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Case Report

Calcinosis Cutis of Left Great Toe- A Case Report

Choudhari Nidhi[1], Meghashree Vishwantha[1*], Subramanian Vidhya[1], Ramamurthy Vedhachalam[1]

Affiliation

[1] Department of Pathology, A.C.S. Medical College & Hospitals, Chennai, India.

***Corresponding Author**
Meghashree V, Assistant Professor, Department of Pathology, A.C.S. Medical College & Hospitals, Chennai, India.
E-mail: meghavishwanth@gmail.com

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Abstract

Calcinosis cutis, a rare benign disorder, entails systemic calcium deposition in soft tissues, often associated with autoimmune and renal disorders. Its pathophysiology varies across five main types: dystrophic, metastatic, idiopathic, iatrogenic, and calciphylaxis. Dystrophic calcification, the most prevalent type, correlates with normal calcium and phosphorus levels alongside autoimmune diseases. Conversely, metastatic calcification arises from abnormal serum calcium and phosphorus levels. Idiopathic calcification manifests without underlying tissue damage or abnormal laboratory values. Iatrogenic calcification is triggered by substances containing calcium or phosphate. Calciphylaxis involves vessel calcification and is linked to chronic renal failure and dialysis. A 19-year-old female presented with pain and swelling on the plantar aspect of her left great toe, diagnosed as unilateral idiopathic calcinosis cutis. A comprehensive diagnostic approach, including histopathological, radiological, and blood investigations, is crucial for effective management. The study aimed to spotlight idiopathic cutaneous calcinosis, emphasizing its accurate diagnosis through clinical, pathological, and metabolic correlation. Surgical excision offers a complete cure for this rare condition, leading to an excellent prognosis.

Keywords: Calcinosis cutis, Toe, Pain, swelling, Calcification

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Introduction

Calcinosis cutis was first described in 1855 by Virchow. [1] It was characterized histologically by deposition of calcium within the dermis. [2] It can involve any part of the skin and is classified into five main types: dystrophic, metastatic, idiopathic, iatrogenic, and calciphylaxis. [3] Identification of the type of calcinosis cutis is crucial for accurate management. Calcinosis cutis can indeed mimic other skin conditions, and a careful evaluation was necessary for accurate diagnosis. Conditions such as calcified epidermoid cysts, calcified nodules, and tumoral calcinosis may present with similar clinical features.

A comprehensive and systematic approach to diagnosis, including a consideration of differential diagnoses and appropriate diagnostic investigations, is essential for managing calcinosis cutis effectively. [4] Additionally, preventive measures, especially in cases of iatrogenic calcinosis, can play a role in minimizing the risk of calcium deposition. Limited cases of idiopathic calcinosis have been reported. [5] The aim of the study was to present the rare entity of Idiopathic cutaneous

calcinosis which can be diagnosed accurately with clinical, pathological and metabolic correlation, is completely curable by surgical excision and has an excellent prognosis.

Case Report

A 19-year-old female presented at the surgical outpatient department (OPD) with complaints of hardness and pain in her left toe. The lesion appeared diffuse and had been excised three times previously. She presented again with complaints of pain upon applying pressure. On examination, a firm to hard, immobile swelling measuring 2x2 cm was noted over the plantar aspect of her left great toe (Figure 1).

Investigations revealed normal complete hemogram, blood sugar, liver function and kidney function tests. Serum calcium and phosphate done on two different occasions were within normal limits. Serum uric acid, electrolytes and alkaline phosphatase were normal. 24-hour urinary calcium and phosphate excretions were normal serum calcium, phosphorus, uric acid, erythrocyte sedimentation rate, C-reactive protein, alkaline phosphatase, and creatine kinase were all normal. Low vitamin D levels were noted (26.42 ng/mL). Hormonal testing, encompassing calcitonin, parathyroid hormone, and thyroid hormone levels, also fell within the normal range. To exclude connective tissue disorders, extensive immunological investigations were performed, including complement C3 and C4, ANA, anti-Ds DNA, RNP Ab, SS-A Ab, SS-B Ab, and anti-Sm Ab. All values were normal.

X-rays of the foot showed no joint involvement (Figure 2). There was no history of an inciting traumatic event. Medical history, family history, and social history were all negative. There had been no increase in the number of lesions since their first appearance during the examination. The patient did not exhibit any systemic symptoms. No other abnormalities or deformities were observed. A skin biopsy revealed the deposition of homogeneous basophilic material in the dermis, suggestive of calcium deposition (Figure 3A and 3B).

Discussion

Dystrophic calcification is the most common cause of calcinosis cutis and is associated with normal calcium and phosphorus levels [5]. This condition is characterized by tissue damage that leads to the release of phosphate-binding proteins by dying cells. The phosphate-binding protein binds phosphate and results in calcification. This tissue damage also results in chronic inflammation and vascular hypoxia. Dystrophic calcification is associated with

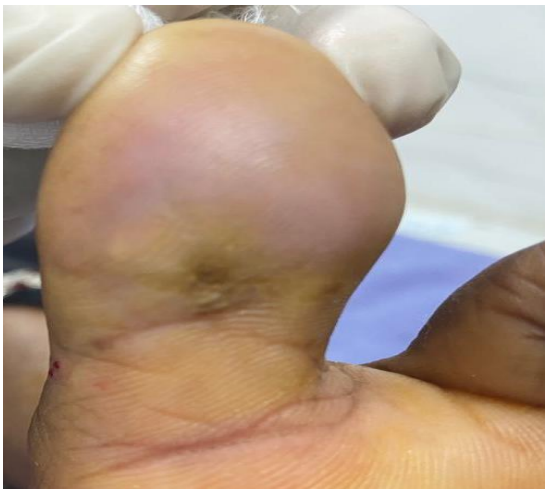


Figure 1: The examination reveals a firm to hard swelling on the plantar aspect of left toe.

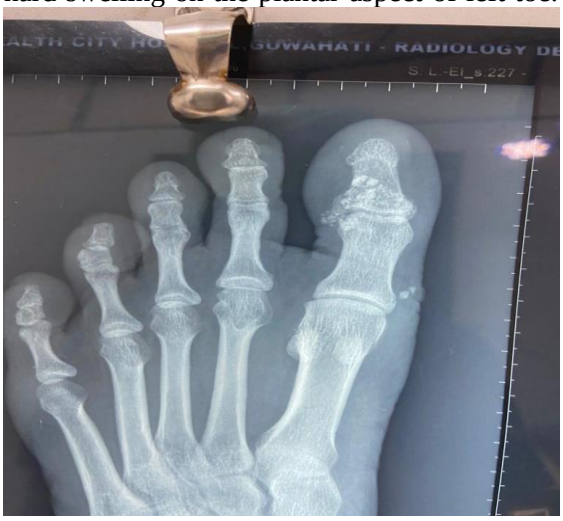
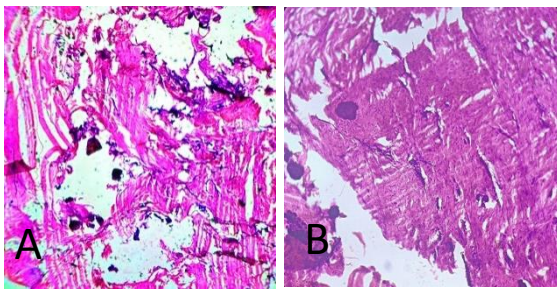


Figure 2: The above X-ray image shows calcification of the left great toe without any joint involvement.

diseases that lead to connective tissue damage. The most common diseases are systemic sclerosis, dermatomyositis, and systemic lupus erythematosus. [6]

Metastatic calcification is deposition of calcium salts in the presence of abnormal serum calcium and phosphorus levels. Calcium deposition occurs when the calcium phosphate

product exceeds 70 mg/dL and resolve with correction of the calcium, phosphorus levels. [2] These deposits are usually located in periarticular regions. The most common cause of metastatic calcification is chronic kidney failure. [3]



Figures 3A&B: Show basophilic deposits indicating presence of calcium, H&E 40x

Idiopathic calcification occurs when there is deposition of calcium salts without underlying tissue damage or abnormal calcium or phosphorus levels. There are three types: familial tumoral calcinosis, subepidermal calcinosis, and scrotal calcinosis. [7] Familial tumoral calcinosis is seen in healthy adolescent patients. There is increased uptake of phosphate in the proximal tubule of the kidney. Calcification occurs around major joints and can be subcutaneous or intramuscular. Subepidermal calcified nodules or Winder nodular calcinosis can be seen in children and can present at birth. It occurs on the head and extremities as solitary, hard, white-yellow papules. Scrotal calcinosis presents as nodules and masses on the scrotum. [8] Histopathologically, calcium deposits stain dark blue with haematoxylin and eosin (H and E) and black with von Kossa stains. [4]

Iatrogenic calcification occurs in patients that are receiving calcium or phosphate containing substances. [8] It has been seen with intravenous calcium gluconate, calcium chloride, and para-amino salicylic acid during the treatment of pulmonary tuberculosis. It is also seen after administration of electrodes with pastes containing calcium chloride for electroencephalography. Calcification can be prevented by diluting the calcium solution and lowering phosphorus levels before administration. [9]

Calciphylaxis, or calcific uremic arteriolopathy, is a life-threatening condition characterized by progressive calcification of small- and medium-sized vessels of the subcutis, often accompanied by necrosis. It most frequently arises in the setting of hyperparathyroidism associated with chronic renal failure. [10] Calciphylaxis is often associated with an elevated serum calcium/phosphate product, with other factors, including several glycoproteins (G1a protein and glycopontin), likely playing a role in the development of vascular calcification. [1]

Treatment

Smaller lesions have been reported to respond to warfarin, ceftriaxone, and intravenous immunoglobulin (IVIG). Surgical excision and carbon dioxide laser can also be effective treatments. Larger lesions respond to diltiazem, bisphosphonates, probenecid, aluminium hydroxide, and surgical excision or curettage. Patients with small and localized lesions are good candidates for surgical treatment, whereas more generalized disease will require medical management.

Conclusion

In this case, there was no evidence of connective tissue disorders known to cause dystrophic calcification. Abnormal mineral metabolism was ruled out by investigations, so metastatic calcinosis was not present. Skin biopsy showed calcium deposition in the dermis and subcutis without evidence of tissue necrosis. Hence, the case was concluded as idiopathic calcinosis cutis and reported for its interesting and uncommon presentation.

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