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A reflection on screening for neoplasms in glomerulonephritis☆

Reflexion sobre el screenning de neoplasias en glomerulonefritis

Dear Editor,

We present the case of a 34-year-old man, a smoker, without a personal or family history of interest, who attended the emergency department with oedema of a week of evolution, presenting a nephrotic syndrome with: proteinuria of 15 g/24 h, with microhematuria, serum creatinine 0.9 mg/dl, eGFR > 60 ml/min/1.72 m^2 (MDRD), albumin 1.9 g/dl, cholesterol 557 mg/dl, leukocytes 5,500/μl (normal formula), haemoglobin 15.8 g/dl and 239,000/μl platelets.

He reported itching of during the last 2 months, so he took an antihistamine with a partial response. He had no skin lesions, and had no remarkable findings on physical examination.

In the complementary tests, the autoimmunity study was negative (anti-nuclear, anti-DNA, anti-neutrophil cytoplasm antibodies, antibodies against extractable nucleus antigens) and normal complement values. IgG levels were decreased: 393 mg/dl (normal range 700–1,600) with IgA and IgM values being normal, and a notable elevation of IgE levels : 565 IU/ml (< 100). The chest X-ray and abdominal ultrasound showed no pathological findings.

A renal biopsy was performed evidencing a minimal change disease.

Treatment was started with prednisone 60 mg/day obtaining a complete response 2 months after treatment (proteinuria 0.1 g/24 h) and starting a descending regimen thereafter. In the fifth month, while on 30 mg of prednisone, he suffered a relapse of the nephrotic syndrome (proteinuria 8 g/24 h), so the dose of prednisone was increased again to 60 mg/day with partial response at 2 months (proteinuria 0.6 g/24 h). In the descending regimen, with prednisone 40 mg/day, he relapsed again with proteinuria of 8.5 g/24 h, he was catalogued as a corticodependent and treatment with tacrolimus was started.

He was referred to the allergy department for the pruritus and high levels of IgE, and it was detected a cat hair allergy. He lived with multiple cats, so its causality with pruritus seemed reasonable, and he got rid of them.

The patient commented on the onset of atypical chest pain, which worsened with movements. The ECG showed no pathological changes. His wife attributed the pain it to the anxiety he had because of the clinical picture. Given the persistence of pain, he was assessed by cardiology and a stress test was performed that was clinically and electrically negative.

Given the increase in pain intensity, he went to the emergency department where they performed a CT scan to rule out pulmonary thromboembolism and pathological mediastinal adenopathies were detected.

Biopsy of the adenopathy showed classic nodular sclerosis Hodgkin lymphoma, stage II-A, with viral load of the Epstein-Barr virus (EBV) 11,200 IU/ml. He was treated with polychemotherapy (ABVD) and rituximab, with a very good response and a complete remission of lymphoma after the third cycle of chemotherapy, negativization of EBV and complete response of the nephrotic syndrome without the need for specific treatment. After one year of follow-up, the patient continues in complete remission of lymphoma and nephrotic syndrome.

The relationship of glomerulonephritis with a neoplastic process is well known. It was described for the first time in 1966,[1] and has been repeatedly confirmed over the years.[2,3] It often represents the first clinical manifestation of an underlying cancer. It has even been described that, sometimes, the neoplasia does not manifest itself until months or even years after the diagnosis of glomerulonephritis,[2,3] reasoning that a molecular phenomenon exists very early on in the neoplastic process.[4]

In a study that assessed 5594 patients with different forms of glomerulonephritis, there were found 911 neoplasms of

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multiple aetiologies, in parallel to the diagnosis of glomerular disease, with a prevalence of 5.5 % (with the expected being 3.1 %). After a 10-year follow-up, the incidence of neoplasm in this population was 1.8 times higher than expected, without being able to rule out the influence of the immunosuppressive treatment received (both by the treatment itself or by accelerating the existing neoplastic process).[2]

Given our case and the evidence in the literature, we wanted to reconsider on how nephrologists perform the screening of neoplasms in a patient with new diagnoses of glomerulonephritis. The KDIGO guidelines[5,6] recommend the screening of neoplasms in the case of membranous nephropathy, GEFyS and IgA nephropathy, but not in other forms, and without specifying the recommended tests to perform. Some authors propose an exhaustive study that includes a first level: chest x-ray, skin examination, breast/testicular examination, abdominal and cervical ultrasound, faecal occult blood (if positive: colonoscopy/gastroscopy). If everything is negative on a second level: mammography and gynaecological review; cystoscopy if haematuria; PSA and rectal exam (prostate biopsy if neoplasia is suspected). If everything is negative and in patients with a high risk (> 60 years, smokers, alcoholic; thromboembolism; HIV/HBV/HCV infection; prolonged immunosuppressive treatment; negative anti-PLA2R antibodies in Membranous GN): CT scan, colonoscopy, fibroendoscopy should be performed. And they recommend repeating the screening in patients who have received or receive prolonged immunosuppressive treatment, every 5 years if patients are < 60 years and every 3 years if> 60 years.[7]

According to the evidence described in the current study[2] with the various forms of glomerulonephritis and the multiple associated cancer, we suggest that the suspicion and investigation of the nephrologist should be greater, so as not to delay the diagnosis of an underlying cancer and to avoid the harmful consequences of an immunosuppressive treatment that can accelerate the development of neoplasia.

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Aureimonas altamirensis: The first case of peritonitis on peritoneal dialysis☆

Aureimonas altamirensis: primer caso de peritonitis en diálisis peritoneal

Dear Editor,

Aureimonas altamirensis (*A. altamirensis*) is a bacterial specie from the caves of Altamira, Cantabria, Spain; considered as an environmental pollutant, but isolated for the first time in biological samples in 2008 by Luong et al., it is a potential human pathogen, with a variable clinical presentation.[1,2]

A case of peritonitis due to *A. altamirensis*, isolated in the ascites fluid of an oncological patient, has been described in the literature,[3] no cases of peritonitis due to this micro-

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