Calcinosis cutis secondary to facial acne vulgaris: A rare complication

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ABSTRACT

Acne vulgaris is a common dermatological disease commonly affecting the adolescent and young adults. It is characterized by the presence of pleomorphic skin lesions such as comadones, papules, pustules, and nodules. The common complications are postacne hyperpigmentation and scarring causing psychological impact. Calcinosis cutis is the pathologic deposition of insoluble calcium salt in the skin and subcutaneous tissue. Calcinosis cutis following acne vulgaris is rarely reported in the literature. We report a case of calcinosis cutis in acne vulgaris in a 55-year-old man.

Key words: Acne vulgaris, calcinosis cutis, complications, facial

INTRODUCTION

Acne is a benign dermatological condition caused by overproduction of sebum by sebaceous gland. Calcinosis cutis is the pathologic deposition of insoluble calcium salt in the skin and subcutaneous tissue. It is classified into four main subtypes: Dystrophic, metastatic, idiopathic, and iatrogenic. Dystrophic calcinosis cutis follows inflammation, infection, connective tissue diseases, and so on. Complications of acne vulgaris such as calcinosis cutis are rarely reported previously in the literature.[1] We present such a rare case.

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CASE REPORT

A 55-year-old man who is a diagnosed case of obstructive sleep apnea (OSA) presented with symptoms of sinusitis for a period of 15 days. A computed tomography (CT) scan was done to confirm the diagnosis of sinusitis. However, CT scan showed multiple soft tissue calcification distributed all over the face mainly in the region of distribution of acne vulgaris suggestive of calcinosis cutis [Figure 1]. The patient was given antibiotics and supportive treatment for sinusitis after which he recovered completely. He reported that he had severe acne vulgaris in his adolescence for which he did not take any treatment. On retrospective questioning, there was no history suggestive of connective tissue disease or any malignancy. There was no significant past, personal, and family history. General physical and systemic examination was normal. However, the patient also had multiple skin-colored papules of various sizes ranging from pin head to maximum of 5 mm over the face predominantly on cheeks [Figure 2], nose, and forehead for a period of 25 years. Investigations of the patient were done to look for cause of such calcification. The plasma calcium, phosphate, vitamin D, and parathormone levels were within normal range. However, all other hematological and biochemical investigations including complete blood count and peripheral smear, liver, kidney function tests, and urine microscopy were normal. Serum immunoelectrophoresis was normal. Antinuclear antibody, rheumatoid factor, p- and c-ANCA, anti-topoisomerase, anticentromere antibodies, and HIV serology were negative. Contrast enhanced CT of abdomen and chest was normal. Bone marrow examination was normal.

A punch biopsy was done from the acne scars on the face, which showed areas of small

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calcific deposits in the upper-to-mid dermis. However, no bony trabecules were seen in the biopsy [Figure 3]. A diagnosis of postacne scar calcification was made. The patient was reassured of the benign nature of these findings.

DISCUSSION

Acne vulgaris is a common dermatological disorder. It is caused by abnormal follicular hyperkeratinization and overproduction of sebum by the sebaceous gland. Acne vulgaris affects nearly 80% of the population between 12 and 25 years—without gender, ethnicity, or race prevalence differences.[2] The pathophysiology of acne vulgaris includes increased sebum production, follicular hyperkeratinization, proliferation of *Propionibacterium acnes*, and production of inflammation.[3]

Calcinosis cutis means deposition of calcium in the skin. It is characterized by hydroxyapatite crystals and amorphous calcium phosphates deposited in skin.[4] Usually calcinosis cutis presents as multiple, hard, whitish papules, plaques, or nodules. It is known to occur in a variety of disorders and classified into four subtypes according to its etiology: Dystrophic, metastatic, iatrogenic, and idiopathic. The term dystrophic calcinosis is used for calcification associated with infection, inflammatory processes, cutaneous neoplasm, or connective tissue diseases.[5] It is the most common type of ectopic calcification and develops around localized tissue damage, with no alterations of calcium or phosphate metabolism. Saavendra *et al*. reported two cases of dystrophic calcinosis cutis secondary to acne.[6] Worstman *et al*. found six patients with calcinosis cutis secondary to acne vulgaris.[1] In contrast, metastatic calcification is characterized by abnormal calcium and/or phosphate metabolism that leads to the precipitation of calcium in cutaneous and subcutaneous tissue.

Idiopathic calcification appears without any underlying tissue damage or metabolic disorder. Medical intervention can cause tissue damage or disturbances in calcium and phosphate metabolism, leading to soft tissue calcification in some cases known as iatrogenic calcinosis cutis.

In a normal tissue, ectopic deposits of calcium salts develop when the calcium phosphate product in plasma exceeds 70 mg/dL. However, in a damaged tissue the following pathogenic phenomena may play a role—increased intracellular calcium concentration, denaturation of proteins that preferentially bind phosphate, genetic mutations of elastic fibers and collagen, and increased g-carboxyglutamic acid.

Calcinosis cutis is a rare disease, so there are no controlled clinical trials on its treatment. Various medical and surgical modalities have been found to be beneficial but none has been accepted as standard. Medical management include warfarin, bisphosphonates, minocycline, ceftriaxone, diltiazem, aluminum hydroxide, probenecid, intralesional corticosteroids,

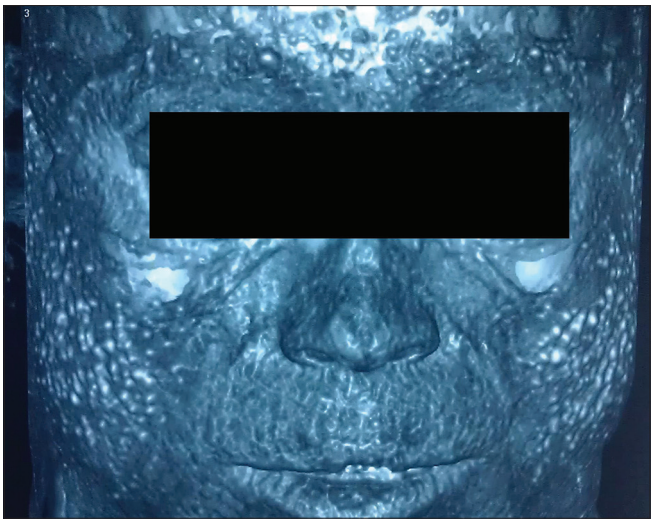

Figure 1: Computed tomography scan showing multiple soft tissue calcification distributed all over the face mainly in the region of distribution of acne vulgaris suggestive of calcinosis cutis

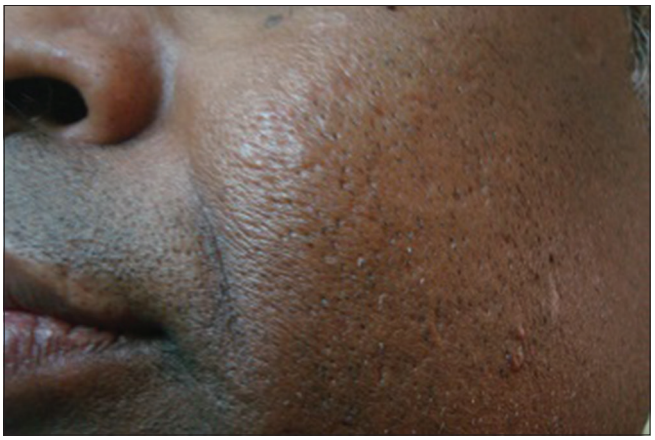

Figure 2: A punch biopsy from the acne scars on the face showing areas of small calcific deposits in the upper-to-mid dermis

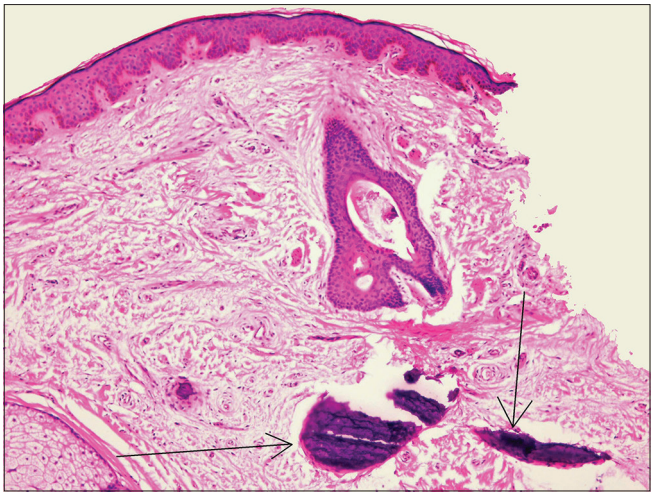

Figure 3: Multiple skin-colored papules of various sizes ranging from pin head to maximum of 5 mm over the left cheek

and intravenous immunoglobulin.[7] Surgical modalities include curettage, surgical excision, carbon dioxide laser, and extracorporeal shock wave lithotripsy.[8]

Our patient had a history of acne lesions during puberty, following which scar lesions were present on his face. A biopsy from those scar lesions showed calcific deposits on biopsy. Our patient was a diagnosed case of OSA since 5 years on treatment. However, despite an extensive search no relationship was found between OSA and calcinosis cutis. Hence, a diagnosis of postacne calcinosis cutis was made.

CONCLUSION

Thus, it is concluded that dystrophic calcinosis cutis can develop rarely secondary to acne vulgaris. Tests to rule out connective tissue disease should be done in all cases.

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Conflicts of interest

There are no conflicts of interest.

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