Atypical Presentation of Pulmonary Tuberculosis in an Immunocompetent Patient☆

Tuberculosis pulmonar con presentación atípica en paciente inmunocompetente

To the Editor,

Tuberculosis (TB) continues to be one of the infectious diseases with the greatest morbidity in the world. Its incidence in Spain remains high due to constant demographic changes, and a greater proportion of the TB population are immigrants, who account for up to 30%–40% of patients diagnosed with TB.[1–4] Pulmonary TB is the most common form of involvement, but lesions can occur in other body systems.

We report the case of a 19-year-old Bulgarian woman, resident in Spain for 3 years. She had been evaluated in the clinic for iron-deficiency anemia and abdominal pain 6 months previously. An abdominal ultrasound was requested, which revealed slight splenomegaly and lymphadenopathies in the hepatic hilum and in the right iliac fossa. The patient did not return for a follow-up examination.

On the day of admission, she came to the emergency room with abdominal pain in the epigastrium and mesogastrium that had worsened during the previous week, fever, night sweats, and occasional dry cough. She reported loss of appetite with a 10 kg weight loss in the last 3 months. Examination revealed bilateral supraclavicular and laterocervical lymphadenopathies, and pain on palpation of the mesogastrium, with no other significant findings.

Blood tests were significant for CRP 24.4 mg/dl and microcytic anemia 94 g/l. Other blood count, coagulation, renal and hepatic parameters were normal. HIV, HBV, HCV and HAV serologies were negative. Standard chest radiograph (Fig. 1A) showed a bilateral diffuse micronodular pattern. A computed tomography of the cervical spine, chest, and abdomen was significant for posterior cervical lymphadenopathies in middle and low jugular chains, some of which were necrotic; the lungs (Fig. 1B) showed multiple irregular nodules randomly distributed predominantly in the upper and middle fields, with mediastinal lymphadenopathies in the hepatic hilum, celiac trunk, and para-aortic and inter-aortocaval nodes adjacent to the iliac chains. Given these findings, we requested an induced sputum test, which confirmed the presence of acid-fast bacilli (AFB) on Ziel-Nielssen staining in 3 different samples with subsequent growth of *Mycobacterium tuberculosis* on culture.

Treatment began with isoniazid, ethambutol, rifampicin and pyrazinamide. No resistances were observed to any of these drugs. The patient presented clinical and radiological improvement over the following weeks. After 2 months of treatment, negativization of AFB in sputum was confirmed, and treatment with isoniazid and rifampin continued for 4 more months.

TB is still present in our environment. The incidence rate of TB in the native Spanish population has declined in recent years. However, migratory flows observed since the beginning of the 21st century have led to an increase in the proportion of immigrants among the total number of TB cases diagnosed in our country, especially among those coming from countries with a high prevalence of TB.[2–5]

The incidence and prevalence of this disease are directly related to the degree of poverty, and eradication requires prevention, early diagnosis, and institutional support.[1,4] Some circumstances, such as coinfection with HIV, the increase in drug resistance, and geographical mobility complicate the efforts of international agencies to control this disease.[2] TB with pulmonary involvement is the most common form of presentation, and in extrapulmonary sites, the lymph nodes are the most common, followed by pleural, osteoarticular, miliary, genitourinary, peritoneal and central nervous system involvement. Symptoms include: fever; night sweats, either isolated or associated with fever; cough, which may be absent at the onset of the disease and be productive or not; dyspnea, if there is extensive involvement of the lung parenchyma; and anorexia and weight loss.[1,5] However, multiple clinical manifestations have been described, depending on the affected organ.[1]

Pulmonary TB can occur along with involvement at other levels, spreading by contiguity, hematogenous dissemination, or by swallowing infected lung secretions. Patients with abdominal involvement, as in our case, usually present with pain, anorexia, weight loss, and fever, while abdominal distention is less common. The most commonly affected abdominal regions are the hepatobiliary system, the peritoneum, and the ileocecal region, due to local implantation of the bacillus generally by the hematogenous route.[6]

If lymphadenopathies are detected in various sites, the differential diagnosis must include granulomatous lung diseases, such as TB, histoplasmosis, and sarcoidosis; connective tissue diseases, such as rheumatoid arthritis; and cancer (lung cancer, metastasis, Hodgkin's or non-Hodgkin's lymphoma).[7] TB with peritoneal lymph node involvement is usually seen in the peri-portal, peripancreatic, and mesenteric regions, causing local compression in

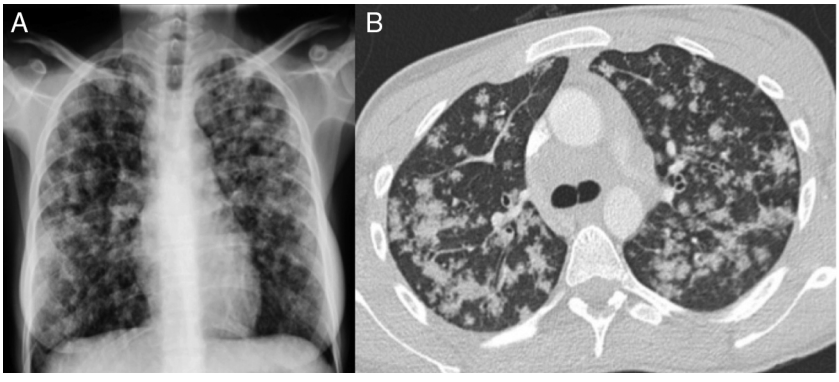

Fig. 1. Posteroanterior chest X-ray (A) with patchy pseudonodular opacities, predominantly in the middle and upper fields. No pleural effusion or mediastinal widening is seen. Chest CT cross-sectional slice (B) showing centrolobular pulmonary opacities with a tendency to coalesce, predominantly in the upper and middle fields.

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these sites.[8] Lymph node involvement in various locations accompanying the lung disease is usually more common in patients with HIV; this, however, and any other factor that would explain an associated immunosuppressive status were ruled out in our patient.

In our case, the radiographic study provided the key data, given the nonspecific nature of the initial symptoms. Bilateral pseudonodular lung lesions are similar to those observed in metastatic involvement ("cannonball metastasis"), however, the socioepidemiological characteristics of the patient led us to consider the presence of TB. These lung lesions have also been described in coinfections with other pathogens, such as cryptococcosis,[9] although this is more typical in immunocompromised patients. In our case, in the absence of pulmonary symptoms, sputum had to be induced to confirm the suspected diagnosis.

In conclusion, TB can occur with concomitant involvement in different sites, even in immunocompetent patients. It must therefore be included in the differential diagnosis when nonspecific symptoms with characteristic radiological data are encountered, particularly in patients with certain socioepidemiological backgrounds.

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Urinothorax and Chronic Renal Failure: A Rare Combination☆

Urinotórax e insuficiencia renal crónica: una rara asociación

To the Editor,

Urinothorax, a rare cause of pleural effusion (PE), consists of the presence of urine in the pleural cavity.[1–10] It is generally associated with obstructive urinary disease or urinary tract lesion, causing accumulation of urine in the retroperitoneal space, which is filtered to the pleural cavity. Pleural fluid (PF) is usually a transudate, ipsilateral to the lesion.[2–9] We report a case of recurrent, difficult-to-manage urinothorax in a patient with stage 4 chronic renal failure, with no apparent obstructive urinary disease. We discuss the pathogenesis of this entity and underline the importance of recognizing this exceptional clinical picture, not previously described in the literature.

This was an 89-year-old man, non-smoker with a history of arterial hypertension, dyslipidemia, and stage IV chronic kidney disease, with atrophic left kidney due to left renal artery stenosis. He was receiving anticoagulation for chronic atrial fibrillation, and had undergone transurethral resection for benign prostatic hypertrophy, and pulmonary thromboembolism in 2015. He presented in the emergency room for progressive worsening of dyspnea and pain in the left hemithorax, without no previous fever or catarrh. Physical examination showed tachyarrhythmia without murmurs, and auscultation of the lungs revealed signs of left PE. Chest X-ray showed PE occupying the lower two thirds of the hemithorax (Fig. 1). Laboratory tests showed 7000 leukocytes with 82.7% neutrophils; creatinine 2.15 mg/dl; total protein 5.56 g/dl; albumin 2.70 g/dl; LDH: 178 IU/l; and CRP: 18 mg/l. Ultrasound and computed tomography (CT) of the abdomen and pelvis showed no signs of hydronephrosis nor free liquid, and left kidney measuring 9 cm with irregular contour and atrophic appearance, and a small amount of peri-renal fluid. Renal Doppler showed intra-renal flows with increased resistance indices in both renal arteries, indicative of severe stenosis. Chest ultrasound showed moderate PE without septations, and a diagnostic and therapeutic thoracentesis was performed, yielding 1200 ml of cloudy, amber-colored PF, smelling of urine. The PF protein/serum protein ratio was 0.69 and the PF LDH/serum LDH ratio was 0.7, confirming the presence of exudate; pH was 7.11. Creatinine in PF was 3.4 mg/dl, with PF creatinine/serum creatinine ratio of 1.58, confirming the suspicion of urinothorax. Microbiological cultures and cytology were negative. Thoracentesis and Pleur-evac® were performed 5 times in the following 2 months due to recurrent PE. An intravenous dose of rifampicin was administered, and the PF drained by the Pleur-evac® was confirmed to be copper-colored, so a tunneled pleural catheter was implanted, which remained in place for 2 months, requiring 2 changes of the recipient every week. The patient died of multiple comorbidities and kidney failure not resolved by dialysis.

The first case of urinothorax was described in 1968 by Corriere et al.[1] and since then less than 100 reported cases have been reported worldwide. A recent systematic review includes 78 cases.[8] Urinothorax is probably underdiagnosed because of its low index of suspicion as a cause of PE.[4] Patients with a history of urinothorax usually present with obstructive disease or lesion of the urinary tract,[1–9] most frequently benign prostatic hyperplasia, hydronephrosis due to acute obstructive lithiasis, bladder

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