DOI: http://dx.doi.org/10.3315/jdcr.2015.1219

Journal of Dermatological Case Reports

PHOTOLETTER TO THE EDITOR

Calcinosis cutis in a burn scar

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Abstract

Calcinosis cutis is a rare condition characterized by the deposition of insoluble calcium salts in the skin and subcutaneous tissue. Dystrophic calcinosis cutis appears as a result of local tissue damage or abnormalities, such as alterations in extracellular matrix proteins or subcutaneous tissue with normal calcium and phosphate serum levels. It has been rarely described as a late complication of burns. Latency periods of 15-54 years have been reported. We describe the case of a 57-year-old man with dystrophic calcinosis cutis in a burn scar, which developed 42 years after the skin injury. The condition was successfully treated with surgical excision. (*J Dermatol Case Rep*. 2015; 9(4): 120-121)

Key words:
calcification, histopathology, surgery, ulcer

Calcinosis cutis is a rare syndrome characterized by the deposition of insoluble calcium salts in the skin and subcutaneous tissue. It can be classified as: dystrophic, metastatic, iatrogenic and idiopathic.[1] Dystrophic calcinosis cutis (DCC) is the most common type and is characterized by the abnormal deposition of calcium salts in degenerated tissues with serum calcium and phosphate levels within normal range. DCC occurs in association with connective tissue diseases, cutaneous neoplasms, infections, trauma and inherited disorders, such as pseudoxanthoma elasticum.[2] It has been rarely reported as a late complication of burn scars. A 57-year-old man presented for evaluation of a 6-month-old non-healing ulcer on his right leg. His medical history was only remarkable for a previous thermal burn on the same site that spontaneously healed with scaring at the age of 15. We was no under any medication. On physical examination within a burn scar a 3 X 1 cm well-defined ulcer with erythematous indurated borders filled with a hard yellowish-brown material was observed (Fig. 1). Some hard material was removed from the ulcers bed and a skin biopsy was performed. Histopathology revealed the particles to be calcium and showed the presence of amorphous dystrophic calcium deposits in the subcutaneous tissue (Fig. 2). The analytical study showed no abnormalities with serum calcium and phosphate levels within normal range. The diagnosis of DCC was made. Six months after surgical excision of the lesion there was no evidence of recurrence.

A review of the literature shows that DCC is a rare event as a late complication of burn scars. Latency periods of 15-54 years have been reported.[2,3] In our case, a latency period of 42 years was observed. DCC with squamous cell carcinoma in a postburn scar has also been documented.[4] The pathophysiology of the disorder is still not clear. High levels of intracellular calcium resulting from damaged cell membranes may form hydroxyapatite crystals and the precipitation of calcium phosphate caused by the increased alkalinity of the necrotic tissue are suggested hypothesis.[1] There isn't no standard treatment for DCC. Due to its rarity, the therapeutic approach is only based in single case reports or small case series. Treatment modalities for calcinosis cutis include: warfarin, bisphosphonates, minocycline, ceftriaxone, diltiazem, hydroxide aluminium, intralesional corticosteroids, probenecid, immunoglobulin, surgical excision, carbon dioxide laser or extracorporeal shock wave lithotripsy.[5] In the postburn DCC, the treatments were surgical, sometimes associated with local injection of triamcinolone.[2,3] Recurrence

of the disease is frequently observed and may be justified by the calcification of the subcutaneous tissue not adjacent to the ulceration. Therefore, surgical excision with skin grafting may be required to prevent the reappearing of the lesions.[2] Physicians should be aware of such complication on burns. In addition, as malignant transformation of the chronic ulcerative lesions may occur it must be ruled out.

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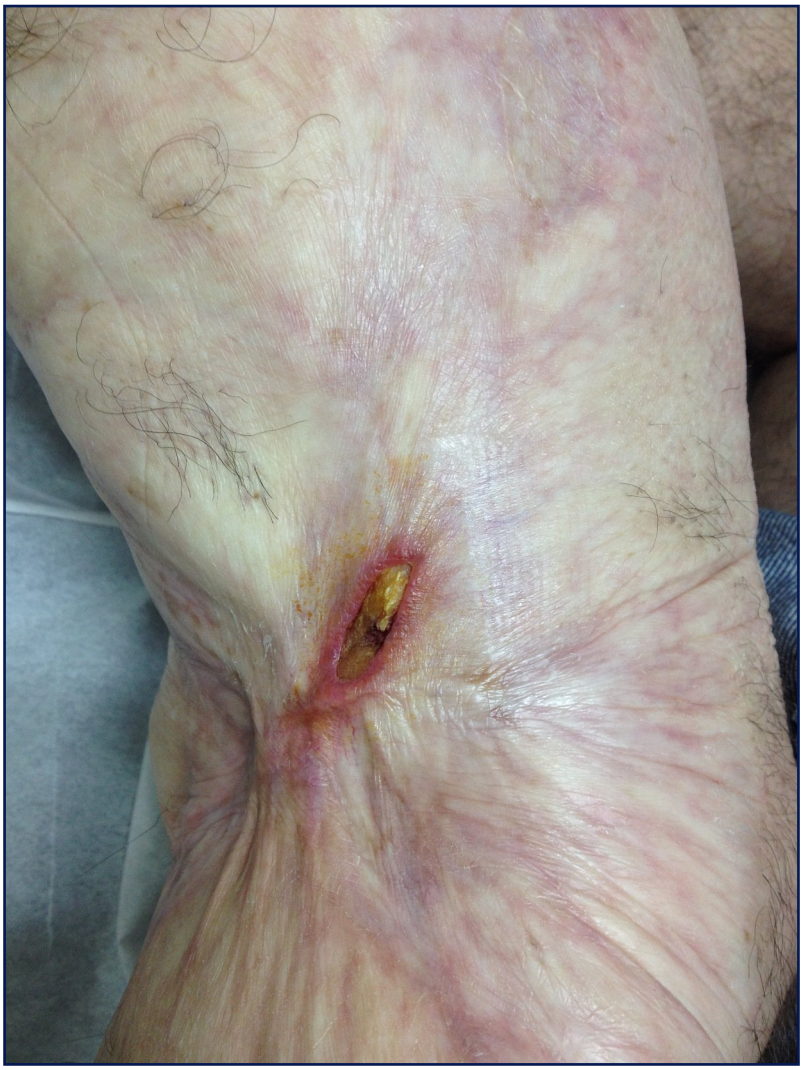

Figure 1

Well-defined ulcer with erythematous indurated borders filled with a hard yellowish-brown material on the burn scar.

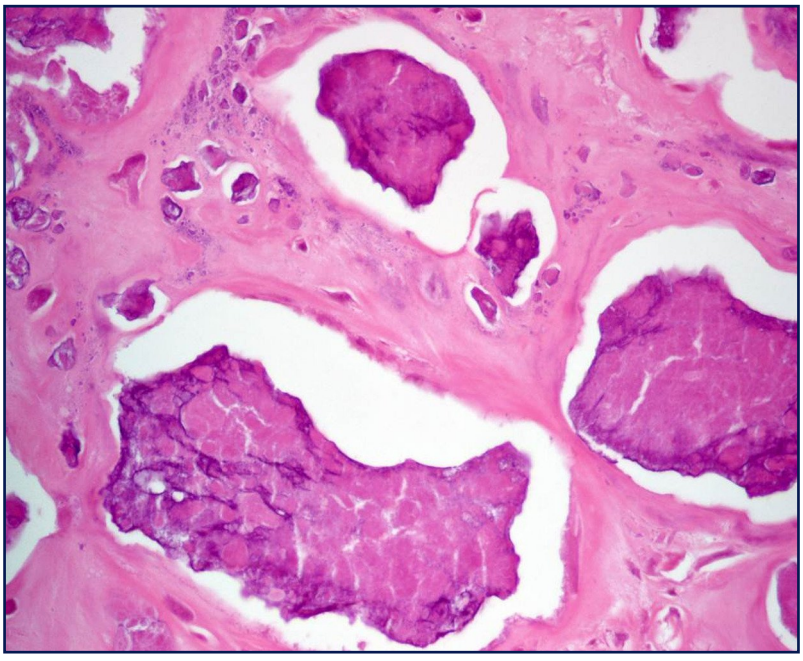

Figure 2

Amorphous dystrophic calcium deposits on the subcutaneous tissue. (Hematoxylin-eosin staining, X10).