

Facial Dystrophic Calcinosis Cutis Post Acne

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Abstract: *Acne vulgaris is a disorder of the pilosebaceous unit, characterized by inflammatory and non-inflammatory skin lesions. Common sequelae of acne include post-acne hyperpigmentation, erythema, and scarring, often associated with significant psychological distress. Calcinosis cutis is the deposition of calcium salts in the skin, and its occurrence following acne vulgaris is rarely reported in the literature and is likely an underdiagnosed entity. Herein, we report a case of dystrophic calcinosis cutis post acne in a 52-year-old female.*

Keywords: Acne, dystrophic, calcinosis, calcium, basophilic

1. Introduction

Calcinosis cutis is the pathological deposition of insoluble calcium salt in the skin and subcutaneous tissue. It is broadly classified into four main subtypes: Dystrophic, metastatic, idiopathic, and iatrogenic. Calcinosis following acne represents a form of dystrophic calcinosis cutis, wherein prior tissue damage or inflammation act as a nidus for deposition of insoluble calcium salts.[1]

2. Case Report

A 52-year-old female presented with skin-colored, raised, asymptomatic lesions on both cheeks for 4 years. She reported a history of long-standing acne beginning in adolescence and persisting into adulthood for which she received multiple topical and oral anti-acne treatments without improvement, resulting in low self-esteem. Examination revealed multiple asymptomatic firm skin-colored papules ranging in size from 1 to 3 mm over both cheeks, with few rolling scars on both cheeks (Fig 1) (Fig 2). Dermoscopy showed multiple ill-defined to well-defined yellowish opaque structureless areas, with attenuation of the pigment network and absent vascular structures (Fig. 3). Skin Biopsy revealed a sparse chronic inflammatory infiltrate surrounding the sebaceous glands and basophilic well-circumscribed areas of acellular material corresponding to calcification in the mid-to-deep dermis (Fig 4). Plasma calcium, phosphate, parathyroid hormone, and vitamin D were within normal limits. A diagnosis of dystrophic calcinosis cutis post acne vulgaris was made.

3. Discussion

Acne vulgaris is a condition of the pilosebaceous unit characterized by non-inflammatory lesions like comedones and inflammatory lesions such as papules, pustules, and cysts, commonly seen in the adolescent and young adult age group. A comedo is a plug of keratin and sebum in a dilated pilosebaceous canal. Inflammatory lesions, on the other hand, arise when lipid material is released into the dermis following rupture of the pilosebaceous unit. This, in turn, sets up an inflammatory response in the dermis. [2]

Healing of inflammatory lesions typically involves fibrous tissue deposition, eventually leading to scar formation. However, sometimes the epidermal portion of the comedone wall forms sheaths of epithelium to surround inflammatory material in the dermis. This encapsulated mass, along with necrotic tissues, which are produced as part of healing, provides a nidus for the formation of calcifications known as calcinosis cutis. [3][4][5]

Hopkins first reported the phenomenon in 1928, wherein chronic long-standing acne vulgaris (AV) led to dystrophic calcinosis cutis as a complication.[6] The condition usually presents as multiple firm to hard, asymptomatic papules and nodules, and rarely as a solitary lesion. It occurs when calcium gets deposited in previously damaged or inflamed tissues and is seen with autoimmune diseases, infections, inflammatory processes, and cutaneous neoplasms. Calcification in post-acne vulgaris is considered an inflammatory dystrophic calcification. [7].

Diagnosis of this entity is based on clinical suspicion, radiographic tests, blood tests, and biopsy. It is imperative to check serum calcium, phosphorus, vitamin D, and parathyroid levels to exclude any abnormality of systemic calcium homeostasis. Histopathology shows deposits of calcium in the dermis and/or hypodermis appearing as amorphous basophilic material on routine staining and black on Von Kossa staining, surrounded by fibrous tissue or inflammation. [8] Being a rare disease, there are no controlled clinical trials on its treatment. Management of calcinosis cutis typically involves carbon dioxide laser or surgical excision of the lesions, as medical treatments have not proven to be consistently effective. [9]

Our patient had a long-standing history of moderately severe acne, supported by her history and the presence of residual acne scars. The biopsy was confirmatory for calcinosis, while other blood parameters were within the normal range. Hence, a diagnosis of postacne calcinosis cutis was made.

4. Conclusion

Complications of inflammatory acne include post-inflammatory hyperpigmentation, erythema, scarring, and the

rare but often misdiagnosed entity of dystrophic calcinosis cutis. It generally occurs in patients with a history of long-standing or severe inflammatory acne or both.

Source(s) of support: none

Presentation at a meeting: not been presented

Conflicting Interest (If present, give more details): none

Consent for photograph: obtained from patient

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Figures and Legends:



Figure 1: Multiple skin-colored papules ranging in size from 1 to 3 mm over right cheek. with few rolling scars.



Figure 2: Multiple skin-colored papules ranging in size from 1 to 3 mm over left cheek.



Figure 3: Dermoscopy performed with Dinolite dermoscope showing two well-defined yellowish opaque structureless areas, with attenuation of the pigment network and absent vascular structures.

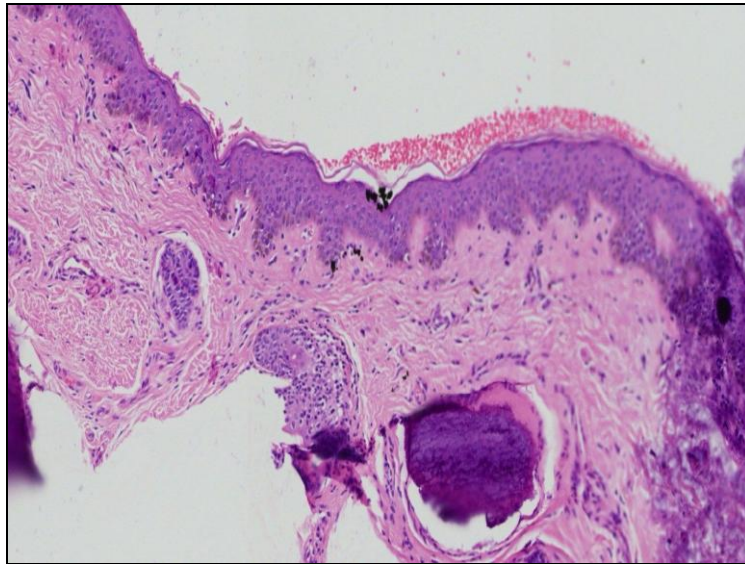


Figure 4: Histopathology showing a sparse chronic inflammatory infiltrate surrounding the sebaceous glands and basophilic well-circumscribed areas of acellular material corresponding to calcification in the mid-to-deep dermis.