CASE REPORT

A 76-year-old female with history of end-stage renal disease due to IgA nephropathy, submitted to a kidney transplant 28 years before, secondary hyperparathyroidism under vitamin D analogue, chronic anaemia, diabetes mellitus, arterial hypertension, atrial fibrillation (anticoagulated with warfarin) and peripheral venous insufficiency presented with a 1-month history of a skin ulcer on the left lower limb. Five months before, she had to go back on haemodialysis by central venous catheter because of renal allograft failure by chronic dysfunction. Her residual urine output was 1L/day and she was still on low-dose prednisone. Physical examination revealed a distal, 4-centimetre, skin ulcer on the left leg, with inflammatory signs and purulent discharge. Distal pulses were absent, but ultrasound image showed no alterations in biphase flux of both anterior and posterior tibial vessels. The patient was started on amoxicillin-clavulanic acid, but the ulcer worsened: it became bigger and more painful. One week later, she was readmitted to the emergency department hypotensive, prostrated and with thoracic pain. Acute coronary syndrome was excluded. Laboratory tests showed worsened anaemia (hemoglobin 7g/dL, previously 10g/dL), leucocytosis (16080 leucocytes/microliter, normal value 4000-11000 leucocytes/microliter), high C-reactive protein (87mg/L, normal value <5mg/L), high parathyroid hormone (246pg/mL, normal value 10-65pg/mL), hyperphosphatemia (5.4mg/dL, normal value 2.7-4.5mg/dL) with normal calcium (2 mmol/L, normal value 2-2.6 mmol/L). She was afebrile. Blood cultures were requested. Multi-resistant Staphylococcus epidermidis was isolated in blood cultures; vancomycin was started and the central venous catheter switched. Infective endocarditis was excluded. Nevertheless, the ulcer grew bigger and evolved to necrosis (image 1). A leg radiography and a skin biopsy were performed. Leg radiography showed calcified vessels surrounding non-calcium-containing radiolucent tissues (image 2). Skin biopsy revealed extensive necrosis of epidermis, dermis and subcutaneous tissue; areas of recent thrombosis of the hypodermic vessels and calciphilia in vessels (amplification 400x, coloration eosin-hematoxylin). The patient died of septic shock before the result of the skin biopsy.

Calciphylaxis or calcific uremic arteriolopathy is a rare, life-threatening condition, that is usually fatal in hemodialysis patients.
associated to end-stage renal disease. Less frequently, it can also occur in renal transplant recipients and other patients, denominated nonuremic calciphylaxis. Most patients present with painful lesions that can progress to ischemic/necrotic ulcers and become superinfected. Vascular calcifications can occur in other localizations\(^1\). Histology reveals dermal and pannicular arteriolar calcification, subintimal fibrosis and thrombotic occlusion. Risk factors include hyperphosphatemia, warfarin use, calcium-based binders, vitamin D analogues, systemic glucocorticoids, female sex, hypoalbuminemia and diabetes mellitus.\(^2\) Optimal treatment is unknown, but sodium thiosulfate, wound care and pain control are reasonable options. Abnormalities in blood calcium and phosphate should be treated\(^3\). Delayed diagnosis can lead to sepsis-related morbidity and mortality, as in this case we report\(^1\).

REFERENCES

Palabras clave: calcifilaxis, trasplante de riñón, úlcera cutánea.
Keywords: calciphylaxis, kidney transplant, skin ulcer.