

- <http://dx.doi.org/10.1177/0004563220922255>. Epub 2020 May 3. PMID: 32266828.
- Spasovski G, Vanholder R, Allolio B, Annane D, Ball S, Bichet D, et al. Hyponatraemia guideline development group: clinical practice guideline on diagnosis and treatment of hyponatraemia. *Nephrol Dial Transplant*. 2014;29 Suppl 2:i1-39.
 - Reynolds RM, Seckl JR. Hyponatraemia for the clinical endocrinologist. *Clin Endocrinol (Oxf)*. 2005;63:366-74.
 - Arlt W, Allolio B. Adrenal insufficiency. *Lancet*. 2003;361:1881-93.
 - Yatagai T, Kusaka I, Nakamura T, Nagasaka S, Honda K, Ishibashi S, et al. Close association of severe hyponatremia with exaggerated release of arginine vasopressin in elderly subjects with secondary adrenal insufficiency. *Eur J Endocrinol*. 2003;148: 221-6.
 - Ishikawa SE, Scriver RW. Effect of arginine vasopressin antagonist on water excretion in adjuvant glucocorticoid and mineralocorticoid-deficient rats. *Kidney Int*. 1982;22:587-93.
 - Saito T, Ishikawa SE, Ando F, Okada N, Nakamura T, Kusaka I, et al. Exaggerated urinary excretion of aquaporin-2 in the pathological state of impaired water excretion dependent upon arginine vasopressin. *J Clin Endocrinol Metab*. 1998;83:4034-40.

José Carlos De La Flor Merino^{a,*}, Laura Mola Reyes^b, Tania Linares Gravalos^a, Ana Roel Conde^c, Miguel Rodeles del Pozo^a

^a Servicio de Nefrología, Hospital Central de la Defensa Gómez Ulla, Madrid, Spain

^b Servicio de Endocrinología, Hospital Central de la Defensa Gómez Ulla, Madrid, Spain

^c Servicio de Medicina Interna, Hospital Central de la Defensa Gómez Ulla, Madrid, Spain

* Corresponding author.

E-mail addresses: josedelaflor81@yahoo.com, jflomer@mde.es (J.C. De La Flor Merino).

2013-2514/© 2020 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

<https://doi.org/10.1016/j.nefro.2020.06.015>

Letter to the Editor

Crateriform plaques in a patient with end-stage renal disease. The case of an acquired reactive perforating collagenosis[☆]

Placas crateriformes en un paciente con enfermedad renal crónica. Un caso de colagenosis perforante reactiva adquirida

Dear Editor,

The skin conditions which can occur in chronic kidney disease patients are: xerosis, pruritus, calciphylaxis, pseudoporphyria, uraemic frost and acquired perforating skin disorders; the latter are characterised by presenting transepidermal elimination of material from the dermis, and consist of Kyrle disease, reactive perforating collagenosis and elastosis perforans serpiginosa.¹

We present the case of a 78-year-old male with a history of type 2 diabetes mellitus, systemic hypertension, long-term hypothyroidism and chronic kidney disease on replacement therapy with peritoneal dialysis for the past five years, with adequate control. The patient was hospitalised due to healthcare-associated pneumonia. We were consulted due to a disseminated, bilateral and symmetrical dermatosis which affected the lateral side of both arms and the anterior side of the knees, made up of multiple plaques measuring 0.5-1 cm in diameter which were circular, erythematous, had a crateri-

form appearance with a haematic scab on their surface, were itchy and had first appeared six weeks previously (Fig. 1).

A biopsy of one of the lesions was performed which showed in the haematoxylin and eosin stain: epidermis with moderate acanthosis which surrounded a plug made up of the scab, keratinous detritus and inflammatory cells; in the base of this plug, flattened epidermis with introduction of collagen fibres running vertically from the dermis could be seen; with Masson's trichrome stain, the presence of transepidermal elimination of collagen fibres was confirmed (Fig. 2). With these findings, the diagnosis of acquired reactive perforating collagenosis (ARPC) was made. The patient was treated with fluocinolone acetonide cream 0.01% every 12 h plus levocetirizine tablets 2.5 mg/day, with an adequate response in treatment weeks, an interval during which his symptoms of pneumonia resolved and he was discharged. The patient remained under follow-up by our department.

DOI of original article:
<https://doi.org/10.1016/j.nefro.2019.04.005>.

[☆] Please cite this article as: Villela-Segura U, Miranda-Aguirre AI, Estrada-Aguilar L. Placas crateriformes en un paciente con enfermedad renal crónica. Un caso de colagenosis perforante reactiva adquirida. *Nefrología*. 2020;40:358-360.

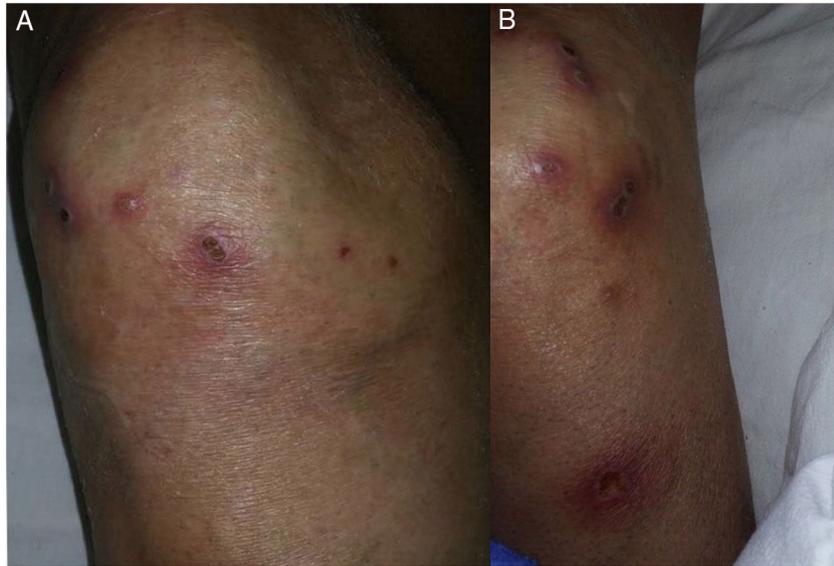


Fig. 1 - (A and B) Multiple plaques of crateriform appearance with keratosis and haematic scabs on their surface.

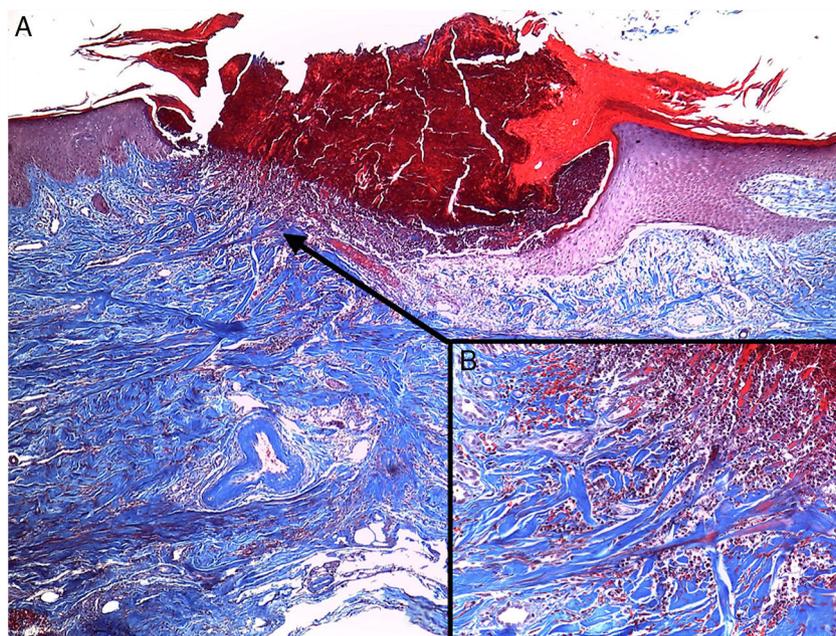


Fig. 2 - Masson's trichrome stain 40 \times . (A) Epidermal acanthosis surrounding a plug composed of cellular detritus, keratin and inflammatory cells. (B) Presence of collagen fibres which are introduced from the dermis towards the epidermis.

ARPC is an entity commonly associated with patients with diabetes mellitus and chronic kidney disease on replacement therapy; however, it can also be observed in Hodgkin's lymphoma, scabies, atopic dermatitis, tuberculosis, systemic lupus erythematosus, AIDS, systemic hypertension and chronic venous insufficiency; it mainly affects patients between the third and fifth decades of life. This entity is more common than its hereditary counterpart, which presents in the paediatric population and has few reported cases in the medical literature.²

Its pathophysiology includes the presence of receptors for advanced glycation end products (RAGE), which cause the cross-linking of collagen fibres and an increase in TGF- β 3 and metalloproteinase-1, which cause changes in the metabolism of extracellular proteins and delayed epithelialisation, com-

bined with the presence of occasional microtrauma and chronic pruritus, which perpetuate the lesions.³

The lesions generally present on the trunk and extensor areas of the limbs; they are made up of multiple plaques of various centimetres in diameter, are circular, erythematous, of crateriform appearance, with haematic scabs or keratosis on their surface, are itchy and, due to continuous trauma, are associated with the Koebner phenomenon. The histological findings show acanthosis which surrounds a plug composed of basophilic material that contains keratin, cellular detritus and inflammatory cells; below this a flattened epidermis with the presence of collagen fibres which are introduced from the dermis is observed; they are viewed with greater accuracy with the Masson's trichrome stain.⁴ To make the diagnosis, it is necessary for the patient to be over the age of 18, mani-

fest characteristic features of dermatosis and for there to be evidence of transepidermal elimination of collagen.⁵ Calciophylaxis, another entity that can be found in this group of patients, is clinically different due to the presence of pain that precedes the appearance of livid or violet plaques, which evolve to ulceration and necrosis, and can be found in central areas (abdomen and thighs) and peripheral areas (fingers and even the penis). The biopsy reveals calcification of the arterioles and the small-medium sized arteries, fibroblastic proliferation of the intimal layer and narrowing of the lumen.⁶

The cornerstone of treatment is to avoid trauma; this measure leads to reduction of new lesions, which are generally self-limiting in six to eight weeks; due to the substrate of chronic pruritus, the use of antihistamines is recommended. Due to the low frequency of this disease, the use of topical and oral retinoids, topical corticosteroids, emollients and narrow-band UVB radiation have been tried in case reports with moderate response⁷; allopurinol has been used in a series of 12 patients at doses of 100–300 mg/day, under the premise of reducing cross-linking of collagen fibres by inhibiting xanthine oxidase, with the consequent reduction in the production of free radicals and RAGE, with improvement in seven patients after four weeks and the rest with improvement in the subsequent four months.⁸

The importance of presenting this case lies in showing another variety of dermatosis that may occur in patients with chronic kidney disease, to alert the treating physician to its presence, and to carry out the appropriate diagnosis and treatment due to the impact that it has on the quality of life of this patient population.

REFERENCES

1. Fernandes KA, Lima LA, Guedes JC, Lima RB, Dácri AM, Martins CJ. Acquired perforating dermatosis in a patient with chronic renal failure. *An Bras Dermatol*. 2016;91:10–3, <http://dx.doi.org/10.1590/abd1806-4841.20164619>.

2. García AJ, del Valle E, Sánchez MP, del Prado E, Coscojuela C, Gilaberte Y. Acquired perforating dermatosis: clinicopathological study of 31 cases, emphasizing pathogenesis and treatment. *J Eur Acad Dermatol Venereol*. 2017;31:1757–63, <http://dx.doi.org/10.1111/jdv.14220>.
3. Fei C, Wang Y, Gong Y, Xu H, Yu Q, Shi Y. Acquired reactive perforating collagenosis: a report of a typical case. *Medicine (Baltimore)*. 2016;95:e4305, <http://dx.doi.org/10.1097/MD.0000000000004305>.
4. Kim SW, Kim MS, Lee JH, Son SJ, Park KY, Li K, et al. A clinicopathologic study of thirty cases of acquired perforating dermatosis in Korea. *Ann Dermatol*. 2014;26:162–71, <http://dx.doi.org/10.5021/ad.2014.26.2.162>.
5. Wagner G, Sachse MM. Acquired reactive perforating dermatosis. *J Dtsch Dermatol Ges*. 2013;11:723–9, <http://dx.doi.org/10.1111/ddg.12131>.
6. Villela-Segura U, Peralta-Serna J, Guerrero-Álvarez A, Estrada-Aguilar L. Glans penis necrosis caused by calcific uremic arteriolopathy. *Dermatol Online J*. 2019;25, pii:13030/qt2kg3n28d.
7. Reid J, Almond L, Matthewman N, Stringer H, Francis N, al Abadie M. A case of acquired reactive perforating collagenosis. *Aust J Dermatol*. 2018;59:75–6, <http://dx.doi.org/10.1111/ajd.12618>.
8. Titz H, Becker JC, Legat F, Schettini AP, Inzinger M, Massone C. Allopurinol in the treatment of acquired reactive perforating collagenosis. *An Bras Dermatol*. 2013;88:94–7, <http://dx.doi.org/10.1590/S0365-05962013000100012>.

Uriel Villela-Segura *, Alessandra Irais Miranda-Aguirre, Lorena Estrada-Aguilar

Servicio de Dermatología, Hospital Regional Licenciado Adolfo López Mateos, ISSSTE, Ciudad de México, Mexico

* Corresponding author.

E-mail address: uvise08@gmail.com (U. Villela-Segura).

2013-2514/© 2019 Sociedad Española de Nefrología. Published by Elsevier España, S.L.U. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>). <https://doi.org/10.1016/j.nefro.2020.06.003>

Letter to the Editor

Relapsing polychondritis and focal segmental glomerulosclerosis: Coincidence or causality[☆]

Policondritis recidivante y glomeruloesclerosis segmentaria y focal: coincidencia o causalidad

Dear Editor,

Relapsing polychondritis (RP) is a rare autoimmune condition of unknown aetiology which is characterised by recurrent

inflammatory flare-ups of cartilage structures mainly affecting the nasal, auricular and laryngotracheal cartilage. Renal involvement is unusual and, when it does present, it is associated with poorer survival.¹ Below we describe a case of a patient with RP and associated glomerular disease.

DOI of original article:

<https://doi.org/10.1016/j.nefro.2019.04.003>.

[☆] Please cite this article as: Canllavi E, Alonso M, Fernández M, Gutiérreza E, Morales E. Policondritis recidivante y glomeruloesclerosis segmentaria y focal: coincidencia o causalidad. *Nefrologia*. 2020;40:360–362.