

## Images

### Calcinosis cutis

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This is a case of 55 years old lady, diagnosed case of diffuse cutaneous systemic sclerosis 5-6 years back. She had a history of systemic hypertension for last 2 years. She referred to the cardiology department for the evaluation of new onset of dyspnea on exertion NYHA Class II. Physical examination was significant for the presence of multiple large conglomerate masses of calcinosis cutis over the fingers, with yellowish-white calcium deposition on an erythematous base (Figure 1a and b). Her 12-lead electrocardiogram was within normal limit. 2D echocardiography with color Doppler imaging study revealed no significant cardiac abnormalities. Hence, a significant cardiac illness was excluded. We are reporting the image of such a large conglomerate mass of calcinosis cutis over the finger, with yellowish-white calcium deposition on an erythematous base, which is not seen commonly in clinical practice.

Calcinosis cutis is characterized by deposition of calcium in the skin. According to their origin, calcification of soft tissues can be classified into different variants: Dystrophic, idiopathic, metastatic and iatrogenic. Dystrophic calcinosis

is calcification associated with infection, inflammatory processes, cutaneous neoplasm or connective tissue diseases. Idiopathic calcinosis cutis is cutaneous calcification of unknown cause with normal serum calcium. Subepidermal calcified nodule and tumoral calcinosis are idiopathic forms of calcification. Metastatic calcification results from elevated serum levels of calcium or phosphorus. Iatrogenic and traumatic calcinosis are those types, which are associated with medical procedures.<sup>1,2</sup>

A classic example of dystrophic type of calcinosis cutis is systemic sclerosis. Calcinosis cutis is more common in patients with limited cutaneous systemic sclerosis who are positive for anti-centromere antibodies in comparison to diffuse cutaneous systemic sclerosis. It usually involve finger pads, palms, extensor surfaces of the forearms, and the olecranon and prepatellar bursae. Its size can be varying from tiny punctate lesions to a large conglomerate masses. It is composed of calcium hydroxyapatite crystals deposited in soft tissues, without causing direct joint involvement. Main pathophysiological mechanism for the development of calcinosis in scleroderma is tissue hypoxia. This decrease in perfusion is accompanied by inflammatory cell activity and macrophage activation as well as an imbalance between various mediators that cause increased calcium influx to cells.<sup>3</sup>

Calcinosis cutis may a manifestation of different underlying pathophysiology. Therefore, after clinical diagnosis of calcinosis cutis, a laboratory workup to rule out abnormalities of calcium and phosphorus metabolism, malignant processes, collagen vascular diseases, renal insufficiency, excessive milk ingestion, vitamin D poisoning must be carried out to detect the underlying cause of the disease.

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**Figure 1:** (a) Dystrophic type calcinosis cutis involving the ring and index fingers in a case of diffuse cutaneous systemic sclerosis. (b) Large conglomerate mass of calcinosis cutis with yellowish-white calcium deposition (see arrow) on an erythematous base

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