Pulmonary Lymphoma. A Case Report☆

Linfoma pulmonar: a propósito de un caso

To the Editor,

We report the case of a 78-year-old woman, no toxic habits, with a history of arterial hypertension, diabetes mellitus, dyslipidemia, and atrial fibrillation, who consulted due to a 1-month history of left chest pain, refractory to her usual analgesic treatment. Physical examination showed hypoventilation in the lower half of the left hemithorax. Moderate left pleural effusion was seen on the chest radiograph, and blood tests revealed elevated LDH and D-dimer. A diagnostic thoracocentesis was performed, obtaining pleural fluid consistent with predominantly mononuclear exudate with high LDH and normal ADA levels. Pleural fluid cytology was negative.

A chest-abdominal computed tomography (CT) was performed, which showed a soft tissue mass in the left posterior pleura, measuring 7.4×8.2 cm, extending along the chest wall and muscles of the left posterior abdominal wall, infiltrating all planes and the subcutaneous cell tissue, accompanied by significant ipsilateral pleural effusion. Significant cavoportal and retroperitoneal lymphadenopathies were observed in the abdomen. Given these findings, a CT-guided biopsy was performed of the lesion. Results were consistent with diffuse large B-cell non-Hodgkin's lymphoma, with a proliferation index of 70%, negative for EBV or *c-myc*. Bone marrow biopsy was normocellular, and HIV, HBV, HCV, and CMV serologies were negative.

Finally, a positron emission tomography was performed (Fig. 1), showing an extensive hypermetabolic focus, corresponding to a left pleural mass with an SUV(max) of 10.3, consistent with primary neoplastic lesion. Left axillary, abdominal (in the hepatic hilum), retropancreatic, and panaortic lymphadenopathies, suggestive of tumor infiltration, were also observed.

Diffuse large B-cell lymphoma, stage IV, AX, IPI 4, was diagnosed, and chemotherapy was started with rituximab, cyclophosphamide, vincristine, and prednisone (R-CVP), after tumor lysis prevention. In the follow-up CT, after 4 cycles of chemotherapy, the tumor had significantly reduced. A discrete increase in residual soft muscle planes and mild pleural effusion remained, but no pathologically enlarged lymph nodes or pleural thickening was observed.

Primary pleural lymphoma (PPL) is an uncommon entity, accounting for approximately 7% of lymphomas. It usually affects patients with HIV infection or chronic pyothorax. PPL in immunocompetent patients with no history of these conditions is exceptional.[1] Principal clinical symptoms are chest pain and dyspnea, if pleural effusion is significant. Radiological signs include diffuse nodular pleural thickening, accompanied by pleural mass. Sometimes isolated pleural effusion may appear before the development of the pleural mass.[2] For diagnosis, pleural biopsy should

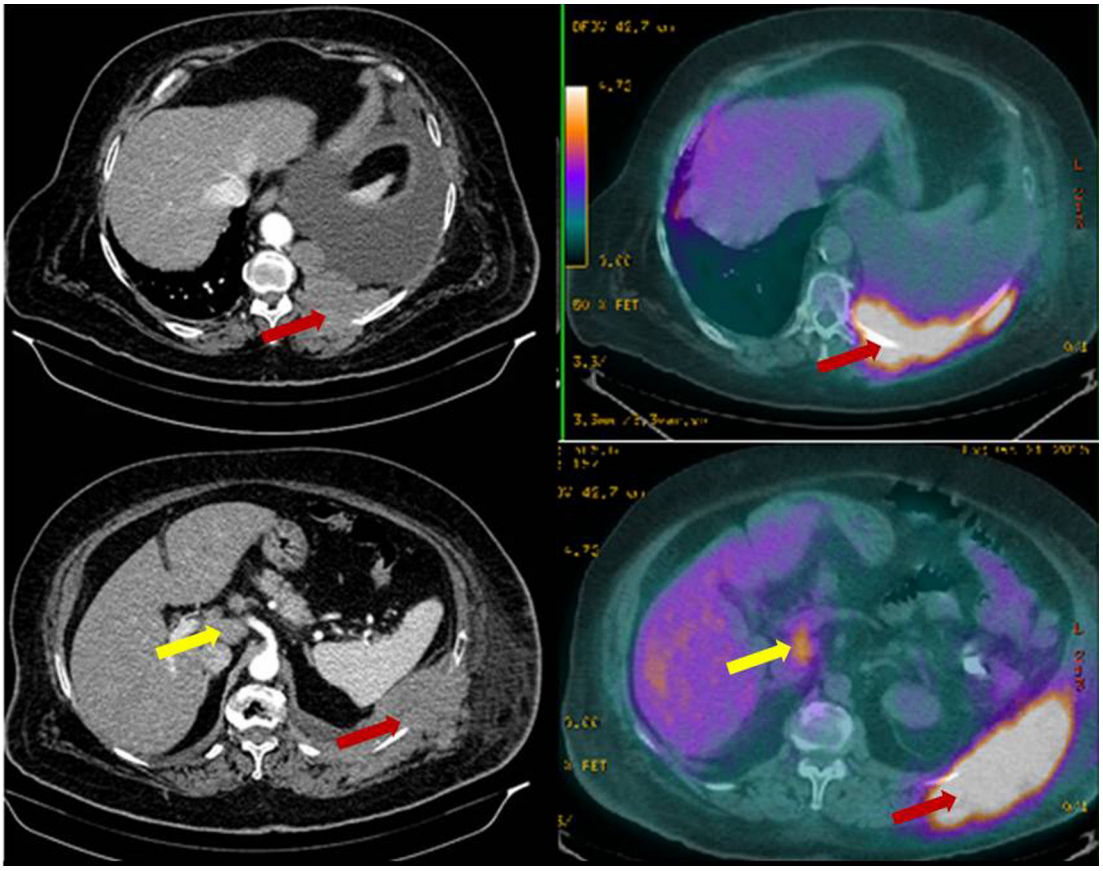

Fig. 1. Positron emission tomography: hypermetabolic left pleural mass, with SUV(max) 10.3 consistent with primary neoplastic lesion (black arrows) and abdominal lymphadenopathies in the hepatic hilum, and retropancreatic and para-aortic lymphadenopathies, suggestive of tumor infiltration (white arrows).

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be obtained under ultrasound or CT-guidance or by video-assisted thoracoscopy.[2] With regard to histology, any type of lymphoma may be involved, but the most commonly described type is large B-cell lymphoma, followed by follicular lymphoma, with rates of 60% and 20%, respectively.[3] Treatment requires systemic chemotherapy based on combinations of cyclophosphamide, doxorubicin, vincristine, and prednisone.

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Emphysematous Cystitis and Infectious Exacerbation of COPD. A Highly Unusual Finding☆

Cistitis enfisematosa y agudización infecciosa de la EPOC. Un hallazgo altamente infrecuente

To the Editor,

Emphysematous cystitis (EC) is an uncommon complication of urinary tract infection, defined by the presence of gas in the bladder and/or bladder walls.[1] It mainly affects elderly women with diabetes mellitus, poor blood glucose control, and a history of bladder catheterization. Other factors, while less important, may also be associated with this disease, such as renal transplantation, immunosuppression, and recurrent urinary tract infections.[1] The most commonly isolated causative pathogens on urine culture are *Escherichia coli*, *Klebsiella pneumoniae* and *Enterococcus*.[2] Clinical presentation is non-specific but most patients present urinary symptoms and abdominal pain. Pneumaturia is a key but infrequent pointer to suspecting EC.[3] For diagnosis, standard radiography of the urinary tract is unspecific, and the superior resolution and greater ability of computed tomography (CT) to rule out other causes of EC, such as enterovesical fistula due an inflammatory or malignant process, make it the examination of choice.[4] Initial treatment consists of empirical antibiotics, intensive metabolic control, and urinary diversion (transurethral catheterization or bladder drainage).[5] The clinical response of patients varies from rapid clinical improvement to general deterioration with a high rate of complications, such as emphysematous pyelonephritis, bladder perforation, septic shock, and rapid death.[5]

We report the unusual case of a male patient, non-diabetic, with normal blood glucose levels and no significant urological history, who presented EC during admission to the pulmonology ward for a severe COPD exacerbation caused by infection. *E. coli* was cultured from both sputum and urine samples. Our patient was a 66-year-old man, retired from employment in an iron foundry, who had given up smoking 5 years previously (70 pack-years). He had a history of childhood tuberculosis, arterial hypertension, moderate COPD, non-exacerbator emphysematous phenotype (GOLD category A) and, 5 years previously, community-acquired pneumonia. He reported a 5-day history of clinical symptoms of infection, with rhinorrhea, increased cough with purulent sputum, dyspnea, and fever 38.3 °C. He had no urinary symptoms and no changes in bowel habits. He also showed overall acute respiratory failure, with left-shift leukocytosis and increased PCR on clinical laboratory tests. Blood glucose was normal. Urine antigen testing for pneumococci and *Legionella* and sputum culture were performed, which were negative. The physical examination showed significant deterioration of the patient's general status, with tachypnea, and disperse rhonchi on lung auscultation. No signs of abdominal disease were observed at that time.

On day 3 of admission, the patient's clinical situation deteriorated, with symptoms of urinary infection (tenesmus, dysuria, and hematuria). An examination of urinary sediment was requested, showing abundant red blood cells per field and positive nitrites. Gram-negative antibiotic cover was administered, and urine culture was requested, which was positive for *E. coli*, so piperacillin–tazobactam was given, in line with the microbiological sensitivity results. A chest-abdominal computed tomography was performed to complete the study, revealing gas in the bladder lumen, infiltrating the walls (Fig. 1). After diagnosing EC, and following consultations with the urology department, we introduced a bladder catheter, obtaining 400 ml of urine with a slightly hematic tinge and abundant air. Intravenous piperacillin–tazobactam was also initiated and continued for 15 days. The urine culture was repeated after the antibiotic cycle, and results were negative. The bladder catheter was removed and the patient's abdominal symptoms resolved. Despite the initial improvement, respiratory

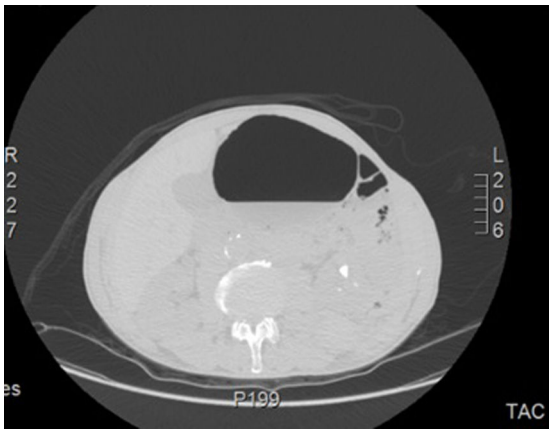

Fig. 1. Abdominal computed tomography showing gas in the bladder and the bladder wall.

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