

Case study

Calciophylaxis in hemodialysis, diagnosis and management : case report

ABSTRACT:

A 59-year-old woman followed for chronic end-stage renal failure on hemodialysis for diabetic nephropathy. She had presented large, very painful, ulcerated lesions on the inner surfaces of the knees, shallow, with no signs of secondary bacterial infection. Biology revealed hypercalcemia at 2.74 mmol/l normal phosphatemia at 0.55 mmol/l. Thigh X-rays revealed vascular calcifications of the superficial femoral and popliteal arteries, and of the leg arteries bilaterally. Histology had objectified dermal neo-angiogenesis with stasis thrombi and secondary epidermal necrosis, testifying to an underlying thromboembolic phenomenon. Treatment consisted of analgesic treatment and daily local care. Intensification of dialysis sessions associated with sodium thiosulphate, as well as treatment by rheopheresis. In addition, the striking event was the accidental occurrence of a self-limiting gas embolism after a hyperbaric oxygen therapy session playing an important role in the regression of skin necrosis. Thus the evolution was favorable with healing of skin lesions and regression of pain.

KEYWORDS : Calciophylaxis; End-stage renal disease, hemodialysis

INTRODUCTION :

Calciophylaxis is a rare and severe pathology that affects patients with end-stage chronic renal failure. It is a phenomenon of ischemic, cutaneous and sometimes systemic necrosis due to the obliteration of the arterioles first by subintimal calcium deposits, then by thrombosis [1] Evolving in a context of primary hyperparathyroidism, under anticoagulant treatment or neoplasia. We report a case of uraemic calciophylaxis in the context of terminal renal failure.

CASE REPORT

A 59-year-old woman, followed for end-stage renal failure on hemodialysis on diabetic nephropathy since September 2021, having as main ATCD an ischemic stroke complicating emboligenic heart disease under vitamin K antagonists, hypertension, DNID and morbid obesity (BMI=46.8) had presented since July 2022 with large, very painful, ulcerated, livedoid, fibrinous lesions with induration and deep secondary necrosis on the lateral surfaces of the two thighs, without bone contact, involvement of the internal surfaces of the knees, shallow, without signs of bacterial superinfection (figure 1).

Biology revealed hypercalcemia at 2.74 mmol/l, normal phosphatemia at 0.55 mmol/l and hyperparathyroidism at 11 times normal. The thigh X-ray showed vascular calcifications of the superficial femoral and popliteal arteries, and of the leg arteries at the edge of the field bilaterally without any notable abnormality in the bone structure (figure 1). Histology found no calciphylaxis lesions on 2 skin biopsies one month apart, the first objectified dermal neo-angiogenesis with stasis thrombi and secondary epidermal necrosis, testifying to an underlying thromboembolic phenomenon. Also found in the second biopsy with the presence of area of hypodermic ischemic necrosis in favor of a process of vascular occlusion and fibrino-cruoric thrombi of small hypodermic capillaries whose primary or secondary nature is difficult to determine. Even in the absence of images of calciphylaxis on the two histologies, the clinical aspect is compatible with calciphylaxis.

The initial management was local care with hydrocellular dressings and Algosteril and treatment with sodium thiosulfate 25 mg 3 times a week, the evolution after one month was unfavorable in terms of pain without improvement in the skin.

The patient was then hospitalized in the dermatology department by undergoing dialysis in the dialysis department at the Henri Mondor hospital in Créteil. Secondary management consisted of analgesic treatment and daily local care by cleansing with saline solution, mechanical debridement of fibrin and wicking with Algosteril and secondary hydrocellular dressing. Intensification of dialysis sessions from 3 to 6 times per week with also sodium thiosulfate 25 mg three times per week IV, as well as treatment by rheopheresis twice a week for the first 2 weeks then once a week. week as maintenance phase with a total of 8 sessions. We also stopped VKAs as a factor favoring calciphylaxis and given the increased risk of

In addition, the patient had presented a resolving gas embolism after a session of hyperbaric oxygen therapy which also allowed a partial regression of the cutaneous necroses.

The evolution was favorable with healing of skin lesions and regression of pain.

DISCUSSION :

Calciphylaxis is a rare and severe pathology that affects patients with end-stage chronic renal failure. It is a pathology of the microvessels of the dermis and the hypodermis which are calcified and whose thrombosis leads to skin necrosis. Calciphylaxis lesions can be distal and axial. They cause intense pain, infections and are associated with a high mortality rate (40 to 80% at 1 year) [2]. The physiopathology is based on the mineral and bone abnormalities of CKD [3] then the major role of vitamin K antagonists (VKA) in the formation of arteriolar microcalcifications of Calciphylaxis [4,5]. Its prognosis remains poor with a mortality at 1 year ranging from 40 to 80% [6].

The clinical picture is characterized by hyperalgesic and indurated lesions. At first sluggish in appearance with a livedoid periphery, like a geographical map, they evolve towards necrosis. The development of vascular microcalcifications is secondary to an imbalance between the inducing and neutralizing factors of calcification associated with a change in the phenotype of smooth muscle cells [7].

Correction of risk factors, optimization of dialysis, wound care and pain management should be routine for all patients with Calciphylaxis. Treatment with sodium thiosulfate which is an antioxidant agent would act by chelating calcium and attenuating prothrombotic endothelial dysfunction induced by oxidative stress.

A beneficial effect of hyperbaric oxygen therapy in this indication has been reported in small retrospective series [8,9].

CONCLUSION :

Calciphylaxis remains a rare but extremely serious complication of chronic end-stage renal failure requiring multidisciplinary management. Early diagnosis allows rapid and appropriate treatment. The beneficial effect of hyperbaric oxygenation can be discussed as a systematic treatment for calciphylaxis

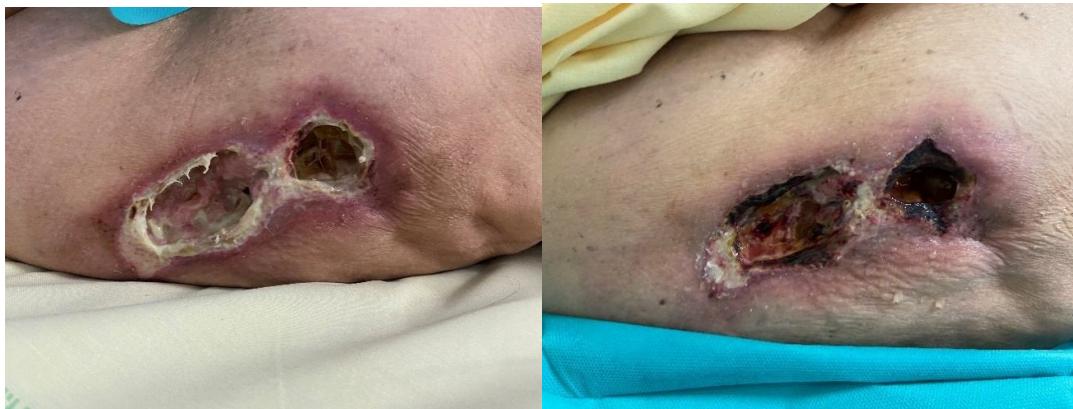


Figure 1: skin lesions of calciphylaxis before treatment

REFERENCES :

1. Duval A, Moranne O, Vanhille P, Hachulla E, Delaporte E. Calcium arteriopathy (calciphylaxis). *The Journal of Internal Medicine*. 2006; 27(3):184-195.
2. A. Zabornyk et al. A novel anti-crystallization agent inhibits induced calcification in an in vitro model of human aortic valve calcification *Vascul Pharmacol* 2019
3. C. Baldwin et al. Multi-interventional management of calciphylaxis: about 7 cases *Suis J Kidney Dis* 2011
4. CL Ellis et al. Questionable specificity of histological findings in uremic calcifying arteriopathy *Kidney Int* 2018
5. NX Chen et al. Adipocyte-induced arterial calcification is prevented with sodium thiosulfate *Biochem Biophys Res Commun* 2014
6. CK Gabel et al. Assessment of outcomes of calciphylaxis *J Am Acad Dermatol* 2021
7. C. Cassius et al. Calciphylaxis in haemodialysed patients: diagnostic value of calcifications in cutaneous biopsy *Br J Dermatol* 2018
8. Basile C, Montanaro A, Masi M, Pati G, De Maio P, Gismondi A. Hyperbaric oxygen therapy for calcific uremic arteriopathy: a case series. *J Nephrol*. 2002
9. Vassa N, Twardowski ZJ, Campbell J. Hyperbaric oxygen therapy in calciphylaxis-induced skin necrosis in a peritoneal dialysis patient. *Am J Kidney Dis*. 1994