episode of diabetic ketoacidosis. While hospitalised, multiple maculopapular erythematous lesions were detected with universally distributed central hyperkeratotic scaly areas (Figure 2). We consulted the Dermatology department, a skin biopsy was taken of the lesions and the patient was diagnosed with Kyrle disease.

DISCUSSION

Perforating skin disorders are characterised by the transepidermal elimination of some components of the dermis and they have classically been classified into four types: elastosis perforans serpiginosa, reactive perforating collagenosis, perforating folliculitis and Kyrle disease.

These four disorders have been reported in patients with CRF, DM or both conditions. APD lesions associated with CRF or DM are usually 2-10mm, hyperkeratotic and are often umbilicated papules generally located in the limbs, particularly the legs. The lesions are usually very pruritic, with positive Koebner phenomenon on scratching. In the cases that we reported, there was a predominance of lower limb lesions and in both cases, pruritus was the main symptom. The presence of lesions on the face, hands and feet is exceptional. Our second patient had major lesions in both feet.

APD was also reported in other cases of CRF not due to DM, including obstructive nephropathies, hypertensive nephroangiosclerosis, AIDS, etc. This suggests that the cutaneous changes typical of CRF may act as a trigger in the development of APD. Microdeposits of substances such as calcium salts may promote a local inflammatory reaction, as well as connective tissue degradation. In fact, microcrystal deposits have been observed in the upper dermis in ultrastructure studies carried out on patients with APD.

In conclusion, this pathology is relatively common in dialysis units (prevalence varies between 4% and 10%). It is not always diagnosed and it is occasionally debilitating, due to the pruritus that it causes. Its pathogenesis is unknown, although it may be influenced by the trauma caused by pruritus itself. In any case, it is a relatively unknown condition, and as such, new studies are necessary in order to better define this collagen abnormality.

Conflicts of interest

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

1. Rapini RP. Perforating disorders. In: Ardnt KA, Le Boit PE, Robinson JK, Wintroub BU, eds. Cutaneous medicine and surgery. 1st ed. Philadelphia: W.B. Saunders Company; 1996. pp. 407-11.
2. Pedragosa R, Knobel HJ, Huguet P, Oristrell J, Valdés M, Bosch JA. Reactive perforating collagenosis in Hodgkin's disease. Am J Dermatopathol 1987;9:41-4.
3. Henry JC, Jorizzo JL, Apisarthaanarax P. Reactive perforating collagenosis in the setting of prurigo nodularis. Int J Dermatol 1983;22:386-7.
4. Faver IR, Daoud MS, Su WP. Acquired reactive perforating collagenosis. Report of six cases and review of the literature. J Am Acad Dermatol 1994;30:575-80.
5. Bank DE, Cohen PR, Kohn SR. Reactive perforating collagenosis in a setting of double disaster: acquired inmunodeficiency syndrome and end stage renal disease. J Am Acad Dermatol 1989;21:371-4.
6. Zelger B, Hintner H, Auböck J, Frtsch PO. Acquired perforating dermatosis: transepidermical elimination of DNA material an possible role of lukocytes in pathogenesis. Arch Dermatol 1991;127:695-700.
7. Hood AF, Hardegen GL, Zarate AR, Nigra TP, Gelfand MC. Kyrle's disease in patients with chronic renal failure. Arch Dermatol 1982;118:85-8.
8. Morton CA, Henderson IS, Jones MC, Lowe JG. Acquired perforating dermatosis in a British dialysis population. Br J Dermatol 1996;135:671-7.
9. Kawakami T, Saito R. Acquired reactive perforating collagenosis associated with diabetes mellitus: eight cases that meet Faver's criteria. Br J Dermatol 1999;140:521-4.
10. Haftek M, Euvrard S, Kanitakis J, Delawari E, Schmitt D. Acquired perforating dermatosis of diabetes mellitus and renal failure: Further structural clues to its pathogenesis. J Cutan Pathol 1993;20:350-5.

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Treatment of infection due to hepatitis C virus in haemodialysis

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To the Editor:

Chronic hepatitis due to the hepatitis C virus (HCV) in patients with chronic renal failure (CRF) reduces survival in haemodialysis and kidney transplant and graft survival, as well as worsening liver disease, increasing the frequency of fibrosing cholestatic hepatitis and accelerating the progression to cirrhosis,[1,2] particularly in kidney transplant patients or those with hepatocellular carcinoma.[3]

We report the case of a 67-year-old female, with a dry weight of 41.5kg and a history of CRF secondary to Alport syndrome. The patient underwent deceased donor renal transplantation in May 1995 after receiving peritoneal dialysis over a one-year period. The patient initially displayed acute vascular rejection and did not respond to corticosteroids, and as such, she was treated with muromonab (OKT3),

and her creatinine levels remained stable. She subsequently presented with chronic allograft nephropathy with elevated levels of anti-HLA-2 antibodies and received immunosuppressive therapy with prednisone, mycophenolate mofetil and tacrolimus. In 2003, a liver biopsy was performed and the results were consistent with chronic periportal hepatitis due to HCV genotype 2, functional CPD8 and MEDE of 15-17. At this time, she had fluid retention with ascites and started haemodialysis due to a deterioration of her general condition, severe acidosis, resistant high blood pressure and symptomatic uraemia along with hepatic encephalopathy.

The patient initially displayed a viral load (VL) of 2,790,000 copies/ml. However, after 4 months on haemodialysis, this decreased (43,200 copies/ml). Treatment was started with 135µg/week peginterferon and 200mg/day ribavirin,[4] although due to a haemoglobin decrease to 9.3g/dl in week 10, we decreased the ribavirin dose to 200mg/48h in non-dialysis days, with a break one day a week; we also increased the darbepoetin and erythropoietin dose. In week 16, we decreased the peginterferon dose to 135µg/14 days due to a decrease in platelets to 77,000µl.

The patient had a rapid viral response (negative VL in week 4), maintaining viral suppression until the end of the 38-week treatment.

After 3 weeks of treatment with combination therapy, we decreased the darbepoetin and erythropoietin dose to 30mg/week due to haematocrit of 41.2% and intravenous iron administration was discontinued; an improvement in residual renal function was also observed (glomerular filtration rate = 15ml/min, Kt/V: 1.8), with dialysis frequency being reduced to 2 days/week. However, although there was still acceptable residual clearance and diuresis, it was necessary to increase the frequency of dialysis to 3 times/week due to high blood pressure with hyperphosphataemia and acidosis.

Twenty-four days after the end of treatment, a sustained viral response was confirmed, with an improvement in liver disease and normalisation of transaminases, and the patient was included in the dual kidney and liver transplant waiting list. However, she died a sudden death secondary to acute myocardial infarction before receiving a transplant.

DISCUSSION

Currently, treatment with peginterferon and ribavirin in patients with kidney disease is limited by creatinine clearance.[5,6] The Kidney Disease Improving Global Outcomes,[7] the American Association for the Study of Liver Diseases[8], the Japanese Society for Dialysis Therapy[9] and the Spanish Society of Nephrology[10] guidelines do not recommend treating HCV in kidney transplant patients with peginterferon because it may cause acute rejection and/or acute interstitial nephropathy, or peginterferon in combination with ribavirin due to the risk of anaemia. In light of our results, we believe that it is possible to achieve a sustained viral response with combination therapy by carrying out a strict clinical control on the patient and minimising potential adverse effects, and thus achieve retransplantation in this patient group.

Conflicts of interest

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

1. Marcelli D, Stannard D, Conte F, Held PJ, Locatelli F, Port FK. ESRD patient mortality with adjustment for comorbid conditions in Lombardy (Italy) versus the United States. Kidney Int 1996;50(3):1013-8.
2. Nakayama E, Akiba T, Marumo F, Sato C. Prognosis of anti-hepatitis C virus antibody-positive patients on regular hemodialysis therapy. J Am Soc Nephrol 2000;11(10):1896-902.
3. Van Leusen R, Adang RP, de Vries RA, Cnossen TT, Konings CJ, Schalm SW, et al. Pegylated interferon alfa-2a (40 kD) and ribavirin in haemodialysis patients with chronic hepatitis C. Nephrol Dial Transplant 2008;23(2):721-5.
4. Ashley C, Currie A. Interferon alfa-2b and Rivabirin. In: The renal drug handbook. 3 ed. United Kingdom: Radcliffe Publising; 2009. pp. 387-8, 641-42.
5. AEMPS. Ficha técnica de Pegasys®. Available at: http://www.ema.europa.eu/docs/es_ES/document_library/EPAR_-_Product_Information/human/000395/WC500039195.pdf. 6 Mayo 2013.
6. AEMPS. Ficha técnica de Rebetol®. Available at: http://www.ema.europa.eu/docs/es_ES/document_library/EPAR_-_Product_Information/human/000246/WC500048210.pdf. 6 Mayo 2013.
7. KDIGO. Guideline 4: Management of HCV-infected patients before and after kidney transplantation. [serie en internet: Kidney International (2008) 73 (Suppl 109)]. 2008. [cited Mayo 5 2013]. Available at: http://www.kdigo.org/pdf/KI%20Hep%20C%20GL%20Apr%202008.pdf.
8. Ghany MG, Strader DB, Thomas DL, Seeff LB; American Association for the Study of Liver Diseases. Diagnosis, management, and treatment of hepatitis C: an update. Hepatology 2009;49(4):1335-74.
9. Akiba T, Hora K, Imawari M, Sato C, Tanaka E, Izumi N, et al. 2011 Japanese Society for Dialysis Therapy guidelines for the treatment of hepatitis C virus infection in dialysis patients. Ther Apher Dial 2012;16(4):289-310.
10. Sociedad Española de Nefrología. Guías sobre enfermedades víricas en hemodiálisis (HD). 2003. [cited Mayo 6 2013]. Available at: http://www.senefro.org/modules/webstructure/files/guiasvirusb.pdf?check_idfile=816

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