated with cancer, inflammatory disease and TB. LDH levels of over 90 IU l⁻¹ exhibit high sensitivity (90%) and low specificity for the diagnosis of peritoneal TB (López Rodríguez et al., 2004), and in our patient the LDH level was 440 IU l⁻¹.

The determination of ADA in peritoneal fluid is also helpful for TB diagnosis. This enzyme involves the conversion of adenosine to inosine in the catabolism of purines, and its levels are elevated in peritoneal TB due to stimulation of T-lymphocytes by the immune response to antigens of mycobacterial cells. The ADA test in ascitic fluid has been used in the diagnosis of infectious diseases such as viral hepatitis, HIV infection, typhoid fever, malignancies and connective tissue disorders (Brant et al., 1995). In a meta-analysis, Riquelme et al. (2006) studied the ADA levels in ascitic fluid as a diagnostic test for peritoneal tuberculosis and showed high sensitivity (100%) and specificity (97%) using cut-off values of 36–40 IU l⁻¹; the optimal cut-off point was determined as 39 IU l⁻¹. In another study by Kang et al. (2012), an ADA test cut-off level of 21 IU l⁻¹ was found to yield the best results for differential diagnosis between peritoneal TB and peritoneal carcinomatosis, and ADA showed better discriminative capability than tumour markers. Furthermore, the ascitic fluid in peritoneal TB has high protein (exudate) content and a predominance of lymphocytes, as was seen in our patient.

Identification of mycobacteria through coloration (Ziehl–Neelsen technique) for acid/alcohol-resistant bacilli is only positive in less than 3% of cases (Farias Llamas et al., 2005). In our case, the acid/alcohol-resistant bacillus test was negative. For this reason, in order to confirm the TB diagnosis, a biopsy for pathological examination was necessary. The laparoscopic technique is considered the main route of intraperitoneal approach because it not only ensures a correct display of the peritoneal cavity but allows taking of peritoneal fluid, multiple peritoneal biopsies or biopsies from other intra-abdominal locations (Runyon, 1994; Uzunkoy et al., 2004; Hou & Sanyal, 2009; Tan et al., 2009). Our patient was taken for surgical laparoscopic mesenteric lymph biopsy, with results suggestive of TB commitment at the mesenteric lymph node level.

Extension of the abdominal TB can involve any part of the gastrointestinal tract from the mouth to the anus, including the peritoneum, the pancreatobiliary system and mesenteric lymph nodes (Vázquez Muñoz et al., 2003; López Rodríguez et al., 2004; Sharma & Bhatia, 2004; Uzunkoy et al., 2004; Tan et al., 2009; Chou et al., 2010; Chalya et al., 2013).
The transmission of TB infection to the abdomen involves the following pathogenic mechanisms: (i) haematogenous spread from a primary lung focus that healed or stayed latent with subsequent reactivation – this is the most frequent mechanism; (ii) ingestion of bacilli in sputum from an active pulmonary focus; (iii) ingestion of bacilli from infected sources such as milk or milk products; and (iv) contiguous spread from tuberculous lesions of adjacent organs (e.g. lesions in the fallopian tubes and intestines) (Aston, 1997; Vázquez Muñoz et al., 2003; López Rodriguez et al., 2004; Sharma & Bhatia, 2004; Uzunkoy et al., 2004; Khan et al., 2006; Tan et al., 2009; Chou et al., 2010; Chalya et al., 2013).

As the mesenteric lymph nodes and peritoneum are infected during the primary bacteremic phase of pulmonary TB, we proposed that our patient had haematogenous spread as the mechanism of extrapulmonary TB, with secondary reactivation (Aston, 1997; Khan et al., 2006). This is consistent with both the mesenteric lymph node involvement and the peritoneal compromise in our case. Although our patient had a background of ingestion of unpasteurized milk, this rare possibility of transmission was ruled out as Mycobacterium bovis was not found to be the cause of her infection (Aston, 1997; Khan et al., 2006).

Two other disseminated forms of TB, miliary and meningeal, were also considered. Miliary TB can involve many organs such as the choroids (tubercles are pathognomonic of miliary TB), skin, liver, spleen, lungs and meninges. Patients may present with fever for several weeks (daily morning temperature spikes are usually reported), night sweats, anorexia, weight loss, weakness, dyspnoea and cough (Sharma et al., 2012; Ray et al., 2013). However, fever may be absent and the patients may present with progressive wasting mimicking a metastatic carcinoma (cryptic miliary TB), especially in older people. Miliary TB is diagnosed by the presence of a diffuse miliary infiltrate on chest radiograph or CT, or evidence of miliary tubercles in multiple organs at laparoscopy or on abdominal CT. The disease is more frequently encountered in immunosuppressed individuals (Sharma et al., 2012; Ray et al., 2013).

Our patient had no choroidal, skin or hepatic involvement.

TB meningitis develops when an intracranial tubercle ruptures, causing predominantly basal meningitis and subsequent brain damage related to tuberculous vasculitis. The primary source of TB is usually the lung but could be miliary dissemination. Patients tend to present subacutely with low-grade fever, malaise, headache, stiff neck, dizziness, vomiting and/or personality changes for a few weeks, after which they can then develop altered mental status, ischaemic brain injury, hydrocephalus and cranial nerve palsies or seizures (Bhigjee et al., 2007; Marx & Chan, 2011). Our patient did not have neurological abnormalities on physical examination.

Various diseases associated with opportunistic infections were discarded, as the risk of peritoneal TB infection increases with a number of risk factors such as steroid therapy, immunosuppression, HIV, cirrhosis, diabetes mellitus, malignancy and peritoneal dialysis, among others (Vázquez Muñoz et al., 2003; López Rodriguez et al., 2004; Sharma & Bhatia, 2004; Uzunkoy et al., 2004; Tan et al., 2009; Chou et al., 2010; Chalya et al., 2013).

Considering the patient’s indigenous origin, the results of different screening studies and an ascitic fluid culture positive for TB, the diagnosis of abdominal TB with peritoneal and mesenteric lymph node involvement was made. For this, we started treatment with rifampicin and isoniazid (Rimactazid), pyrazinamide, ethambutol and pyridoxine, and the patient displayed a remarkable clinical improvement.

**Conclusion**

In patients with ascites belonging to special population groups such as indigenous or older people, TB should always be considered in the diagnosis. In patients with ascites as the primary symptom, abdominal TB should be suspected.

**References**


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