but to be able to withdraw the causal agent and achieve healing.

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Conflict of interests

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Lower gastrointestinal bleeding as a presentation of miliary tuberculosis

Hemorragia digestiva baja como presentación de una tuberculosis miliar

A 44-year-old male, native of Bolivia but living in Spain for the last 10 years without recent travels to his country, and with no history of interest, attended the emergency room complaining of rectal bleeding with hemodynamic instability. The patient also addressed a three-month period of diffuse abdominal pain (predominantly in left flank), dysmotility, dysthermia, night sweats and weight loss. Physical examination, including neurological examination, was unremarkable except for malnutrition and pain in the left flank's deep palpation without detecting masses, organomegaly or ascites.

Tests revealed 7.6 g/dL hemoglobin levels with ferropenia profile, 135 mg/dL CRP, 60% Quick I. and severe malnutrition. The rest of the parameters, including WBC count, electrocardiogram and chest radiograph resulted without alterations. Colonoscopy was performed without finding mucosal lesions, though with hematic content in terminal ileum. Gastroscopy was normal. CT scan showed jejunal thickening with plenty intraluminal hematic content without actually observing bleeding points. In addition, a thickening of the omentum, multiple peritoneal implants, ascites, and lymphadenopathy were demonstrated. Double contrast CT and gastrointestinal transit were performed without new discoveries (Fig. 1).

On admission the blood culture, HIV and Mantoux tests were negative. Early laparoscopy was performed, obtaining images that suggested miliary TB vs. peritoneal carcinomatosis (Fig. 2).

Histology showed abundant caseating granulomas. The Ziehl Neelsen stain and PCR for M. Tuberculosis in peritoneal fluid were negative. Due to these findings the patient was isolated until the negative result of sputum smear microscopy arrived. Pleural effusion was found in a second radiography with negative RCP for Mycobacteria.

Treatment was initiated with rifampicin, isoniazid, pyrazinamide and ethambutol in weight-adjusted doses, with resolution of symptoms. As an adverse effect the patient presented transient elevation of transaminases. After 8 weeks, sputum results arrived: both solid and liquid medium cultures were positive for Mycobacterium Tuberculosis sensitive to all TB drugs. Peritoneal fluid and urine cultures in both solid and liquid medium were negative. We had no peritoneum sample for culture. Currently our patient is progressing well with a 5 kg weight gain and continues with rifampicin and isoniazid, intending to maintain the treatment for at least 12 months.

In this case gastrointestinal bleeding related to jejunal affectation and probably secondary to peritoneal extension, allowed the diagnosis of miliary tuberculosis.

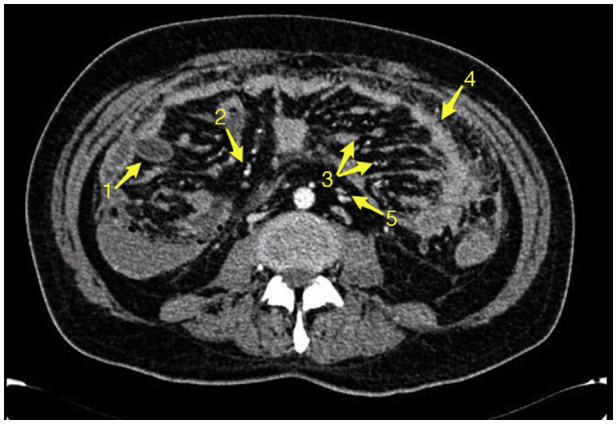

Figure 1 CT showing (1) blood content in jejunum, (2) multiple peritoneal implants, (3) omental cake, (4) thickening of the omentum, (5) lymphadenopathy.

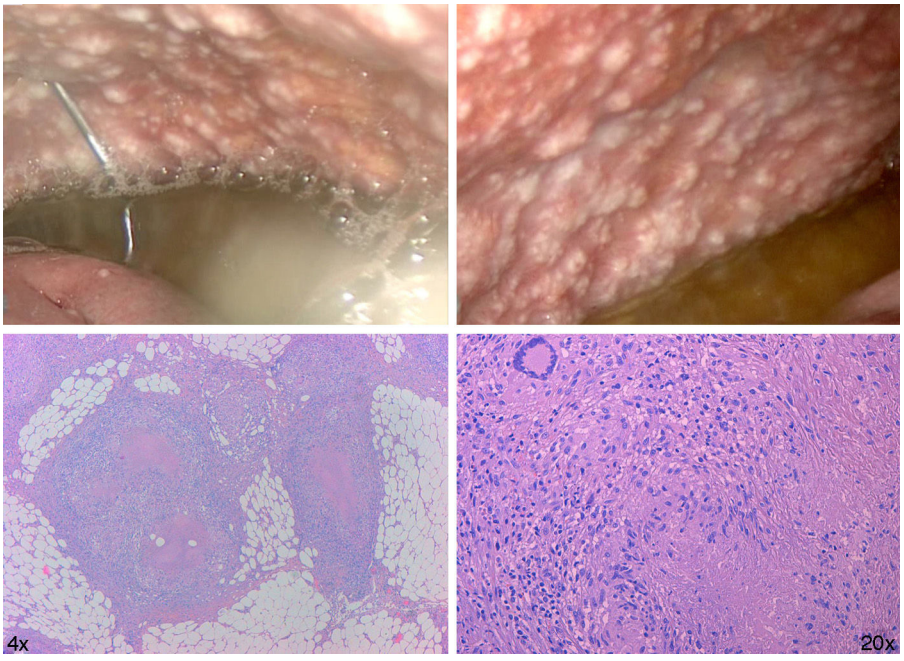

Figure 2 On the top, two captures showing laparoscopy with multiple nodules both parietal and visceral peritoneum. On the bottom, pathology sample of the omentum showing abundant epithelioid granulomas with caseous necrosis (1) and giant cells on the periphery (2).

Despite being a relatively old disease, tuberculosis remains nowadays the World's third leading cause of death by an infectious agent. According to the latest report of the World Health Organization,[1] in 2013 nine million people developed TB and 1.5 million died from it. In recent years we are also witnessing another problem, the increasing incidence of multidrug-resistant TB (3.5% in new patients and 20.5% in previously treated). Although the initial target organ is the lung, tuberculosis is a systemic disease that can affect any organ.[2] The fact that this can occur after the first contact with the organism, in a second time or never, has to do with various adaptive mechanisms of M. tuberculosis, but also and largely with host factors.[3] In the case of intestinal tuberculosis, the organism reaches the abdominal organs through hematogenous dissemination, direct spread from adjacent tissues or, less commonly, through direct ingestion when near a TB focus (positive sputum-smear patients). It is worth noting that up to 25% of abdominal affectation presents itself with pulmonary tuberculosis, and that M. tuberculosis can implant anywhere in the abdominal cavity. This leads to varied and nonspecific clinical manifestations, making the diagnosis difficult.[4] There are four main forms of abdominal TB[5]; lymphadenopathy, peritoneal, gastrointestinal and visceral, being the most common manifestations the first two or a combination of them. It is important to realize that only 30% of cases have fever and that the most common clinical sign is ascitis.[6] For early diagnosis and treatment the biopsy is essential since it allows us to obtain histologic results, and most importantly, cultures and mycobacteria RCP (60–70% diagnosis sensitivity).[7] However, as we have seen in our case, the profitability of ascites fluid culture and even its RCP is low (15% and 7% respectively). In gastrointestinal manifestation cases all this gains special importance when making the differential diagnosis with other processes such as Crohn[8] disease or intestinal lymphomas. If, as in our case, endoscopy misses suspicious lesions' biopsies, laparoscopy becomes an essential tool in the diagnosis of abdominal TB with high yield and minimal risks compared with the high morbidity and mortality rate in non-treated patients pending completion of the study.[9]

Conflict of interest

The authors declare no conflict of interest.

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Right pleural effusion secondary to a pancreaticopleural fistula in a patient with asymptomatic chronic pancreatitis☆

Derrame pleural de predominio derecho secundario a fístula pancreatopleural en paciente con pancreatitis crónica asintomática

We present the case of a 51-year-old man, a smoker of 60 pack-years with a 20-year history of excessive alcohol consumption (180 g of ethanol per day), who had been diagnosed in another centre in 2011 with chronic pancreatitis with development of pancreatic pseudocysts.

He was admitted to the respiratory medicine department in November 2012 for a 7-day history of dyspnoea on moderate exertion and pleuritic pain in the right hemithorax. On examination, he was afebrile, with a cachectic appearance and spider naevi. Auscultation revealed dullness and hypophonesis in the right lung base. The abdomen was soft, palpable and non-painful, with no masses, megalias or findings suggestive of peritonism. Laboratory tests found: glucose 141 mg/dL, creatinine 0.69 mg/dL, total bilirubin 0.1 mg/dL, glutamic oxaloacetic transaminase 12 U/L, glutamic pyruvate transaminase 11 U/L, gamma-glutamyl transferase 23 U/L, alkaline phosphatase 79 U/L, amylase 1536 U/L, lipase 975 U/L, lactate dehydrogenase (LDH) 170 U/L, albumin 3.9 g/dL, sodium 143 mEq/L, potassium 4.2 mEq/L, pro-brain natiuretic protein 76 pg/mL, C-reactive protein 12 mg/L, haemoglobin 13.2 g/dL, haematocrit 41%, platelets $815 \times 10^3/mm^3$, leukocytes $12.2 \times 10^3/mm^3$ with $9.4 \times 10^3/mm^3$ neutrophils, international normalised ratio 0.91, and prothrombin time 118%. Chest X-ray and computed tomography (CT) (Fig. 1) revealed right pleural effusion occupying 45% of the hemithorax and a small left pleural effusion. Abdominal CT (Fig. 2) showed a pancreas with calcifications in the tail, body and uncinate process, with discrete dilatation of the duct of Wirsung related to known chronic pancreatitis. A 3.5-cm hypodense collection was observed in the pancreatic tail–splenic hilum, another 2.6-cm collection in the pancreatic head and another extending from the pancreatic body around the celiac trunk,

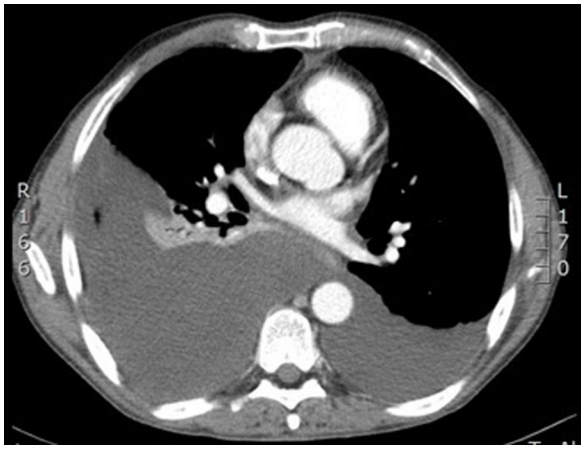

Figure 1 Right pleural effusion occupying 45% of the right hemithorax and a small left pleural effusion.

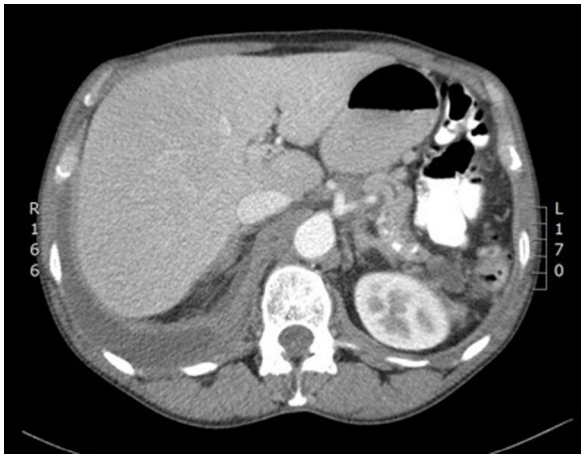

Figure 2 Pancreas with calcifications in the tail, body and uncinate process with discreet dilation of the duct of Wirsung.

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