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SCIENTIFIC LETTERS

Renal aspergillosis after liver transplant: Report of an unusual case☆

Aspergilosis renal posterior a trasplante hepático: informe de un caso infrecuente

Invasive aspergillosis (IA) with a compromised urinary tract is extremely rare. We present the case of a patient who developed renal abscesses due to *Aspergillus fumigatus* following liver transplant (LT).

28-year-old man who had an LT 6 months ago due to autoimmune fulminant liver failure. User of tacrolimus (0.1 mg/kg/day), prednisone 12.5 mg/day and trimethoprim–sulfamethoxazole. He was admitted after experiencing dull lower back pain for two days, extending to the flank and right iliac fossa. He had no fever or vomiting. On admission, he was haemodynamically stable, afebrile and lucid. On the physical examination by anatomical region, the abdomen was sensitive to deep palpation and fist percussion of the flank and right iliac fossa. The patient presented with no palpable masses or signs of peritoneal irritation.

The lab tests showed preserved liver and kidney function. Urinalysis revealed leukocyturia (more than 50 white blood cells/mm^3) with no bacteriuria. The CT scan of the abdomen and pelvis showed mild right-sided nephromegaly, with multiple irregular cystic lesions that spread into the pyelocalyceal system towards the third lower and middle segments. On the left side, a cystic lesion with slightly thickened walls was observed in the upper pole, measuring 2.5 cm in diameter. The urine culture and bacilloscopies were negative. The chest CT scan revealed no pathological findings.

The kidney fluid collections were drained and significant amounts of purulent material were found. Neither bacteria nor microbacteria were investigated during the microbiological tests. The Kinyoun stain was negative. However, *A. fumigatus* was isolated in the culture.

Voriconazole was initiated with a loading dose, followed by 4 mg/kg every 12 h, with a good initial clinical response. One month later, the patient developed fever and abdominal pain. Urinalysis showed persistence of the aseptic leukocyturia. The control CT scan revealed significant progression of the right kidney fluid collections. It was decided to perform a right nephrectomy, which was carried out without incident. The immunosuppressive therapy was also changed to rapamycin (2 mg/day) due to its antifungal effect. Voriconazole was discontinued after 9 months due to stabilisation of the left kidney lesion. To date, after 3 years of follow-up, the patient has suffered no relapses.

Invasive fungal infections (IFIs) are a significant cause of morbidity and mortality in post-transplant patients. In the case of those receiving liver transplants, the frequency ranges between 5.4% and 42%, with candidiasis being the most common aetiology, followed by *Aspergillus* spp.[1]

Between 1% and 10% of all cases of IFIs that occur in liver transplants are caused by *Aspergillus* spp.[2,3] The vast majority relate to cases of pulmonary aspergillosis. Of these, approximately half present with systemic dissemination.[1]

Renal compromise due to *Aspergillus* spp is uncommon; however, it is possible that there may be a sub-diagnosis of this condition in patients with IA. A *post-mortem* study in patients with IA revealed that up to 12% had inflammatory kidney lesions that were not previously investigated.[4]

To date, only two cases of renal aspergillosis have been reported in liver transplant patients.[5]

The risk factors for developing IA in liver transplant patients include kidney failure, repeat transplant, fulminant liver failure as a reason for transplant, cytomegalovirus or herpes-6 viral infection, and severe immunosuppression.[1,2]

The aetiopathogenesis of renal compromise is not fully known. Nevertheless, several hypotheses have been proposed, including: haematogenous spread from another point; haematogenous seeding (in users of intravenous drugs); compromise of the upper urinary tract; and local seeding in cases of prior surgeries of the urinary tract.

Leukocyturia with a negative urine culture, although non-specific, is typical in lab tests.[4]

In liver transplant patients, the death rate due to IA ranges between 33% and 100%.[2] Furthermore, the prognosis for renal compromise due to aspergillosis is poor. The majority of the cases reported in the literature show relapses and/or death despite treatment.[4] Even where the few cases of renal compromise reported in the literature failed to reach definite conclusions regarding treatment, patients who underwent radical nephrectomy seemingly had better survival rates. Radical nephrectomy was performed in the two reported cases of renal aspergillosis in LT, showing good progress with no relapses after 1.5 and 7 years of follow-up.[5]

Although IA is an uncommon disease, a high degree of suspicion is required and an active search for fungal infectious agents, including *Aspergillus* spp, should be performed.

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Lymphocytic esophagitis: A rare finding in adult patients with dysphagia and food impaction

Esofagitis linfocítica: una observación poco frecuente en pacientes adultos con disfagia e impactación alimentaria

Lymphocytic esophagitis (LyE) is a novel clinicopathological condition, first described in 2006 by Rubio et al. in Sweden,[1] in a series of 20 patients as a histological subset of chronic esophagitis characterized by >20 intraepithelial lymphocytes (IELs) per high-power field (HPF) with no more than rare granulocytes. Since then, a number of case reports from all over the world have confirmed the identity of this new condition.[2]

We report two recent patients from our center meeting clinical and histopathologic criteria compatible with LyE.

Case #1: A 52 yr-old male with smoking habit and a history of kidney stones presented with heartburn and non-progressive dysphagia over the past year. Upper endoscopy revealed no caliber or mucosal abnormalities and esophageal biopsies were taken to rule out eosinophilic esophagitis (EoE). Histopathologic analysis exhibited increased IELs increased (≥25/HPF) with marked basal–parabasal cell hyperplasia and intercellular edema (spongiosis). No eosinophils or neutrophils were detected (Fig. 1).

Case #2: A 63-year-old male with a history of hypertension, type 2 diabetes mellitus, ischemic cardiopathy and dyslipemia presented with recurrent esophageal food impaction requiring three emergency endoscopies. The patient denied previous heartburn and/or regurgitation. Esophageal endoscopic appearance was normal and esophageal biopsies revealed no eosinophilic but dense intraepithelial lymphocytosis (≥40/HPF) with papillar and peripapillar localization, and intercellular edema.

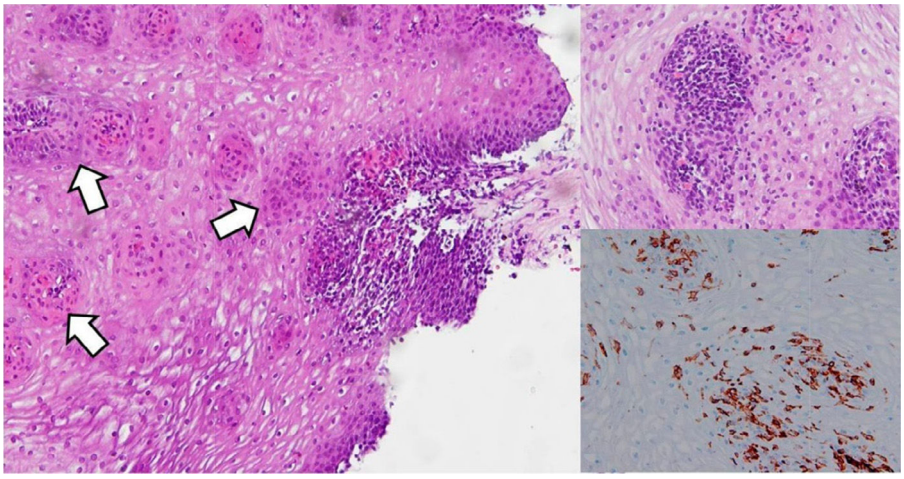

Figure 1 Left panel. Esophageal biopsy revealing heavy lymphocytic infiltration with papillar and peripapillar localization (white arrows) and intercellular edema (spongiosis), in the absence of either neutrophils or eosinophils. Right panel. A more detailed picture of lymphocytic infiltration, showing >25 CD4+ intraepithelial lymphocytes per 100 epithelial cells.