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Chronic Actinic Dermatitis: An Unusual Photodermatosis

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Abstract

Chronic actinic dermatitis (CAD) is a photosensitivity disorder that is defined as a persistent eczematous eruption in sun-exposed areas. The pathogenesis of CAD has not been completely understood. It typically afflicts men in the fifth decade of life or older. It manifested as an eczematous process with pruritus, erythematous and lichenified plaques involving sun-exposed areas mostly head and neck and distal extremities, sparing areas covered by clothing. Phototesting is recommended for further evaluation of suspected CAD. The key histological diagnostic clues included a brisk lymphoid infiltrate, eosinophils, plasma cells, and prominent dermal dendrocytes with multinucleated cells in the background or reticular fibroplasias. Current management for CAD includes broad-spectrum sunscreens, physical protection from sunlight, allergen avoidance, topical corticosteroids and tacrolimus, systemic corticosteroids and immunosuppressive agents such as azathioprine, cyclosporine, and mycophenolate mofetil, and photo (chemo) therapy.

Keywords: Chronic actinic dermatitis; Photosensitivity; Photodermatosis

Abbreviations: CAD: Chronic Actinic Dermatitis; UV: Ultraviolet; MED: Minimal Erythema Dose.

Introduction

Chronic actinic dermatitis (CAD) is a rare and severe idiopathic Photodermatosis classified under the term immunologically mediated Photodermatosis. It typically afflicts men in the fifth decade of life or older and characterized by pruritic eczematous and lichenified plaques sharply demarcated to sun- exposed areas. The diagnosis is based on clinical, histopathological and photo biological features. The treatment is essentially based on strict photo protection measures [1-5]. We report the case of an adult woman with this rare entity.

Case Presentation

A 52-year-old woman, with a long history of chronic photo exposure, who consulted for erythematous itchy lesions affecting the face evolving for 2 years. Otherwise the patient reporte`d photosensitivity, arthralgia or dry syndrome. Dermatologic examination revealed an eczematous and lichenified pruritic patches limited to sun-exposed areas of the face. Linear erosions in the chin and cheeks. Accentuation of wrinkles and diffuse xerosis (Figure 1). Dermoscopy demonstrated the presence of an erythematous background and telangiectasia (Figure 2). The main diagnoses were actinic dermatitis, airborne eczema and pigmented lichen. Histological examination of a punch biopsy specimen of the lesion showed an epidermis of irregular thickness, spongiosis, edema,

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telangiectasia with inflammatory infiltrate of the dermis. As well as, actinic elastosis with mononucleated inflammatory infiltrates in the deep dermis (Figure 3&4). In light of the characteristic clinical and histopathologic features, a diagnosis of CAD was made. A treatment with topical corticosteroids has been initiated, in association to application of sunscreen and wearing sun protective clothing. A significant improvement and regression of erythema and pruritus, was noted with a decline of 18 months.

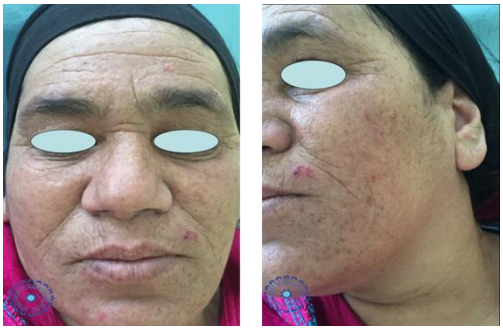

Figure 1: An eczematous and lichenified pruritic patches of the face with linear erosion and accentuation of wrinkles.

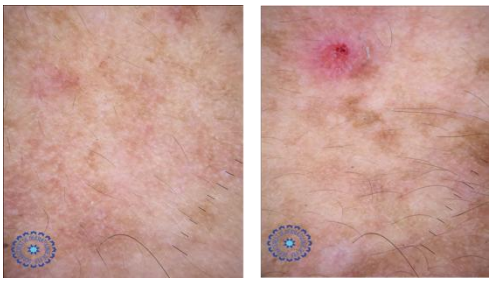

Figure 2: Dermoscopy showing an erythematous background, telangiectasia and erosions.

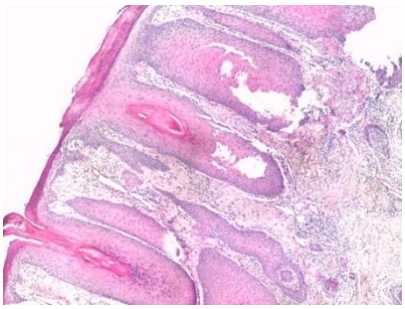

Figure 3: HES stain G x 50 => Epidermis of irregular thickness, spongiosis, edema, telangiectasia with inflammatory infiltrate of the dermis.

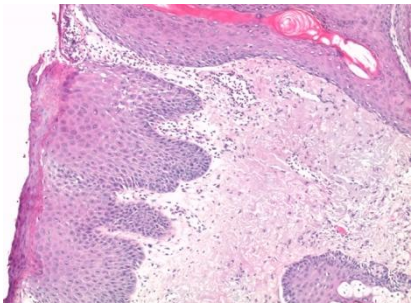

Figure 4: HES stain G x 100 => Chronic spongiotic dermatosis + Actinic elastosis.

Discussion

Chronic actinic dermatitis (CAD), previously known as actinic reticuloid, photosensitivity dermatitis, photosensitive eczema, and persistent light reaction [1,3], It is an uncommon inflammatory dermatosis characterized by dermatitis involving ultraviolet (UV) light-exposed skin with notable sparing of sun-protected areas. Rarely, spread of the exanthema to UV-protected areas of skin [2]. It commonly affects men older than 50 years who work or enjoy the outdoors. This condition can affect individuals of all skin types, although in the United States it is more commonly reported in persons with Fitzpatrick skin types V and VI [3]. Chronic actinic dermatitis is a disabling condition that is generally believed to worsen during the summer months or after prolonged exposure to sunlight [5]. The pathogenesis of CAD has not been completely elucidated. The clinical and histologic features, the presence of mostly CD81 T cells in the dermis, and the pattern of adhesion molecule activation in CAD resemble allergic contact dermatitis. Thus, a mechanism of delayed-type hypersensitivity can be inferred [6]. However, rather than an exogenous agent, the cutaneous antigen is likely endogenous and photo induced [4].

There are two postulated theories:

a. The patients have a neither exaggerated immune reaction to a photo antigen for some unknown reasons, while such reaction is suppressed in normal individuals.
b. There is cross-reactivity between a photo allergen and endogenous antigens in CAD patients, thus the endogenous anti- gen allows the immune response initiated by a photo allergen to persist even though the photo allergen is no longer present [1].

The clinical appearance is that of an eczematous process with pruritus, erythematous and lichenified plaques involving sun-exposed areas mostly head and neck and

distal extremities, sparing areas covered by clothing. However, generalized erythroderma with palmoplantar hyperkeratosis can ensue in severe cases [2,4]. Conditions that may mimic CAD include drug eruption, allergic or photo allergic contact dermatitis, cutaneous T-cell lymphoma ,and connective tissue diseases (such as acute or sub-acute cutaneous lupus erythematosus) [7]. Photo testing is recommended for further evaluation of suspected CAD. The most common action spectrum for CAD is UVB plus UVA, resulting in a decreased minimal erythema dose (MED) for both UVB and UVA in most patients. However, CAD may be seen with decreased MED- B or MED-A alone (12%–25%), or with a combination of sensitivity to UVB, UVA, and visible light [3]. Histopathological, CAD is characterized by epidermal spongiosis and acanthosis, often with hyperplasia, exocytosis, and a perivascular lymphocytic infiltrate with eosinophils. The upper dermis may contain large activated lymphocytes with some pleomorphism with eosinophils, plasma cells, and scattered stellate fibroblasts embedded in reticular fibroplasias [8]. Small collections of intraepithelial lymphocytes can be observed, but they differ from Pautrier micro abscesses by lacking sharp boundaries, halo effect, and being associated with ample spongiosis [9].

A recent study identifies a brisk lymphoid infiltrate, eosinophils, plasma cells, and prominent dermal dendrocytes with multinucleated cells in the background or reticular fibroplasias as key histological diagnostic clues [4]. CAD is commonly refractory to treatment. The management of patients with CAD requires sunlight avoidance, sunscreen protection, topical emollients, and topical steroids [4]. For more severe cases, oral corticosteroids and immunosuppressive therapy, such as azathioprine, methotrexate, tacrolimus, cyclosporine, and mycophenolate mofetil may be indicated [1,5]. First line therapy consists of strict photo protection and topical corticosteroids. Second line therapy uses systemic immunosuppression. Although, PUVA is considered an effective treatment for patients who had failed systemic immunosuppressant's, its place may be as a second line alternative rather than a third line treatment after failure of systemic immunosuppression [10]. Spontaneous resolution of chronic actinic dermatitis has been reported to be 10% over 5 years, 20% over 10 years, and 50% over 15 years [11]. Although, a few case reports have suggested the possibility of malignant transformation of pseudolymphomatous CAD or association with other malignancies [3].

Conclusion

Actinic dermatitis is the most severe of idiopathic photodermatosis. We report a new case confirmed histologically and had well responded to photo protection measures and topical corticosteroid.

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