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Acquired perforating dermatosis associated with oral carcinoma: A rare association

Dr. Kansaria Jayti B. (MBBS); Dr. Jerajani Hemangi R. (D.V.D, M.D, FIAD); Dr. Udare Satish (M.D, D.V.D)
(Department of Dermatology, MGM Medical College and Hospital, MGMIHS, India.)

Abstract: *Acquired perforating dermatosis (APD) is an uncommon cutaneous perforating disorder, frequently associated with diabetes mellitus (DM) and chronic renal failure (CRF). This skin disorder may also develop in association with other systemic disorders like malignancy. We present a rare case of 53 year old male with clinical, dermoscopic and histopathological features suggestive of reactive perforating collagenosis (RPC), in absence of DM or CRF, and in association with history of oral squamous cell carcinoma (SCC).*
Keywords: *Acquired perforating dermatosis, dermoscopy, oral cancer, oral squamous cell carcinoma, Reactive perforating collagenosis*

I. Introduction

Acquired perforating dermatosis (APD) is characterized clinically by hyperkeratotic papules and nodules, and histopathologically by transepidermal elimination of various substances such as keratin, collagen and elastic fibers. The disease arises in adulthood, usually in association with DM or CRF.[1] In recent years, however, it has also been reported with various other disorders, such as malignant, [2],[3] hepatic and endocrinological disorders, AIDS, tuberculosis, pulmonary aspergillosis, neurodermatitis, atopic dermatitis and scabies.[1] Here we present a rare case of APD, in absence of DM or CRF, associated with history of buccal SCC, its dermoscopy findings and probable pathophysiology of APD in oral SCC.

II. Case Report

A 53 year old male farmer, presented to us with complaints of raised reddish severe itchy lesions on back and both legs since two weeks. Initially lesions were few to start, progressively increasing in number. Patient had taken multiple treatments for the skin lesions. He had partial temporary improvement following which the lesions reoccurred. Patient had no features suggestive of diabetes, hypertension and renal disorder but gave history of tobacco chewing since several years, discontinued since past three months. Patient was a known case of right buccal mucosa ulcero-proliferative growth three months ago which was biopsied, diagnosed as well-differentiated SCC & operated for the same with no post-cancer chemotherapy, radiotherapy or medication. Patient was advised post-operation radiotherapy, but did not comply with the same.

On cutaneous examination, multiple bilaterally asymmetrical pruritic erythematous dome-shaped, umbilicated, hyperkeratotic papulonodules predominantly on back& lower legs, few on abdomen & forearms. Face, neck had post-operative scars present [Fig. 1]. Koebner phenomenon was absent.

The marked lesion was punch biopsied and examined dermoscopically. Dermoscopy showed an elevated structureless yellowish-brown centre with a white keratin rim surrounded by thick pink glossy peripheral translucent area on an erythematous base, measuring 5mm in size [Fig. 2].

On investigating, all routine blood reports including blood sugars, thyroid, liver and renal function tests showed no abnormality.

Histopathology H & E stain showed a cup –shaped depression of epidermis filled with plug of parakeratotic keratin, some collagen and inflammatory cells. Underlying epidermis is thin with fine slits through which collagen fibers in vertical orientation are extruded [Fig. 3 (a),(b)]

So the diagnosis of acquired perforating dermatosis was made and special stains were done for confirmation. Masson Trichrome stain was done and found to be positive. Transepidermal elimination of collagen through several vertical epidermal slits was clearly appreciated.[Fig. 3 (c),(d)]. Verhoeff-van Gieson (VVG) staining for elastic fibers was negative. [Fig. 3 (e)].

Biopsy of right buccal mucosa done three months ago revealed parakeratotic, hyperplasic stratified squamous epithelium with papillary surface and dysplastic features. Invasion of small and large malignant epithelial islands into superficial connective tissue stroma along with peritumoral moderate inflammatory cell infiltrate was seen. It was diagnosed as well-differentiated squamous cell carcinoma [Fig. 4 (a),(b)].

Thus a final diagnosis of acquired perforating dermatosis: reactive perforating collagenosis-like, was made with most likely association with right buccal mucosal well-differentiated SCC.

III. Figures

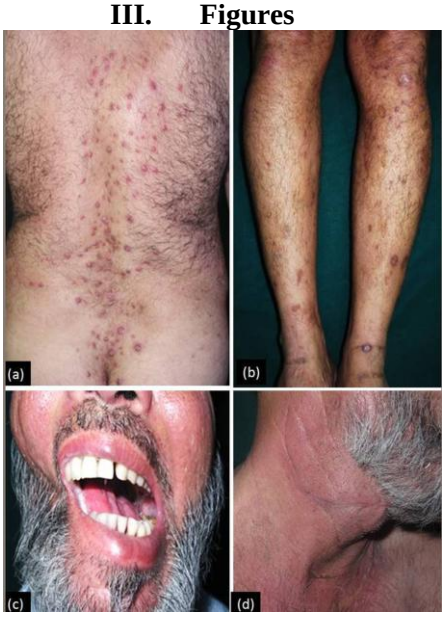

Fig. 1: Multiple hyperkeratotic papulonodules on back (a), on both legs (b). Operated site of oral cancer seen on right cheek (c), post-operative scars on neck (d).

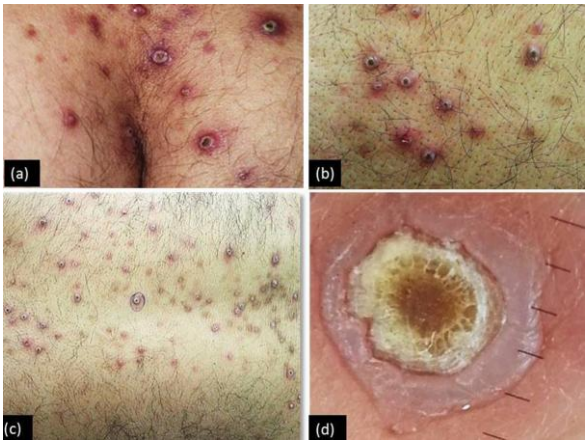

Fig. 2: Closer view of lesions on buttocks (a), on back (b). Single marked lesion on back (c), and its dermoscopic appearance (d).

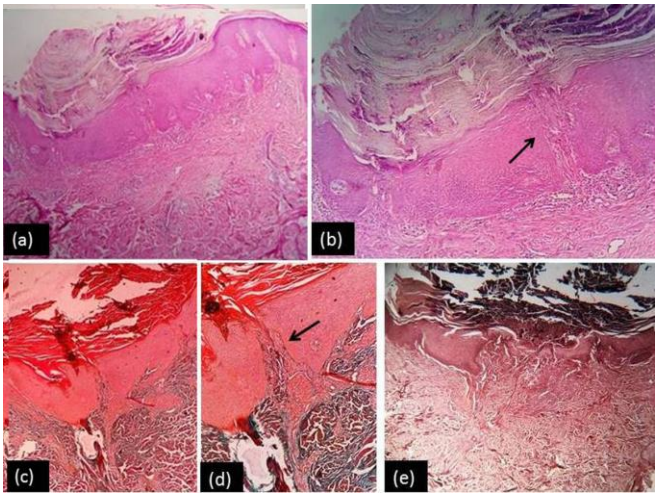

Fig. 3: H&E stain on low power showing cup-shaped depression in epidermis (a) and high power showing TEE (b). MT stain on low power (c) and high power (d) showing TEE of collagen (black arrow). Negative VVG stain (e).

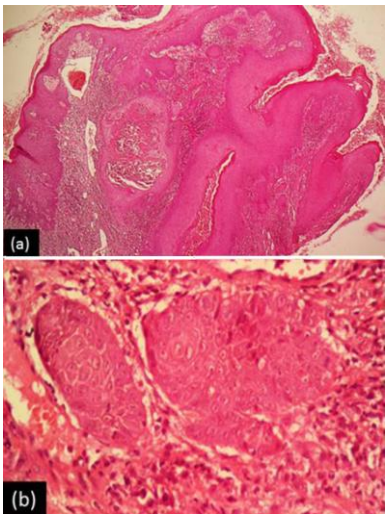

Fig. 4: H&E stain on low power (a) and high power (b) suggestive of well-differentiated oral SSC.

IV. Conclusion

APD presents clinically as multiple hyperkeratotic papulonodules and histopathologically by transepidermal elimination of various substances such as keratin, collagen and elastic fibers.[1]

RPC (reactive perforating dermatosis) which is a subtype of APD clinically presents as multiple hyperkeratotic umbilicated papulonodules and histopathologically shows transepidermal elimination of collagen with masson trichome stain positive.[1] Although APD is frequently associated with DM and CRF, this skin disorder may also develop in patients with other systemic disorders, and in those without any medical problems.[1] APD (RPC-like) in absence of DM or CRF, associated with history of buccal mucosal well-differentiated SCC, was seen in our case. Also, in our case we have recorded the dermoscopic finding and its histopathological correlation. The structureless yellowish-brown centre on dermoscopy corresponds to the cup-shaped depression in epidermis filled with plug of keratin, collagen and inflammatory cells on histopathology. Also, the white rim at crater margin corresponds to parakeratotic keratin. The pink glossy translucent peripheral area can be correlated with the skin folds surrounding the central crater.[4]

The diagnosis was confirmed with special stains for collagen and elastin, which clearly showed transepidermal elimination (TEE) of collagen fibres through thin vertical slits in epidermis, and VVG stain negative for elastic fibres.

Diseases reported to be associated with primary APD are Hodgkin's disease, mixed histiocytic-lymphocytic lymphoma, liver carcinoma, pancreas adenocarcinoma, prostate carcinoma, myelodysplastic syndrome, hypothyroidism, hyperparathyroidism, sick euthyroid syndrome, neurodermatitis, hepatic dysfunction, primary sclerosing cholangitis, pulmonary aspergillosis, AIDS, lupus vulgaris, atopic dermatitis, scabies, Poland syndrome and thyroid papillary carcinoma.[1] However, to the best of our knowledge, no association with oral cancer has been reported till date.

Pathogenesis of itch associated with malignancy and manifesting as APD is poorly understood. Cytokines such as IL-6 and IL-8, which have been reported to increase in end-stage renal disease (ESRD) associated itch and atopic dermatitis respectively, are closely related to pathophysiology of lymphoma.[5] Kimmel et al. found that hemodialysis patients with pruritus had significantly enhanced Th1 cytokines and IL-6 levels. Lymphoma and atopic dermatitis thus have IL-6 and IL-8 as probable mediators of itch in malignancy and other systemic diseases leading to APD.

The expression of two cellular genes which are uniquely associated with oral SCC: interleukin (IL)-6 and IL-8 have been identified at high concentrations in serum and saliva respectively, using laser-capture microdissection and global gene expression profiling using high density oligonucleotide arrays. These cytokines have also been linked with increased tumor growth and metastasis,[6] and could thus contribute to the itch in our patient leading to pathogenesis of APD.

However in our case, patient started developing skin lesions soon after complete excision of tumour and neck dissection. Severe pruritus has known association with malignancy, and severe pruritus per se has been reported to cause perforating dermatosis.

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