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Mesangial and endocapilar proliferative glomerulonephritis and scabies infection: A causal or circumstantial relationship?

Glomerulonefritis proliferativa mesangial y endocapilar e infección por escabiosis. ¿Una relación causal o circunstancial?

Dear Editor,

IgA-dominant postinfectious glomerulonephritis (PIGN) is a morphological variant of PIGN that is increasingly common in our environment. Unlike classic post-streptococcal PIGN, in which there is deposition of C3 and IgG or C3 only, it is characterised by the dominant deposition of IgA at the glomerular level. It usually coexists with less intense positivity for IgG, IgM or C3, and histological patterns of mesangial or extracapillary proliferation have also been described.[1,2]

IgA PIGN mainly affects elderly men with different comorbidities, mainly diabetes mellitus, but also tumors, alcoholism or HIV[2] infection. It is more aggressive than the classic variant, with a poor prognosis and in most cases (70–80% begins with acute renal failure, proteinuria, hematuria and hypocomplementemia). The differential diagnosis should be made with post-streptococcal PIGN and IgA nephropathy. It is usually associated with active infections caused by *Staphylococcus aureus*, frequently methicillin-resistant, but there are also cases due to other staphylococci, and some are occasionally due to gram-negative bacteria of urinary origin and parvovirus B19.[3,4] The most common focus is cutaneous, but there are descriptions of respiratory, bone, urinary and cardiac infections.[5–7]

Here we are presenting the clinical case of a patient with a pathological diagnosis of IgA PIGN after cutaneous superinfection in the context of scabies.

She is a 59-year-old woman with no personal history of interest, except for follow-up by dermatology in the context of a pruritic skin reaction, classified after a skin biopsy as chronic eczematous dermatitis. She inniciated treatment with cyclosporine, with inadequate response; also she received treatment with ivermectin due to skin superinfection of *Sarcoptes scabiei* (Norwegian scabies associated with immunosuppressive treatment).

After 3–4 weeks of starting antiparasitic treatment, she went to the emergency room due to oedema, dyspnoea on minimal exercise and decreased diuresis, with little response to depletion treatment. The analytical control revealed acute renal failure (urea 176 mg/dl, creatinine 2.28 mg/dl, that were previously normal, and a serum K of 6 mEq/dl) with nephritic syndrome (albumin 2.8 g/dl, triglycerides 324 mg/dl, protein/creatinine ratio (PCR) 16,116 mg/g, haematuria ++ and HTN that is difficult to manage). Regarding the rest of the complementary tests, the proteinogram and complement were within range (C3 155.7 mg/dl, C4 32 mg/dl), with increased IgA (629 mg/dl), and autoimmunity and viral serologies were negative. A renal ultrasound was performed, which reported normal-sized kidneys with preserved cortical thickness without duct dilation. Subsequently, a progressive deterioration of renal function was observed (peak creatinine 3.4 mg/dl), thus an ultrasound-guided renal biopsy was performed with an anatomopathological result of mesangial and endocapillary proliferative glomerulonephritis with an epithelial crescent and deposits of C3 and IgA at the glomerular level

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in immunofluorescence, starting treatment with boluses of methylprednisolone for 3 days and subsequently prednisone 1 mg/kg/day (80 mg/24 h) with a descending regimen. After the start of treatment, she presented a favourable clinical evolution, with a resolution of oedema, progressive improvement of kidney function, and resolution of the skin condition, requiring no monitoring by the Dermatology department during hospital admission.

The patient is currently being followed-up by Nephrology, presenting stable kidney function with glomerular filtration of 41.05 ml/min (using CKD-EPI) and non-nephrotic proteinuria without requiring immunosuppressive treatment.

The importance of our clinical case lies in the infrequent appearance of IgA PIGN secondary to a skin superinfection due to scabies. Scabies is usually difficult to detect and produces an intense itchy skin disease, and may affect anyone of any age or level of personal hygiene. In relation to the clinical context of the patient, cases of PIGN associated with scabies have been described in adults, mainly documented in the early 1980s, without histopathological evidence of renal involvement in these publications, unlike our clinical case, in which the main predisposing factor was previous immunosuppressive treatment.[8] To date, there is no scientific evidence that recommends the routine use of steroids in the treatment of IgA PIGN, since it could increase mortality in patients with active infection. However, considering our case, treatment with corticosteroids should be contemplated in those patients with acute renal failure that do not improve after appropriate antibiotic treatment as There are additional case series reported [9,10] in which steroid treatment was associated to a progressive improvement of renal function.

Ethical considerations

The authors declare that they have followed the protocols established in their place of work to access the patient's clinical records in order to create this manuscript for dissemination within the scientific community.

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Conflicts of interest

The authors have no conflicts of interest to declare.

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