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Chronic Actinic Dermatitis With Eosinophilia And Elevated Total Immunoglobulin E Serum: A Case Report

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Abstract
Chronic actinic dermatitis (CAD) is a chronic photosensitivity disease, characterized by pruritic eczematous lesions, mainly in sun-exposed areas. The etiology of CAD is still not well understood. Furthermore, increased levels of eosinophils and total immunoglobulin E (IgE) are common in CAD patients due to shifment of T-helper 2 (Th2) immunity from the T-helper 1 (Th1)/Th2 balanced status. We present a case of CAD in a 72-year-old male with a 7-month history of pruritus and erythematous lesions on his neck, forearms, and both thighs. The patient underwent skin biopsy, revealing histopathological features of spongiotic reaction in the epidermis and exocytosis of inflammatory lymphocytes which consistent with CAD. Laboratory results showed an elevated IgE serum and peripheral blood eosinophils. The patients were managed with photoprotection, topical corticosteroids, and oral antihistamine. Careful history taking, dermatological examination, and appropriate laboratory investigations are essential for diagnosis of CAD. In addition to blood eosinophilia and elevated IgE, there is evidence that UV radiation induces Th2 immunity through a variety of mechanisms. Interleukin-36γ (IL-36γ), IL-8, chemokine (C-C motif) ligand 17 (CCL17), and CCL18 have been shown to correlate with both eosinophil count and elevated IgE serum levels. Subsequent investigations should be conducted to determine the exact mechanism by which Th2 immunity and other factors such as IL-36 can contribute to the development of CAD.
Keywords: *chronic actinic dermatitis, eosinophil, immunoglobulin E, sun-exposed, ultraviolet radiation.*

1. INTRODUCTION

Chronic actinic dermatitis (CAD) is a rare, acquired, immune-mediated photodermatosis characterized by pruritic eczematous lesions in sun-exposed areas induced by ultraviolet B (UVB), ultraviolet A (UVA), and occasionally visible light (Amagai et al., 2019); (Chen et al., 2021); (Hsiao & Chu, 2014). CAD was previously known as actinic reticuloid, photosensitive eczema, photosensitive dermatitis, and persistent light reaction (Hsiao & Chu, 2014). By 1979, Hawk and Magnus were the first to introduce the term "chronic actinic dermatitis" and became widely accepted (Hsiao & Chu, 2014); (Lin et al., 2021); (Kim & Kim, 2018). The etiology of CAD is still not well understood. Evidence suggests that CAD is a delayed–type hypersensitivity reaction to endogenous photo-induced antigens (Amagai et al., 2019); (Lin et al., 2021); (Gu et al., 2023). CAD appears to be an allergic contact dermatitis–like reaction against UV–altered DNA or similar or associated molecules, perhaps as a result of enhanced immune reactivity resulting from concomitant airborne contact dermatitis, or other longstanding preexisting dermatitis, or a reduced immunosuppressive capacity in ultraviolet-exposed skin (Amagai et al., 2019). Moreover, there is a tendency of shift into T–helper 2 (Th2) immunity from the T–helper 1 (Th1)/Th2 balanced status in patients with CAD through UV radiation and leads to blood eosinophilia and increased IgE serum (L. Wang et al., 2023).

Clinical manifestations include eczematous pruritic patches and coalescing plaques on sun–exposed areas of scalp, face, neck, arms, hands, and back. Lichenification of affected areas is common due to chronic and pruritic nature of lesions (Artz et al., 2019); (Gu et al., 2023). There is sparing of deep skin creases, upper eyelids, finger webs, nasolabial folds, and skin behind the ear lobes (Amagai et al., 2019); (Artz et al., 2019). Earlier investigations of CAD were mostly from countries with temperate climates. However, the disease appears to have worldwide distribution (Artz et al., 2019). The incidence of CAD was thought to be related to sun exposure time (hours) (Lin et al., 2021). It is commonly seen in men older than 50 years with outdoor and sunlight exposures. Although, it affecs persons of all skin types, CAD is more

commonly reported in Fitzpatrick skin types V and VI in the United States (Lin et al., 2021); (Artz et al., 2019).

CAD may arise de novo in apparently normal skin or occur in areas previously affected by endogenous dermatitis (often atopic or seborrheic dermatitis); it has been reported to develop following drug photosensitivity, or in patients with polymorphic light eruption (PMLE) (Amagai et al., 2019); (Lin et al., 2021). Comprehensive diagnosis of CAD relies mainly on typical clinical manifestations, phototesting, histopathological examination, and laboratory evaluations including antinuclear antibody (ANA), human immunodeficiency virus (HIV), Sezary count, and total IgE serum (Amagai et al., 2019); (Artz et al., 2019); (Ko et al., 2016). Total IgE serum and eosinophil count may be elevated (even among those whose CAD has not supervened upon atopic dermatitis), with higher levels of IgE correlating with more severe disease. However, the reasons for their elevation remain unclear (Amagai et al., 2019); (Ko et al., 2016); (L. Wang et al., 2023). In this study, we present a 72–year–old male with CAD who had elevated total IgE serum and high eosinophil count without history of atopic dermatitis

2. CLINICAL CASE

A 72–year–old Sundanese male patient came to our Dermatology and Venereology Clinic, presented with a 7–month history of pruritus and erythematous lesions on his neck, forearms, and both thighs. The patient, a retired, engaged in sunbathing almost every day that exposing him to sunlight for 4–6 hours daily without any photoprotection. He was diagnosed with eczema at other clinic, and did not respond to antihistamines or to various topical treatments for about 2 months. There is no history of dandruff, red patches with greasy scales on both eyebrows, behind the ears, and chest. The patient denied a previous history of allergic diseases (urticaria, atopic dermatitis, allergic rhinitis, asthma), and no family history of allergic diseases was reported. History of applying topical preparations before the onset of the skin disorder is denied. There was no family history of comparable illnesses.

On physical examination, vital signs were within normal limits, normoweight, and no lymph node enlargement was observed. Dermatological assessment revealed multiple erythematous macules and plaques on the neck, forearms, and both thighs, with lichenification predominantly on both forearms (Figure 1). Multiple site punch biopsy in size of 5 milimeter (mm) was taken from the skin lesions on neck, left forearm and left thigh, and sent to Department of Pathological Anatomy for further investigation. Histopathological examination showed mild epidermal spongiosis with exocytosis of inflammatory lymphocytes. Edematous fibrocollagenous tissue were seen in dermal papillae, accompanied with perivascular lymphocytic infiltrate (Figure 2). Serum total immunoglobulin E was 629,1 IU/mL (normal <100 IU/mL) with a high eosinophil count was 15 % (normal 0–4%). ANA, Sezary count, and HIV test were examined with negative results. Consequently, the patient received a diagnosis of chronic actinic dermatitis.

We managed the patient with strict sun avoidance during peak hours, advised to adjust their lifestyle to minimise sun-exposure, and wearing photoprotective garments such as a close-weaved, long-sleeved, and dark colored clothes when going outdoors. We also initiated therapy with topical corticosteroids (clobetasol propionate 0.05% ointment), broad spectrum sunscreen, urea 10% lotion, and oral antihistamine namely cetirizine 10 mg/day. Systemic corticosteroids have not been given because the patient only has mild symptoms with a good response to topical corticosteroids. One month after therapy, the patient achieved progressive improvement, leaving hyperpigmented macules. The Dermatology Life Quality Index (DLQI) scores improved from 14 before treatment to 2 at the last visit. The patient is still under follow-up, with continuous improvement and no adverse effects.

3. RESULT AND DISCUSSION

CAD is a spectrum of diseases with chronic photosensitivity occurring mostly among middle-aged and older men, characterized by pruritic eczematous lesions, mainly in sun-exposed areas (Gu et al., 2023); (Ko et al., 2016). It is characterized by an abnormal broad–spectrum photosensitivity in the UVB and/or UVA spectra and even in the visible light range (Ko et al., 2016). CAD has been reported in the United States, Europe, Asia, and Africa, with increased incidence in the summertime, although also been reported from subtropical or tropical regions, such as Australia, India, and Singapore (Lin et al., 2021); (L. Wang et al., 2023). CAD typically affects men who are over 50 years old and either had outdoor activities with no familial inheritance (Ali et al., 2022). It affects persons of all skin types, but it is more

commonly reported in Fitzpatrick skin types V and VI in the USA (Artz et al., 2019). Research identified mean age among 15 patients with CAD was 58.6 years (range 28–82 years) (Hsiao & Chu, 2014). Most patients (>85%) had CAD after age 40 years. All 15 patients were Han Chinese, and most of the patient (66%) were Fitzpatrick's skin phenotype IV. The interval between disease onset and diagnosis of CAD confirmed by phototesting ranged from one month to more than ten years. A retrospective epidemiological study by (Gu et al., 2023) reported among the 488 patients, 344 were male, and 144 were female (ratio 2.4:1). The mean age was 53.9 years old (range: 17–84 years old), and the mean duration of illness was 4.3 years (range: 3 months–8 years). All patients had Fitzpatrick skin phototype IV. In this case, the patient was a 72–year–old male with a 7–month history of pruritus and erythematous lesions on his neck, forearms, and both thighs. The patient had previous history of sunbathing and many outdoor activities for couple years without sunscreen application.

The pathogenesis of CAD has not been fully uncovered, and many theories have been proposed over the past decades. Evidence suggests that CAD is a delayed type hypersensitivity reaction to endogenous photoinduced antigens. This is supported by the adhesion molecule activation pattern and the presence of CD8+ T cells within the dermis (Hsiao & Chu, 2014); (Artz et al., 2019). CAD is a representative example of the chronic effect of cumulative UV irradiation. Historically, it has been suggested that all CAD patients have a reduced minimal erythematous dose (MED) to UVB, the majority also have a reduced MED to UVA, and a minority respond to the visible wavelengths as well (Hsiao & Chu, 2014); (Kerr & Ibbotson, 2006). While healthy individuals have UV-inducible immunosuppression to photo-induced antigens in the skin, patients with CAD have lost this photoimmunosuppression ability, thus allowing the photo-induced antigen to stimulate the hypersensitivity reaction. DNA is suspected to be the antigenic molecule due to the similar action spectrums seen with sunburn inflammation and CAD (Amagai et al., 2019). Additionally, the prevalence of coexisting contact or photocontact allergy in CAD patients may play a role in CAD pathogenesis. Certain contact allergens, such as fragrance materials and colophony, which are potentially phototoxic, may be able to convert endogenous proteins into allergens. Another hypothesis proposes that a failure in the normal suppression of delayed type hypersensitivity by UV irradiation may be involved. It is likely that combinations of some of the aspects account for the pathogenesis of CAD (Hsiao & Chu, 2014); (Lin et al., 2021). Photoimmunosuppression serves as a physiologic role as an adaptive response of the skin to UV-induced modifications of proteins and an immunologic response to so-called neoantigen, and consequently leading to chronic inflammation. UV irradiation suppresses the antigen presentation to Th1 cells but enhance that to Th2 cells, explained by a shift into the Th2 immunity. The severity of CAD correlated significantly with the peripheral blood eosinophilia or total Ig-–E level, with CAD show a Th2 polarization as in most of allergic diseases, where some correlations existed between tissue eosinophilia and disease severity (Ko et al., 2016). In the present study, the CAD patients exhibited elevated serum levels of IL-36γ, IL-8, CCL17, and CCL18. Notably, IL-36γ was highly expressed in both the serum and skin lesions of CAD patients, particularly in the upper layers of the epidermis. IL-36 is positively correlated with eosinophil number and can promote cell survival, activation, and migration. Human eosinophils are a major source of IL–8. CCL17 and CCL18 are overexpressed in CAD lesioned skin and are correlated with disease severity. Taken together, study revealed the dysregulation of IL–36, IL–8, CCL17, and CCL18 in CAD, with IL–36 playing a critical role in eosinophil proliferation, IgE production, and secretion of other inflammation cytokines (L. Wang et al., 2023).

Research reported prolonged daily sun exposure in CAD from 488 patients in China from January 2014 to December 2018 (Gu et al., 2023). The number of patients with a daily sun-exposure time of more than 1, 3, and 5 hour was 158 (32.4%), 99 (20.3%), and 58 (11.9%). Moreover, more than one–fifth of CAD patients worked outdoors, and more than 80 cases had occupational exposure to strong light sources like sunlight, welding light, indoor artificial light, laser, etc (Gu et al., 2023). CAD can affect normal skin (de novo) or occur in areas previously affected by endogenous (mostly atopic or seborrheic dermatitis) photoallergic or allergic contact dermatitis, or, rarely PMLE (Amagai et al., 2019); (Artz et al., 2019); (Kerr & Ibbotson, 2006). Concurrent allergic contact sensitivity to plant allergens, fragrances, or sunscreens is common (Amagai et al., 2019). The patient in this case study had previously excessive amount of sun exposure without using any photoprotection. In addition, atopic dermatitis, photoallergic or allergic contact dermatitis, and seborrheic dermatitis were not present.

The diagnosis of CAD relies mainly on typical clinical manifestations, MED analysis, histopathological, and laboratory findings (Amagai et al., 2019); (Lin et al., 2021). Clinical manifestations of CAD include eczematous and often lichenified pruritic patches and confluent plaques limited to sun–exposed areas of the scalp, face, neck, chest, arms, hands, and back, with notable sparing of sun–protected areas, such as nasolabial folds, submental chin, upper eyelids, retroauricular areas, skin creases, and finger web spaces. Acute flares are associated with erythematous patches and papules with fine scale on sun-exposed areas (Ali et al., 2022); (Paek & Lim, 2014). Lichenification of affected areas is common due to the chronic and pruritic nature of lesions (Artz et al., 2019). In severe cases, papules and plaques may occur on sun protected sites, erythroderma and hyperkeratosis of palms and soles may be observed (Amagai et al., 2019); (Ali et al., 2022).

CAD can be classified into mild, moderate, or severe groups according to the clinical severity scores of CAD (CSS-CAD). The scoring system is based on the intensity of four clinical signs and the extent of involvement of those signs in the skin. Firstly, the extent of skin involvement was calculated at each anatomical site (face, upper chest, dorsa of the hands, and scalp), and gradings were determined for the percentage of body surface area (BSA) involved at each location (%BSA), and then, the affected area at each location was given a score from 0 to 5 (0, 0%; 1, 1–20%; 2, 21–40%; 3, 41–60%; 4, 61–80%; and 5, 81–100%) based on the % BSA of clinical signs. Next, the intensity of 4 clinical signs (erythema, vesiculation, excoriation and lichenification) was graded as followed: 0, no skin changes; 1, mild degree; 2, moderate degree; and 3, severe degree. Lastly, the % BSA was multiplied by the intensity grade of clinical signs, resulting in the total sum ranged 0–240 points, classified into mild (score 0– 80), moderate (score 81 160) or severe (score 161–240) (Ko et al., 2016). Phototesting shows an abnormal eczematous or erythematous response at doses lower than the expected MED. The most common action spectrum for CAD is UVB plus UVA, resulting in a decreased MED for both UVB and UVA in most patients. However, CAD may be seen with decreased MED UVB or MED UVA alone (12%–25%), or with a combination of sensitivity to UVB, UVA, and visible light (Ali et al., 2022); (C. X. Wang & Belsito, 2020); (Sidiropoulos et al., 2014).

Histopathologic appearances include epidermal spongiosis and acanthosis, sometimes with hyperplasia. There is usually a predominantly perivascular lymphocytic cellular infiltrate confined to the upper dermis that in milder cases may resemble chronic eczema (Amagai et al., 2019); (Ali et al., 2022). Atypical lymphocytes and exocytosis seen in some specimens may mimic histologic changes of cutaneous T–cell lymphoma (CTCL) (Ali et al., 2022). Papillary dermal fibrosis and a brisk infiltrate, both histologic signs of chronicity, may also be found. In severe cases the biopsy may show focal epidermal necrosis, papillary dermal collagen, fibrin deposition in the dermal–epidermal junction, and erosions (Ali et al., 2022); (C. X. Wang & Belsito, 2020); (Sidiropoulos et al., 2014). In this case, the patient had a chief complaint of erythematous macule, papules and plaques with scale and lichenified plaques mainly on his neck, forearms, and both thighs. CSS–CAD in this patient was 18 and categorized as mild degree. Histopathological examinations showed epidermal spongiosis with exocytosis of inflammatory lymphocytes. Edematous fibrocollagenous tissue were seen in dermal papillae, accompanied with perivascular lymphocytic infiltrate. These histopathological findings were consistent with CAD. Among this case report's limitations is its incapacity to perform phototesting. It is hard to diagnose CAD without phototesting. However, clinical features provide valuable point to diagnosis in cases where MED cannot be carried out in suspected CAD patients. We plan to do phototesting on the next visit due to redness on his lower back.

Conditions that may mimic CAD include drug eruption, allergic or photoallergic contact dermatitis, CTCL, and connective tissue diseases (such as acute or subacute cutaneous lupus erythematosus). Careful history taking, examination of morphology and distribution of lesions, and appropriate laboratory investigations are essential in arriving at the correct diagnosis (Amagai et al., 2019); (Ali et al., 2022). Laboratory investigations to rule out differential diagnosis include ANA test, HIV test, Sezary count, and total IgE serum (Amagai et al., 2019); (Artz et al., 2019); (Ko et al., 2016). Assessment of lupus autoantibodies is usually done to exclude the unlikely possibility of cutaneous lupus erythematosus (Amagai et al., 2019); (Ali et al., 2022). HIV testing is recommended, especially in younger patients, in whom CAD may be a presenting sign of HIV. Furthermore, the low CD4 counts observed may indicate that photosensitivity is a late feature of HIV infection (Ko et al., 2016); (Meola et al., 1997). In severe or erythrodermic CAD, there may be large numbers of circulating CD8+ Sézary cells without other

suggestions of malignancy. Serum IgE may be elevated even among those whose CAD has not supervened upon atopic dermatitis, with higher levels of IgE corelating with more–severe disease (Amagai et al., 2019); (Criado et al., 2023).

Previous study showed a tendency of immunological shifting from the Th1/Th2 balanced status into Th2 immunity in CAD patients might be affected by suppressor T cells counts as the CAD got worsened (Ko et al., 2016). Total IgE and the percentage of peripheral blood eosinophils were significantly correlated with the severity of CAD. Nevertheless, some evidences have been recently discovered that UV radiation drives host into Th2 immunity through various mechanisms in addition to blood eosinophilia and increased IgE. The chronic UV photosensitivity would be in favour of an accelerated IgE overproduction and the eosinophilia from the circuit of UV sensitized mast cells – dendritic cells – Th0 cells/Th2 cells – plasma cells in series of the patients with CAD.

Previous research had revealed an elevated serum levels of IL–36 in CAD patient (L. Wang et al., 2023). Notably, IL-36 was highly expressed in both the serum and skin lesions of CAD patients, particularly in the upper layers of the epidermis. IL–36 is positively correlated with eosinophil number and can promote cell survival, activation, and its migration. Furthermore, the release of IL-36 by keratinocytes, in conjunction with IL-4, triggers the class-switching of B cell, differentiation of plasma cells, and elevation of serum IgE level in CAD patient. In this case report, the patient had negative results of ANA, HIV, and Sezary cell test. Besides that, there were elevated IgE serum and peripheral blood eosinophils that can be found in CAD.

CAD treatment is frequently challenging and ineffective. Along with consistent use of broad–spectrum topical sunscreens with a high protection factor and minimal irritancy and allergenic potential, careful avoidance of UV radiation and aggravating contact allergens is crucial (Amagai et al., 2019); (Artz et al., 2019); (C. X. Wang & Belsito, 2020). In addition to photoprotection, patients may use topical corticosteroids or topical calcineurin inhibitors as first–line therapy. Mid–potency to high–potency topical corticosteroids are effective for the control of disease flare. However, the potential risks of skin atrophy, striae, and dyspigmentation preclude their long–term use (Amagai et al., 2019); (Artz et al., 2019); (Chen et al., 2021). Oral corticosteroids (prednisone 0.5–1.0 mg/kg/day) can be given for several weeks at a time for flares. Chronic oral steroid use is not recommended because of its adverse side–effect profile, including, but not limited to, weight gain, high blood sugar, increased risk for infections, osteoporosis, and fractures, adrenal gland suppression, delayed wound healing, glaucoma or cataracts, and steroid psychosis.5 For refractory CAD, however, other agents is often necessary and generally helpful if tolerated. Azathioprine 1.5 to 2.5 mg/kg/day, cyclosporine (3.5–5 mg/kg/day), and mycophenolate mofetil (25–40 mg/kg/d or 1–2 g/day) can produce remission in months after which it may be reduced in dosage (Artz et al., 2019); (Thomson et al., 2005); (Veedu et al., 2021). While less studied for this indication, methotrexate is also used, especially when CAD has arisen on a background of atopic eczema. Recently, oral tofacitinib, 5 mg twice daily, was able to induce near complete remission of signs and symptoms within 2 months in a recalcitrant case (Vesely et al., 2017). More localized skin immunosuppression by psoralen activated by UVA treatment also can be effective and is often initially accompanied by oral and topical corticosteroid therapy to reduce disease flares (Amagai et al., 2019); (Artz et al., 2019). In small series and case reports on CAD, dupilumab, a human monoclonal antibody designed to suppress Th2 signaling and inhibit the effects of IL-4 and IL-13, has recently demonstrated encouraging outcomes (Chen et al., 2021); (Chen et al., 2023).

Once established, CAD usually persists for years before resolving gradually (Amagai et al., 2019). Spontaneous resolution of CAD has been reported to be 10% over 5 years, 20% over 10 years, and 50% over 15 years (Ali et al., 2022). The presence of severe UVB photosensitivity and the identification of a contact allergen are poor prognostic factors for resolution in CAD patients (Rose et al., 2009). Patients may be counseled that their photosensitivity may eventually subside with photoprotection and avoidance of allergens. Fortunately, the proportion of patients with CAD that show clinical improvement or resolution increases as the duration of disease increases. At 5 years from diagnosis, 70% of patients showed improvement or resolution of symptoms, while at 15 years from diagnosis, 90% of patients showed clinical improvement or resolution of symptoms (Ali et al., 2022); (Artz et al., 2019).

4. CONCLUSION

CAD is an immune-mediated photodermatosis characterized by a high eosinophil count and total IgE as Th2 polarization occurs. At present, however, the precise mechanism underlying this phenomenon remains poorly understood. Future researches should be carried out to determine the precise mechanism through which Th2 immunity contributes to the development of CAD.

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