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Letters to the Editor

Renal lymphomatous infiltration in patient with nefrotic syndrome☆

Infiltración renal linfomatosa en paciente con síndrome nefrótico

Dear Editor,

We describe the case of a patient with renal disease associated with a B-cell lymphoid infiltrate, with no associated systemic disease and with the presence of monoclonal IgM-kappa paraprotein.

Renal involvement is known to be associated with lymphoplasmacytic neoplasia and is not considered rare (14–34% depending on whether the biopsy is *pre mortem* or *post mortem*), but it often goes undiagnosed due to the absence of symptoms and sometimes the lack of biopsies performed in these patients.[1] However, the occurrence of primary low-grade B-cell lymphoma of mucosa-associated lymphoid tissue (MALT) is considered unusual.[2] These tumours were initially described by Isaacson and Wright[3] in the gastrointestinal tract, and later in the thyroids, lungs and salivary glands. More recently, B-cell MALT lymphomas have been reported in a large variety of locations, including the urogenital tract, although as mentioned, their occurrence is very rare.[3] The presence of a glomerular lesion associated with lymphoplasmacytic neoplasia is not uncommon, and could be the direct result of the lymphoplasmacytic disorder through deposition of a paraprotein (amyloid) or monoclonal immunoglobulin, or its origin could be mediated by an immune mechanism that causes membranoproliferative, membranous or minimal change glomerulonephritis (GN).[4] There are reported cases of isolated renal involvement with no evidence of systemic disease, haematological malignancy or associated autoimmune disease which are sometimes associated with a serum monoclonal IgG or IgM-kappa component.[5]

This patient was a 71-year-old man admitted because oedema. He had a 1-year history of hypertension and post-traumatic subdural haematoma drained 6 years earlier. Due to the presence of renal failure (serum creatinine 2.5 mg/dL) with of nephrotic syndrome (albumin: 1.9 g/dL; total cholesterol: 315 mg/dL; urinary protein: 6.6 g/24 h) and normal-sized kidneys by ultrasound, a renal biopsy was performed. Other additional tests showed elevated beta-2 microglobulin and lactate dehydrogenase, serum protein electrophoresis and immunofixation with the presence of monoclonal IgM-kappa gammopathy (monoclonal component 0.2 g/dL) and urine negative for Bence-Jones protein. During hospitalisation, the patient presented a deterioration in renal function (Cr 3.8 mg/dL). Treatment was therefore initiated with oral prednisone at a dose of 1.5 mg/kg/day plus diuretics (furosemide and spironolactone) and angiotensin II receptor blockers in an attempt to control the proteinuria.

The renal biopsy (Figs. 1 and 2) revealed the presence of: (1) dense lymphoid infiltrates, immunophenotypically atypical, suggestive of a low-grade B cell lymphoproliferative process, and (2) mesangioproliferative glomerulonephritis, with few exudative changes and no demonstrable immune deposits.

Given the biopsy findings and the presence of monoclonal IgM-kappa gammopathy, the haematology department was consulted to assess the presence of a possible chronic lymphoproliferative process that had associated renal infiltration, as the most likely cause of the symptoms. Iliac crest biopsy was performed, with a diagnosis of mixed mature B and T cell lymphoid infiltrate, probably reactive.

The patient recovered renal function quickly with oral prednisone and diuretics, achieving normal renal function (1.1 mg/dL) and a reduction of proteinuria to 260 mg/day in 2 months, despite persistence of the monoclonal IgM-kappa component (0.1 g/dL). After 9 months of follow-up by nephrology and haematology departments, he continues to have no evidence of progression of the lymphoproliferative process, with normal renal function and persistence of the monoclonal IgM-kappa component (0.1–0.2 g/dL).

In our case, there was no evidence (clinical, analytical or imaging tests) that made us suspect renal lymphoma.

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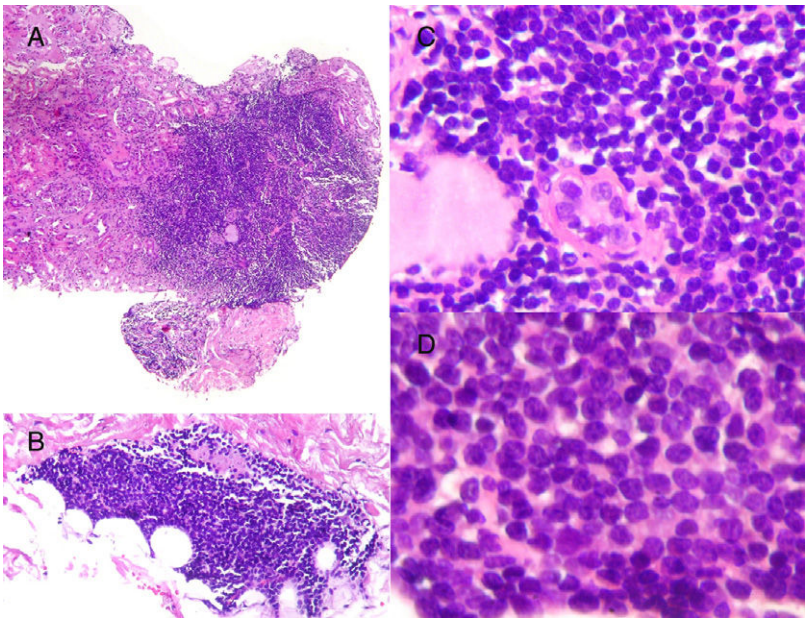

Fig. 1 – Histological characteristics of the lymphoid infiltrates that affected both the renal parenchyma (A: H&E, ×10) and the perirenal tissue (B: H&E, ×10). The lymphoid infiltrate had destroyed the parenchyma, leaving isolated residual tubules with images that may suggest lymphoepithelial lesion (C: H&E, ×20). Cells were monomorphic, small in size, with an irregular nuclear outline, barely visible nucleolus and no significant mitotic activity (D: H&E, ×40).

The biopsy was suggestive of a low-grade B cell lymphoproliferative process together with mesangioproliferative glomerulonephritis with no demonstrable immune deposits. The literature describes the association of lymphomas with numerous forms of glomerulonephritis: membranous GN,[4] membranoproliferative GN[4,5] and minimal change GN.[4] In many cases, like ours, the clinical presentation is determined by the renal disease associated with the lymphoproliferative process, presenting nephrotic proteinuria with or without associated renal insufficiency.[5] Our patient started with nephrotic syndrome secondary to the presence of mesangioproliferative GN; therefore, the initiation of steroid treatment

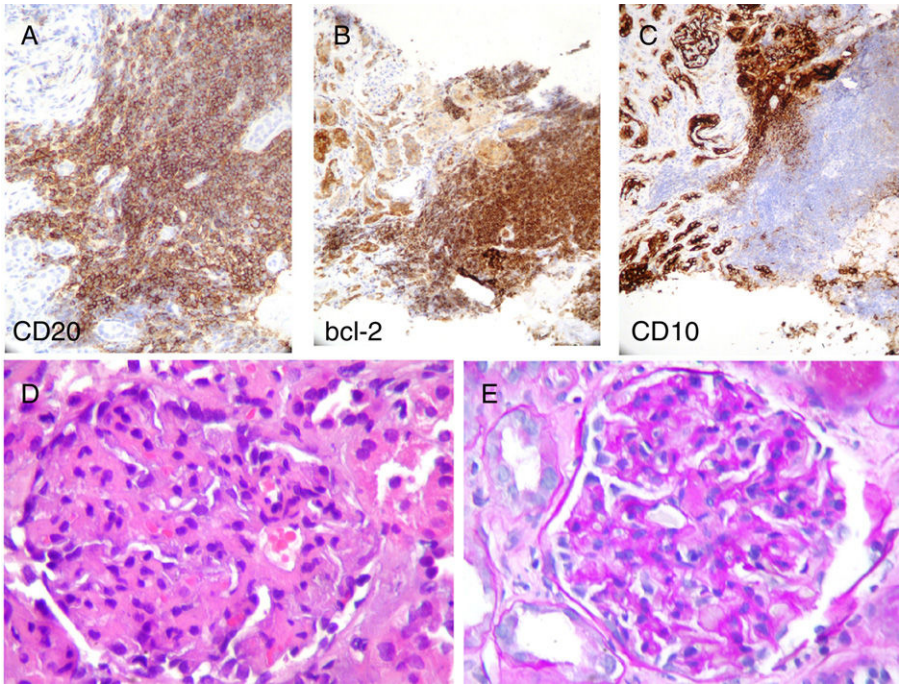

Fig. 2 – The lymphoid population expressed CD20 and bcl-2, with negative staining for CD10 and other B lymphoid lineage markers. (A–C: immunohistochemistry technique indicated in figure, ×20). In preserved normal renal parenchyma, it can see lobular glomeruli with increased matrix and, to a lesser extent some mesangial cellularity with occasional images of endocapillary proliferation (D: H&E, ×40; E: PAS-diastase, ×40).

from the time at which we performed the renal biopsy and a favourable response to steroids resulted in the rapid recovery of renal function (Cr 1.1 mg/dL) with disappearance of the proteinuria (260 mg/day) in only 2 months.

Following the result of the renal biopsy, the haematology department was consulted. Having ruled out an associated chronic lymphoproliferative process, and given the absence of symptoms, we decided to adopt a watch and wait approach with close follow up of the patient's clinical progress. After 9 months, the patient remained asymptomatic, with normal renal function and no proteinuria, with persistence of the monoclonal IgM-kappa component. Although the initial diagnosis suggested a poor short-term prognosis, the absence of systemic involvement seems to have contributed to a favourable outcome.

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Enoc Merino García*, María Pilar Pérez del Barrio, Josefa Borrego Hinojosa, Francisco J. Borrego Utiel, María Carmen Sánchez Perales

Unidad de Gestión Clínica de Nefrología, Complejo Hospitalario de Jaén, Jaén, Spain

* *Corresponding author.*
E-mail address: enocmerino@gmail.com (E. Merino García).

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Serratia marcescens bacteraemia outbreak in haemodialysis. Comment on "Serratia marcescens bacteraemia outbreak in haemodialysis patients with tunnelled catheters due to colonisation of antiseptic solution. Experience in 4 hospitals"☆

Brote de bacteriemia por Serratia marcescens en hemodiálisis. Comentario a «Brote de bacteriemia por Serratia marcescens en pacientes portadores de catéteres tunelizados en hemodiálisis secundario a colonización de la solución antiséptica. Experiencia en 4 centros»

Dear Editor,

We read with particular interest the article published recently by Merino et al., entitled "*Serratia marcescens* bacteraemia outbreak in haemodialysis patients with tunnelled catheters due to colonisation of antiseptic solution. Experience in 4 hospitals",[1] to which we would like to contribute our experience.

As reported in the aforementioned article,[1] between December 2014 and January 2015, we recorded several cases of catheter-related bacteraemia (CRB) due to *Serratia marcescens*

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