Grover's disease in a peritoneal dialysis patient

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To the Editor:

Grover's disease is a dermatosis, which is characterised by erythematous, pruritic maculopapular exanthema, mainly on the trunk. Chronic kidney disease under dialysis is a predisposing condition due to mechanisms that have not been fully explained. We report the case of a 68-year-old male with polycystic kidney disease who had been on continuous ambulatory peritoneal dialysis for six months, with benign prostatic hyperplasia that caused multiple recurrent urinary tract infections. He received prophylactic treatment with trimethoprim/sulphamethoxazole (TMP-SMX). He sought consultation due to 38°C fever, general discomfort, asthenia and, 48 hours previously, he had developed erythematous pruritic maculopapular lesions on his trunk and subsequently on his limbs. On physical examination, the patient was afebrile, with erythematous maculopapular rash on his trunk and limbs (Figure 1). There were no relevant findings in the remaining physical examination. Complementary examinations showed bicytopenia with no other findings. A skin biopsy was performed (Figure 2), revealing suprabasal acantholysis with the presence of dyskeratotic cells in the superficial layers in the form of grains and round bodies and mild dermal perivascular inflammatory infiltrate in the superficial dermis; these findings are consistent with Grover's disease. Icodextrin was replaced with dextrose-based solutions, sulphonamide was discontinued and there was remission of skin lesions and bicytopenia. With TMP-SMX initially being considered as a potential ethological agent, the dialysis solution with icodextrin was reintroduced and the skin exanthema returned in a week. Improvement was observed with the introduction of topical corticosteroids and the discontinuation of icodextrin.

Transient and persistent acantholytic dermatosis (TAD) was initially described by Ralph Grover as a transient papulovesicular rash that compromised the trunk and thighs.[1] It is usually self-limiting[2] and primarily affects Caucasian adults from the fifth decade of life with a 1.6 to 2.1 male/female ratio.[3] Its aetiology is unknown and has been associated with overexposure to the sun, feeling hot, fever, sweating, states of immunodeficiency, neoplasias and other dermatoses.[1] Some cases have been associated with drugs, among them, sulphadoxine/pyrimethamine.[4] Chronic kidney disease receiving treatment with dialysis is a recently identified associated condition. Until present, TAD has been reported in seven haemodialysis patients and only in two patients on peritoneal dialysis.[2] In these cases, the lesions were persistent, apart from in four patients, in whom renal transplantation was performed. In most cases Grover's disease mainly affects the trunk. The rash is papular, erythematous and intensely pruritic. Histopathologically, the common denominator is acantholysis, defined as the dissociation of keratinocytes from the epidermis.[4] The differential diagnosis is made with genodermatosis, pemphigus and Galli-Galli disease. Topical corticosteroids, antihistamines, moisturisers and emollients are the first line therapeutic agents. In persistent or recurrent cases, the following is recommended: systemic corticosteroids, topical vitamin D analogues, systemic retinoids, phototherapy and PUVA (psoralen plus ultraviolet A photochemotherapy).

Figure 1. Erythematous maculopapular rash on the trunk

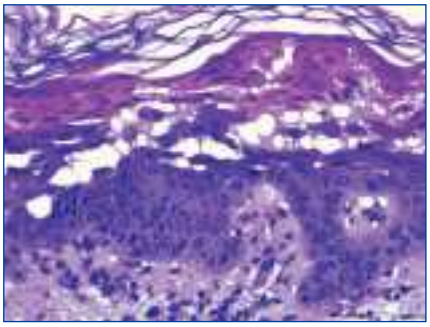

Figure 2. Skin biopsy consistent with Glover's disease.

This case was initially targeted as TAD secondary to sulpha drugs. However, our attention is drawn to the exacerbation and subsequent remission of symptoms with the reintroduction and then discontinuation of icodextrin-based dialysis solutions. The use of icodextrin in peritoneal dialysate has been associated with the onset of erythroderma, atopic dermatitis and other generalised rash exanthemas.[5] TAD should be considered in the differential diagnosis of skin diseases in chronic kidney disease patients who receive renal replacement therapy.

Conflicts of interest

The authors declare that they have no conflicts of interest related to the contents of this article.

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Acute renal failure induced by acute interstitial nephritis secondary to cocaine

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To the Editor:
Cocaine has been used by 2.6% of the Spanish population aged between 15 and 64 at some point in their life, making it one of the most consumed illegal drugs after cannabis.[1] Cocaine use is associated with multiple complications: neurological, cardiovascular, psychiatric, pulmonary, gastrointestinal and nephrological.

Renal complications associated with cocaine use have received little attention, despite the existence of several mechanisms, in addition to secondary high blood pressure, that can cause acute renal failure (ARF) or worsen a pre-existing case of chronic renal failure.[2]

Drug-induced acute interstitial nephritis (DIAIN) represents a high percentage of acute renal failure in clinical practice. Some studies indicate that DIAIN is the lesion responsible for renal failure in about 15% of biopsies with ARF. Furthermore, in many cases of DIAIN, no biopsy is performed and diagnosis is based on clinical data and recent administration of a new drug which, as described below, is sometimes not very easy to identify.[3-5]

CASE REPORT

28-year-old male, admitted with pain at the dimples of Venus, fatigue and nausea, with preserved diuresis.

The patient had used intranasal cocaine (1g) five days before admission. He denied having taken non-steroidal anti-inflammatory drugs or other medication. The physical examination showed a good general condition, with slightly high blood pressure of 147/97mmHg and without fever, rash or arthralgia.

Cardiovascular and respiratory examinations were normal. The abdomen was soft, depressible and painless and the liver was palpable 1cm below the costal margin and there was slight pain on bilateral palpation of lower back.

The initial blood test showed an unremarkable complete blood count (without eosinophilia), normal liver function and albumin within the normal range, serum creatinine: 160µmol/l, urea: 7.5mmol/l, potassium: 3.9mmol/l, sodium: 139mmol/l chloride 101mmol/l. Total creatine phosphokinase was normal (3.3µkat/l) with normal MB fraction. Urine sediment showed 2 leukocytes and 3 erythrocytes per high power field and no dysmorphic erythrocytes or eosinophils. Urine biochemistry: sodium: 46mmol/l, potassium: 33mmol/l and chloride: 63mmol/l, protein ratio: creatinine 5g/mol, negative urine culture.

Protein electrophoresis, immunoglobulins, complement, levels of angiotensin converting enzyme and antinuclear antibody titres were normal. Serology for human immunodeficiency virus, Epstein-Barr virus, cytomegalovirus, hepatitis A, B, and C and mycoplasma did not detect active infection. The ultrasound showed normal-sized, diffusely echogenic kidneys with appropriate arterial and venous flow.

The electrocardiogram was normal. The chest x-ray showed a cardiothoracic ratio <0.5 and lung fields without infiltrates.

After admission, urinary output remained at 50 to 75ml/h and creatinine remained unchanged. The patient underwent a renal biopsy.

Histological findings are as follows: optical microscopy showed a total of 13 glomeruli, all normal, without sclerosis, proliferation or necrotic lesions (Figure 1). Basement membranes and the glomerular mesangium were normal. The interstitium displayed moderate mononuclear inflammatory infiltrate with abundant eosinophils (Figure 2), with presence of focal tubulitis and atrophy (Figure 2). The arterioles did not display remarkable lesions and immune deposits were not shown in the immunofluorescence.

The findings were compatible with the pathological diagnosis of acute tubulointerstitial nephritis (ATN).

This fact, along with the clinical characteristics and recent use of cocaine led us to define this case as cocaine-induced AIN.

The patient obviously suspended drug use and was treated with oral prednisone (initial dose 1mg/kg/day), which was progressively decreased and discontinued after 12 weeks.

In the subsequent follow-up, his progression was good with a gradual improvement in renal function until complete recovery in the month in which treatment started.

DISCUSSION

We report the case of a patient with ARF, with acute tubulointerstitial lesion associated with DIAIN, in which no re-