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FNAC of Bilateral Iliac Idiopathic Calcinosis Cutis: A Rare Case Report

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Abstract

We report a case of idiopathic calcinosis cutis diagnosed by fine needle aspiration cytology in a 70 year old female who presented with subcutaneous swellings near the bilateral iliac crests. Cytological finding of amorphous calcium salts with histiocytes and the appropriate clinical background led to the cytodiagnosis of idiopathic calcinosis cutis as subsequently confirmed on histopathology. Pitfalls in the diagnosis of calcinosis cutis on cytology smears are also discussed.

Keywords: Calcinosis; Skin; Fine needle aspiration biopsy

Introduction

Deposition of hydroxyapatite crystals of calcium phosphate in the skin leads to Calcinosis cutis. There are various causes such as abnormal calcium or phosphorus metabolism, tissue damage or idiopathic factors. Here we observe a case of bilateral idiopathic calcinosis cutis diagnosed on fine needle aspiration (FNA) cytology in an otherwise healthy woman. Points of concern for a correct interpretation of the cytological findings are also discussed.

Case Report

A female patient in her seventies presented with a bilateral slowly growing lump close to iliac crest for the last 5 years. There was no history of trauma or parenteral therapy or family history of similar lesions. No history of pain or discharge from the swelling was present. Clinically, there was no evidence of any inherited or connective tissue disorder.

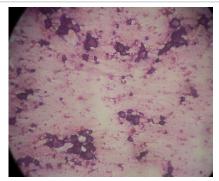


Figure 1: Amorphous granular material staining pinkish on H&E.

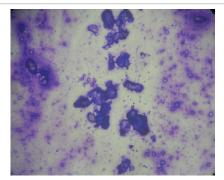


Figure 2: Amorphous granular material staining dark bluish on Giemsa stain.

The patient had no other swelling or cutaneous lesions such as ulceration or blister elsewhere in the body. The nodules were located subcutaneously and measured 4x4 cm of each. It was hard to palpate with no connection with the underlying bone. All biochemical and haematological investigations including serum calcium and phosphorus were within normal limits. Radiological investigation showed a calcified mass separate from adjacent bone. While doing FNA of the nodule with a 22-gauge needle attached to a 10 ml syringe, there was hard, gritty sensation and yielded chalky white granular material. May-Grunwald Giemsa (MGG) and Hematoxylin and Eosin (H&E) stains were used to stain the smears. Microscopy showed amorphous granular material staining pinkish on H&E and dark bluish on Giemsa stain (Figures 1 and 2). A few histiocytes were also seen. The smears were strongly positive with the Von-Kossa stain for calcium. A cytodiagnosis of calcinosis cutis was provided. The patient underwent surgical excision of nodules followed by histopathological examination. The gross specimen showed two hard, irregular lesions measuring of 4 × 4 cm with attached skin. On sectioning, the lesion was gritty with chalky white calcified areas (Figure 3). Representative sections were taken and stained with H&E and the Von-Kossa stain. Sections showed nests of calcified material separated by fibrous septa in the dermis without any significant inflammation (Figure 4). The overlying epithelium was normal. Calcified material was positive by the Von-Kossa stain, indicating a diagnosis of calcinosis cutis.

Discussion

There is localized and organized deposition of calcium in the skin in calcinosis cutis. It was first described by Virchow in 1855 [1]. From the pathogenesis point of view, the condition is classified as metastatic, dystrophic, iatrogenic, idiopathic and calciphylaxis. Serum calcium and phosphate levels remain normal in dystrophic calcification whereas it is abnormal in metastatic calcification.

Calciphylaxis is associated with small vessel calcification in the

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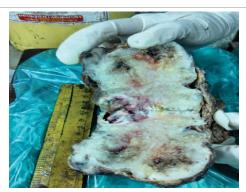


Figure 3: Chalky white solid areas with focal areas of haemorrhage.

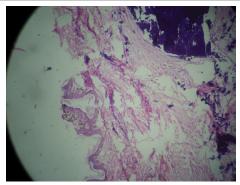


Figure 4: Nests of calcified material separated by fibrous septa in the dermis without any significant inflammation.

dermis and subcutaneous fat causing infarction and there may be associated hyperparathyroidism and disturbances in the phosphate and calcium metabolism [2].

The idiopathic calcinosis term is used in the absence of any identifiable cause of tissue calcification. In the present case, a negative history of trauma and parenteral therapy or any preceding pathological lesion at the site, along with normal serum calcium and phosphorus levels clearly excluded the possibility of dystrophic, iatrogenic and metastatic causes.

The pathogenesis of calcification is unknown. However, levels of gamma carboxyglutamic acid (GIa) have been found to be elevated in calcified tissue as well as in the urine of patients with calcinosis. It has been suggested that de novo production of GIa can lead to ectopic soft tissue calcification [3,4].

FNA samples yielding abundant calcium require careful consideration of certain entities that include calcified fibrous pseudotumor, calcified epidermal cyst, sarcoidosis, tuberculosis, lymphoepithelial lesion, pilomatricoma, osteitis fibrosa cystica, and extra skeletal osteosarcoma in the differential diagnosis. A calcified fibrous pseudotumor shows abundant hyalinised collagen, fat, and neurovascularbundles along with calcification [5]. Calcified tuberculosis and sarcoidosis show a granulomatous reaction [6], whereas calcified

epidermal cyst shows anucleate and nucleate squames. Pilomatricoma shows basaloid cells, ghost cells, and multinucleated giant cells in addition to calcification [7]. Lymphoepithelial lesions show a polymorphous population of lymphoid cells along with histiocytes and calcification [8]. Absence of any tumor cells rule out extraskeletal osteosarcoma. The clinical evaluation helps in the exclusion of osteitis fibrosa cystica. Reiter et al reviewed various conditions that may lead to skin calcification and provided information regarding laboratory tests required to differentiate various types of calcinosis cutis [2]. The treatment for small calcified deposits and large localized lesions is surgical excision which is curative and also allows histopathological examination that is required for confirmation of the diagnosis, whereas systemic therapy is required for disseminated and extended calcinosis. Various reported treatment modalities with beneficial effects include warfarin, bisphosphonates, minocycline, ceftriaxone, diltiazem, aluminum hydroxide, probenacid, intralesional corticosteroids, intravenous immunoglobulins, curettage, carbon dioxide laser, and extracorporeal shock wave lithotripsy [9].

Till date, there are very few case reports on FNA cytology of idiopathic calcinosis cutis [10-12] which if properly interpreted can lead to correct cytodiagnosis of this disorder. The technique is of great diagnostic importance in determining cases requiring medical rather than surgical treatment.

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