A medida que avanza la medicina, nos enfrentamos cada vez con mayor frecuencia a pacientes polimedicados. Este caso subraya la importancia de la realización de una completa anamnesis, incluyendo siempre el tratamiento global del paciente con objetivo de que no pase desapercibido el origen farmacológico, puesto que como sucedió en este caso puede llegar a cursar con un cuadro de diarrea grave. Este ejemplo demuestra una vez más, que una precisa y cuidadosa historia clínica es la clave no solo para alcanzar el diagnóstico de la diarrea farmacológica, si no, para poder retirar el agente causante y lograr la curación de la misma.

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Conflicto de intereses

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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

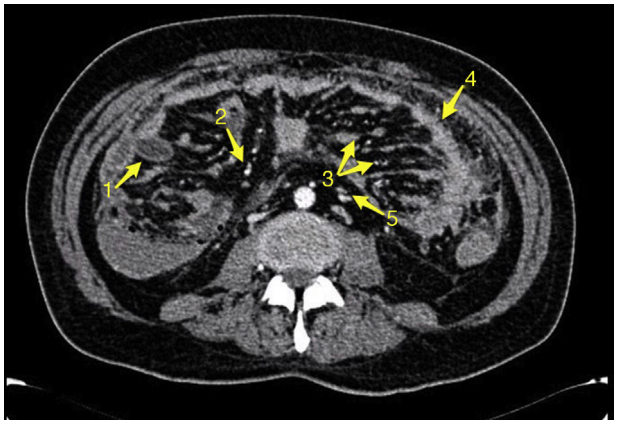

Figure 1 CT showing (1) blood content in jejunum, (2) multiple peritoneal implants, (3) omental cake, (4) thickening of the omentum, (5) lymphadenopathy.

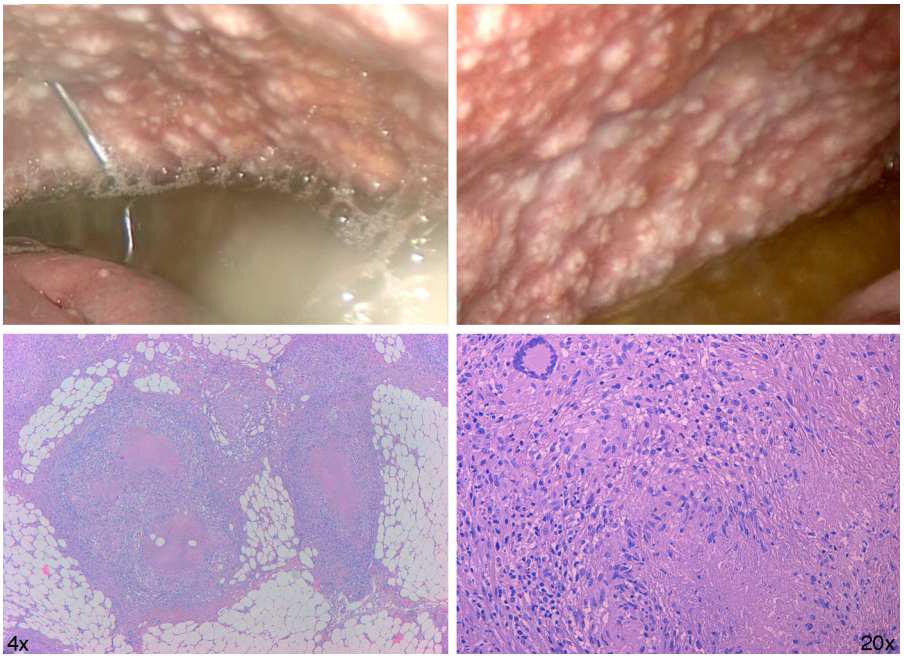

Figure 2 On the top, two captures showing laparoscopy with multiple nodules both parietal and visceral peritoneum. On the bottom, pathology sample of the omentum showing abundant epithelioid granulomas with caseous necrosis (1) and giant cells on the periphery (2).

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.

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

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

Presentamos el caso de un varón de 51 años fumador de 60 paquetes-año y bebedor de 180 g de etanol al día durante 20 años, diagnosticado en 2011, en otro centro, de pancreatitis crónica con desarrollo de pseudoquistes pancreáticos.

Paciente que ingresa en el servicio de neumología en noviembre de 2012 por cuadro de 7 días de evolución de disnea de moderados esfuerzos y dolor de tipo pleurítico en hemitórax derecho. A la exploración se trataba de un paciente afebril, de aspecto caquéctico, con arañas vasculares. A la auscultación destacaba matidez e hipofonesis en la base torácica derecha. El abdomen era blando, depresible, no doloroso, sin masas, megalias ni datos de peritonismo. Analíticamente presentaba: glucosa 141 mg/dl, creatinina 0,69 mg/dl, bilirrubina total 0,1 mg/dl, GOT 12 U/l, GPT 11 U/l, GGT 23 U/l, FA 79 U/l, amilasa 1.536 U/l, lipasa 975 U/l, LDH 170 U/l, albúmina 3,9 g/dl, sodio 143 mEq/l, potasio 4,2 mEq/l, proBNP 76 pg/ml, PCR 12 mg/l, hemoglobina 13,2 g/dl, hematocrito 41%, plaquetas 815 · 10^3/mm^3, leucocitos 12,2 · 10^3/mm^3 con 9,4 · 10^3/mm^3 neutrófilos, INR 0,91, tasa de protrombina 118%. En la radiografía y la TAC de tórax (fig. 1) se observaba un derrame pleural derecho que ocupaba el 45% del hemitórax y un pequeño derrame pleural izquierdo. En la TAC abdominal (fig. 2) se objetivó un páncreas con calcificaciones en cola, cuerpo y proceso uncinado con discreta dilatación del conducto de Wirsung en relación con su ya conocida pancreatitis crónica. Se visualizó una colección hipodensa en cola pancreática- hilio esplénico de 3,5 cm, otra de 2,6 cm en cabeza pancreática y otra que se extiende desde el cuerpo pancreático alrededor del tronco celíaco, por el retroperitoneo hacia el mediastino y el espacio pleural derecho. Se realizó una toracocentesis diagnosticoterapéutica, obteniéndose un líquido pleural con pH 7,38, amilasa de 95.000 U/l, glucosa 132 mg/dl, TG 24 mg/dl, proteínas 3,8 g/dl, LDH 508 U/l, ADA 25 U/l. Ante la sospecha de un pseudoquiste pancreático fistulizado a pleura se trasladó a nuestro servicio.

Se solicitó una colangioRM sin secretina para confirmación de la sospecha de fístula pancreática, objetivándose

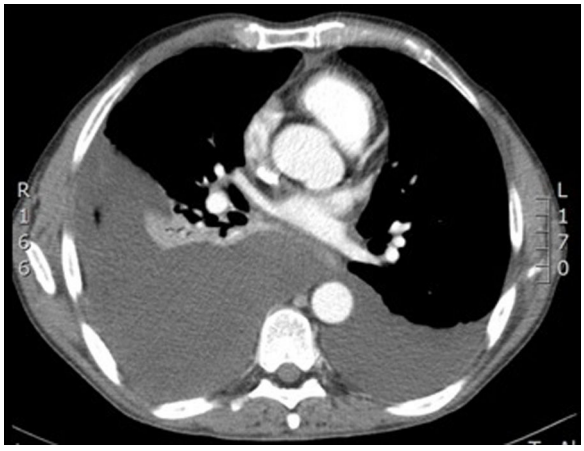

Figura 1 Derrame pleural derecho que ocupa el 45% del hemitórax derecho y un pequeño derrame pleural izquierdo.