cannot be prevented.[9,10] AD is a specific complication in patients with medullary injury that can involve risk of death, therefore a hypertensive crisis must be known and suspected of in this group of patients. Its management will be focused on its detection and avoiding the triggering cause of hypertensive crisis.

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Grover's disease in chronic kidney failure☆

Enfermedad de Grover en fracaso renal crónico

Dear Editor,

Patients with chronic renal failure may present multiple cutaneous manifestations such as pruritus, xerosis, cutaneous pigmentation, metastatic calcinosis cutis, calciphylaxis, pseudoporphyria and late cutaneous porphyria. Grover's disease (GD) must also be included in the differential diagnosis of cutaneous lesions in these patients.

We are presenting a case of a 69-year-old woman who presented with one and a half moth of cutaneous lesions in the back with moderate itching. She had no personal or family history of skin pathology. She had been receiving haemodialysis for five months due to Good pasture syndrome induced rapidly progressive renal failure. During the previous months, she had been treated with plasmapheresis, IV cyclophosphamide and IV methylprednisolone. At the time of consultation, her treatment included prednisone 5 mg/day, calcitriol 0.25 mg/day, omeprazol 20 mg/day, enoxaparin sodium 20 mg 3 days/week and darbepoetin alfa 30 mcg weekly. Physical examination shows non-confluent papular erythematous lesions with keratotic surface, located in the back (Fig. 1a). She did not have lesions on palms or soles, back of the hands or the oral mucosa. She did not have a facial or scalp lesions either. Microscopic evaluation showed areas of hyperkeratosis with parakeratosis, acanthosis and focal acantholysis with presence of round and granular lesions (Fig. 1b). Taking into account the almost asymptomatic character of the lesions, the patient preferred to adopt an expectant attitude. Three months later, the lesions disappeared spontaneously. The absence of a previous family history, the onset at an adult age, and the spontaneous resolution of the symptoms led to the diagnosis of GD.

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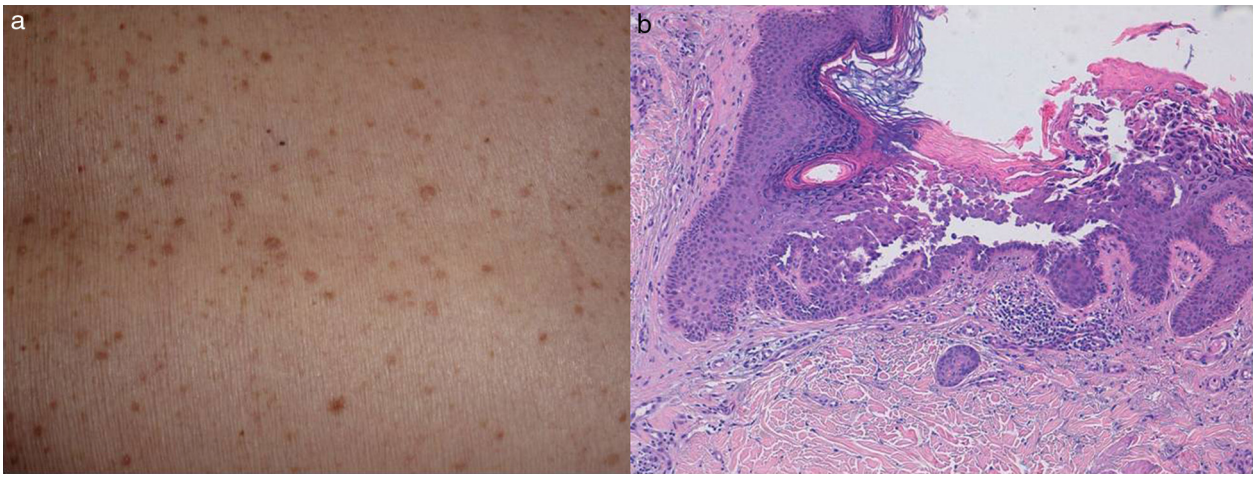

Fig. 1 – (a) Aspect of the skin lesions: non-confluent brownish keratotic papules. (b) Areas of focal acantholysis with dyskeratosis (H&E, 200×).

Discussion

GD, also known as persistent or transient acantholytic dermatosis, is a rare condition characterised by the presence of small papules and papulo-vesicles, of the same color as normal skin or erythematous and pruritic, which usually affect the back. The disease is usually transient and resolves spontaneously within weeks. In some cases, lesions may be recurrent or persist throughout years. The histological changes are characterized by focal acantholysis and dyskeratosis. Four histological patterns have been identified: Darier type, Haley–Haley type, pemphigus and spongiotic type.[1]

The aetiology of GD is unknown. Fever or prolonged bed rest, sweating or excessive heat, exposure to UV radiation, treatment with ionizing radiations, xerosis, some drugs, chronic renal failure and immunosuppression have been associated with this disease.[2]

To date, including our case, there has been twelve cases published of GD associated tochronic renal failure (Table 1).[3–10] The mean age has been 57 years and, except for two cases, all of them were men. The normal presentation was keratotic, pruritic papules located in the back. In three cases, the lesions were asymptomatic.[4,6] The head was affected in four patients[3–5,9] and in one of them it was the only location.[3] The cause of renal failure was variable. In eleven cases the lesions appeared when the patient on regular haemodialysis (7 cases) or peritoneal dialysis (3 cases) and in one case the disease manifested itself after kidney transplant. The mean time from the beginning of dialysis until the appearance of the lesions varied from months to eight and a half years. The most frequent histopathological pattern was the Darier type. Evolution was variable and there is a poor response to the treatment. In seven cases the lesions were transient and in five persisted. Among patients with transient lesions, in three cases resolved spontaneously,[4,7] in one after treatment,[9] in two cases the lesions reoccur after kidney transplant[4,8] and in one patient they resolved after changing the dialysis solution.[10]

Although the reason why GD appears in patients with chronic renal failure is unknown, it has been stated that the decrease in sweat secretion, cutaneous xerosis and the obstruction of sweat ducts may act as triggering factors. In our patient, the treatment with cyclophosphamide and methyl prednisolone previously administered in order to treat her kidney disease could also have played a role in the development of the lesions. The association of GD with other states of immunosuppression,, such as HIV infection, bone marrow transplant, and several haematological and non-haematological malignancies has been described.

With respect to treatment, in the mildest cases, an expectant attitude can be adopted since the disease usually resolves spontaneously. Sun exposure and other triggering factors such as physical exercise and heat should be avoided. If treatment is necessary, corticosteroids, calcipotriol, or calcineurin inhibitors may be used topically as the first-line of drugs. Antihistamines should be used to reduce symptoms. For refractory cases, corticosteroids, oral retinoids and phototherapy can been used.

GD should be taken into account in the differential diagnosis of the cutaneous lesions in patients with chronic renal failure, specifically in those patients on haemodialysis or peritoneal dialysis.

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Table 1 – Grover cases of chronic renal failure disease reported in literature.

	Cases	Age	Gender	Renal disease	Type of dialysis	Time since dialysis	Clinical symptoms	Location	Pruritus	Histological pattern	Progress
1	Chua and Giam,[3] 1997	53	M	Chronic GN	Haemodialysis	8.5 years	Papules	Retroarticular and neck	Y	Darier	Persistent
2	Casanova et al.,[4] 1999	39	M	Chronic GN	Haemodialysis	3 months	Keratotic papules	Face and torso	Y	Darier	Persistent
3	Casanova et al.,[4] 1999	75	M	Hypertensive nephropathy	Haemodialysis	6 months	Keratotic papules	Torso and extremities	N	Darier	Persistent
4	Casanova et al.,[4] 1999	35	M	Chronic pyelonephritis	Haemodialysis	13 months	Keratotic papules and papulovesicles	Torso	Y	Spongiotic	Resolution after transplant
5	Casanova et al.,[4] 1999	44	M	Chronic GN	Haemodialysis	2 months	Keratotic papules	Torso	N	Darier	Spontaneous resolution
6	Wong and Chua,[5] 2000	70	M	NS	Peritoneal dialysis	6 months	NS	Torso and head	Y	Darier	Persistent
7	Pastor et al.,[6] 2003	69	M	Urothelial carcinoma (nephrectomy)	Haemodialysis	2 months	Keratotic papules	Torso and extremities	N	Darier	Persistent
8	Boutli et al.,[7] 2006	58	M	Membranous GN	None (transplanted)	–	Papules	Torso	Y	Darier	Spontaneous resolution
9	González-Sixto et al.,[8] 2007	62	M	Chronic GN	Peritoneal dialysis	32 months	Papules	Torso	Y	Darier	Resolution after transplant
10	Bassi et al.,[9] 2012	43	F	Membranoproliferative GN	Haemodialysis	NS	Keratotic papules	Torso, extremities and scalp	Y	Darier	Resolution with treatment
11	Jatem et al.,[10] 2013	68	M	Polycystic kidney	Peritoneal dialysis	6 months	Maculo-papules	Torso and extremities	Y	Darier	Resolution upon substituting dialysis solution
12	Our case	69	F	Rapidly progressive GN	Haemodialysis	5 months	Keratotic papules	Torso	Y	Darier	Spontaneous resolution

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Severe arrhythmia due to hypokalemia. Influence from diuretic substances☆

Arritmia cardiaca grave por hipopotasemia. Influencia de las sustancias diuréticas

Dear Editor,

This was a case of a 25-year-old woman with no known allergies or relevant medical history and no toxic habits. She is an attorney and drinks 500–750 ml of beverages containing taurine and 1 l of caffeinated soda per day due to stress. The following details were observed: height 170 cm, weight 58 kg and BMI 20. She was admitted to the hospital for headache and tachycardia during the last two days after she did some sports and coinciding with an increase in the consumption of a beverages containing taurine. She denied chest pain or dyspnoea. Had no vomiting or diarrhoea and had no change in diuresis. She did not consume herbal products, drugs, teas, diuretics, liquorice or alcohol. *Physical examine*: Conscious, oriented, blood pressure 108/86, heart rate 110 beats per minute. Afebrile. Anodyne cardiopulmonary auscultation. Rest of the examination was normal. *Blood test*: normal red cell count, no elevation of cardiac or hepatic enzymes and coagulation test without alterations; creatinine 1.04 mg/dL, urea 31 mg/dL, potassium 1.73 mEq/L, sodium 134 mEq/L, magnesium 2.2 mg/dL, chloride 85 mEq/L, Albumin 4 g/dL. Arterial blood gas: Ph 7.580, PCO_2 46 mmHg, PO_2 86 mmHg, bicarbonate 43.1 mmol/L. Plasma anion gap (AG): 5.9 mEq/L. Urine: chloride 22.2 mEq/L, potassium 68.28 mEq/L, sodium 210 mmol/L, urea 920 mg/dL, creatinine 192.72 mg/dL, glucose 15 mg/dL. Urine anion gap: 256 mEq/L. Plasma osmolality: 278.2 mOsm/L. Urine osmolality: 573.3 mOsm/l. Transtubular potassium gradient: 15. Cortisol at 8 am and aldosterone in supine position were within the normal range. No alterations in urinary sediment. ECG: sinus rhythm, markedly enlarged QT (580 ms; corrected 700 ms); with frequent polymorphic ventricular tachycardia (Fig. 1). An infusion of CLK was initiated via central line: 80 mEq within two hours and maintained with an infusion of 120 mEq/day. After 18 h, urine test was: sodium 25.3 mEq/L, potassium 6.2 mEq/L; in serum: sodium 142 mEq/L, potassium 2.8 mEq/L transtubular potassium gradient: 4. Venous blood gas: Ph 7.380, PCO_2 52 mmHg, HCO_3 30.8. Blood test at discharge: sodium 143 mEq/L, potassium 4.84 mEq/L, chloride 105 m Eq/L, pH 7.380, pCO_2 49 mmHg, bicarbonate 29 mmol/L. Urine: potassium 11.59 mmol/L, sodium 89 mmol/L, creatinine 266.27 mg/dL, urea 642 mg/dL. The ECG was normal. The evolution of ions in the urine suggested the presence of a diuretic substance that was suspended at admission. *Diagnoses*: hypokalaemia due to diuretic substances: taurine and caffeine, but not being able to rule out the presence of other diuretics, aggravated by the increase of insensitive losses and alkalemic state. A Bartter vs Gitelman-type tubulopathy was ruled out given the evolution of the ions in urine and the hormonal axis normality. Alteration in heart conduction due to hypokalaemia. Mixed alkalaemia: Chloride-resistant metabolic alkalosis due to diuretic substances and reactive respiratory alkalosis.

Ninety percent of the potassium filtered at glomerular level is reabsorbed in the proximal tube. The distal tubule, by effect

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