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Chronic Actinic Dermatitis in a 48-Year-Old Female: A Case Report on Clinical Insights and Treatment Strategies

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Abstract

Chronic Actinic Dermatitis (CAD) is an immune-mediated photodermatosis primarily affecting elderly males with prolonged sun exposure, though increasing reports highlight its occurrence in younger individuals, particularly those with darker skin types. CAD is characterized by pruritic eczematous lesions, lichenified papules, and plaques on sun-exposed areas, predominantly in response to ultraviolet (UV)B radiation, with some sensitivity to UVA and visible light. Concurrent photo contact allergies contribute to its pathogenesis. Despite well-established diagnostic criteria, clinical features remain crucial for diagnosis, with significant allergenic contact sensitivity observed in many cases. We report a case of a 48-year-old female with no significant medical history presenting with a longstanding, pruritic, painful rash localized to sun-exposed areas. Examination revealed eczematous patches and lichenification on the anterior neck, upper chest, forearms, and hands, involving approximately 25% of the body surface area. Her occupation as a manual labourer with significant sun exposure was a critical factor in disease onset. Photo testing confirmed heightened sensitivity to UVA and UVB radiation, leading to a diagnosis of CAD. The patient was managed with topical tacrolimus, stringent sun protection, and systemic therapy. Significant clinical improvement and sustained remission were achieved. Diagnosis of Chronic Actinic Dermatitis (CAD) relies heavily on clinical features and patient history. This case underscores the importance of occupational factors and sun exposure in CAD development. The patient's presentation of hyperpigmented and erythematous plaques, along with depigmented lesions on the scalp, was successfully managed with corticosteroids, antihistamines, and sun protection strategies. This case highlights the significance of early recognition and tailored therapeutic approaches to achieve effective management of CAD. [*Bangladesh Journal of Infectious Diseases, December 2024;11(2):217-222*]

Keywords: Autoimmune; Chronic; Eczema; Immunosuppressive; Photo testing; Tofacitinib

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Introduction

Chronic Actinic Dermatitis (CAD), an idiopathic photosensitive dermatosis, is a form of eczema that primarily affects sun-exposed areas of the skin. It is induced by UVB radiation, with occasional sensitivity to UVA and visible light[1]. The condition was first described by Hawk and Magnus in 1979, and the term has since been used to encompass a variety of photosensitive reactions, including

persistent light reactivity, actinic reticuloid, photosensitive eczema, and photosensitivity dermatitis[2-3]. These entities share common features but vary in clinical presentation and severity. CAD is most commonly observed in elderly men, particularly those over the age of 50, though cases in younger individuals, including those with darker skin types, have been increasingly reported[4-5].

The pathophysiology of CAD involves immune-mediated mechanisms, with the skin reacting to UV radiation by triggering inflammatory responses that result in eczematous lesions, lichenified papules, and plaques. The most pronounced photosensitivity is to UVB radiation, though in some cases, UVA and visible light can also play a role in exacerbating the condition [6]. This photosensitivity is often coupled with contact sensitivity to various allergens, which may contribute to the disease's development. CAD frequently occurs in individuals with a history of other dermatologic conditions, such as atopic dermatitis or allergic contact dermatitis, and can also present in previously healthy individuals[7].

Diagnosing CAD involves a combination of clinical evaluation, histopathological analysis, and photobiological testing. Histologically, skin biopsies may reveal epidermal hyperplasia, parakeratosis, and perivascular lymphocytic infiltration in the dermis. photo testing, which assesses the minimum erythema dose (MED) to UVB and UVA radiation, is essential in confirming the diagnosis, with affected individuals often demonstrating a significantly reduced threshold for UV-induced erythema. Photo patch testing, which assesses sensitivity to photo allergens, can further support the diagnosis, though its availability is often limited[8].

Management of CAD is multifaceted and typically includes strict sun avoidance, the use of broad-spectrum sunscreens, and protective clothing. In more severe cases, topical and systemic therapies, including corticosteroids and calcineurin inhibitors like tacrolimus, are employed to control inflammation. Phototherapy may also be considered in certain patients. Despite these therapeutic measures, long-term remission often requires diligent sun protection and careful management of both environmental and immune-mediated factors[9-10].

This case report highlights a 48-year-old female patient with CAD, focusing on her clinical features, diagnostic workup, and the treatment approaches that led to significant improvement.

Case Presentation

A 48-year-old female, with no prior significant medical or dermatologic history, presented with a prolonged history of pruritic, painful, and erythematous skin eruptions. The lesions were primarily localized to areas of chronic sun exposure, including the anterior neck, upper chest, forearms, and dorsal hands, encompassing approximately 25% of her total body surface area. *(Figure 1a & 1b)* Her occupation as a manual labourer necessitated prolonged and consistent sun exposure, which was suspected to be a significant contributing factor to the onset of her symptoms. On clinical examination, eczematous patches, areas of lichenification, and scaling were observed, particularly over the sun-exposed areas. In view of the clinical findings, a diagnosis of Chronic Actinic Dermatitis (CAD) was strongly considered. photo testing demonstrated a markedly reduced minimal erythema dose to both UVA and UVB radiation, confirming the heightened photosensitivity. These findings, in conjunction with the characteristic eczematous and lichenified plaques, supported the diagnosis of CAD. Routine laboratory investigations, including complete blood counts and liver and renal function tests, were within normal limits, and peripheral blood smear examination did not reveal any atypical cells. However, antinuclear antibody (ANA) testing returned positive, suggesting the presence of an autoimmune component. A skin biopsy from a lesion on the dorsum of the patient’s hand revealed histopathological features typical of CAD, including parakeratosis, irregular acanthosis, apoptotic keratinocytes, focal basal cell vacuolization, and a lymphocytic infiltrate in the papillary dermis. These histopathological findings, combined with the clinical presentation and positive photo testing results, established the diagnosis of CAD.

Diagnosis: Chronic Actinic Dermatitis (CAD) was diagnosed based on the patient's clinical presentation, phototesting revealing heightened sensitivity to UVA and UVB radiation, histopathological findings, and a positive antinuclear antibody. The diagnosis was further corroborated by the patient’s significant sun exposure due to her occupation and the characteristic involvement of sun-exposed skin areas.

Management: Initial management included topical corticosteroids (clobetasol propionate), topical tacrolimus, oral antihistamines, and broad-spectrum sunscreens to alleviate pruritus and inflammation.

However, the response was suboptimal, with symptom recurrence within two weeks. Given the lack of sustained response, the treatment regimen was modified over the following three months to incorporate oral corticosteroids (prednisolone) during acute flare-ups. Immunosuppressive therapy was escalated with methotrexate, azathioprine, and acitretin. Despite these interventions, the patient's symptoms progressively worsened, with the involvement of additional areas, including the forearms, trunk, and lower limbs. A more aggressive treatment approach was subsequently initiated with the use of oral tofacitinib, a Janus kinase (JAK) inhibitor, after ruling out contraindications such as hepatitis B and C, HIV, and tuberculosis, and confirming normal fasting lipid profiles. Within six weeks of initiating tofacitinib, the patient reported a significant reduction in pruritus, burning sensations, and lesion infiltration. After three months of continuous therapy, near-complete clearance of lesions was achieved, and pruritus was entirely resolved. Additionally, the Dermatology Life Quality Index (DLQI) demonstrated substantial improvement, reflecting a marked enhancement in the patient's overall quality of life.

Follow-up and Outcome: At the six-month follow-up, the patient maintained sustained remission with continued positive response to tofacitinib. The dose was reduced to 5 mg once daily after six months, with ongoing regular monitoring of complete blood counts, liver function tests, and lipid profiles. No adverse effects were noted during the follow-up period. The patient remained symptom-free, with no further progression or recurrence of disease.

Figure Ia: Eczematous rash on forearm

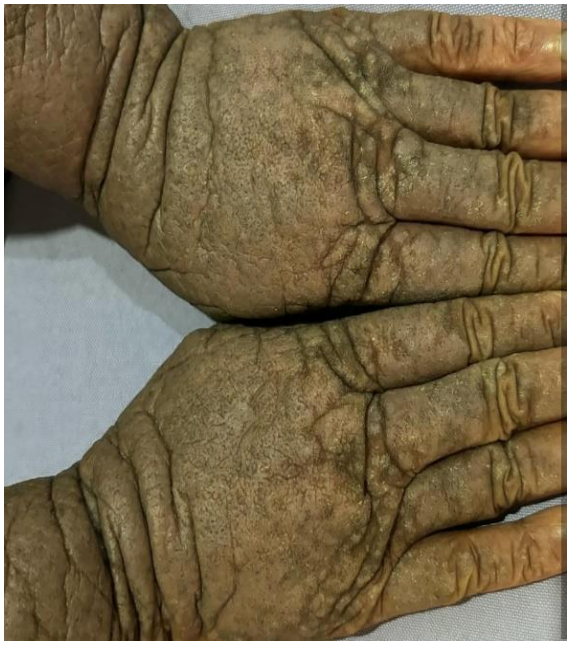

Figure Ib: Surface of patient's hands

Discussion

Chronic Actinic Dermatitis (CAD) represents a challenging photodermatosis with profound implications for patients exposed to significant ultraviolet (UV) radiation, whether occupationally or recreationally. The data presented in table 1, encompassing 15 cases reported by Chia-Yu Chu et.al 2014 [11] provides a detailed examination of CAD's clinical features, histopathological findings, and therapeutic responses. This analysis not only highlights the heterogeneity of CAD but also underscores the necessity for individualized treatment strategies tailored to patient-specific needs[12].

Clinical and Demographic Insights: The findings in this case series reaffirm CAD's predilection for males above the age of 50, a trend documented in earlier studies. Occupations necessitating prolonged outdoor activities, such as construction and farming, emerged as significant risk factors. This underscores the critical influence of chronic UV exposure in the etiology of CAD. Moreover, the seasonal exacerbation of symptoms during sunnier months further emphasizes the role of UV radiation in disease progression, underscoring the importance of robust photoprotective measures for at-risk populations[13].

Pathophysiological Insights: The pathogenesis of CAD involves intricate interactions between immune dysregulation and photosensitivity. The histopathological findings, including parakeratosis, apoptotic keratinocytes, and lymphocytic dermal infiltrates, align closely with features seen in allergic contact dermatitis (ACD) [14-15]. This overlap suggests shared mechanisms, including the activation of T-lymphocytes in response to photo-induced neoantigens. Cross-reactivity between exogenous allergens and endogenous antigens appears to sustain the inflammatory cycle, while heightened sensitivity to visible light in certain cases expands the range of potential triggers beyond UV radiation[16].

These findings call for more comprehensive investigations into non-UV light contributions to CAD's pathogenesis and the potential role of autoimmune processes. A deeper understanding of these mechanisms could refine diagnostic and therapeutic approaches, ensuring better disease control.

Therapeutic Perspectives: Photoprotection remains the cornerstone of CAD management. The consistent use of broad-spectrum sunscreens, protective clothing, and avoidance of photosensitizing agents is critical. High-potency topical corticosteroids, such as clobetasol, have demonstrated efficacy in reducing acute inflammation, while calcineurin inhibitors (e.g., tacrolimus and pimecrolimus) serve as valuable alternatives, particularly in sensitive regions like the face [17-18]. For patients with severe or refractory CAD, systemic immunosuppressants, including azathioprine and methotrexate, have shown promise, although their long-term use necessitates caution due to potential adverse effects. Recent advancements in understanding CAD's immunopathology have paved the way for targeted therapies. Janus kinase (JAK) inhibitors and biologics like dupilumab have emerged as promising options, offering effective disease control by modulating key inflammatory pathways. These therapies mark a significant shift toward personalized medicine in CAD management[19-21].

Challenges and Future Directions: CAD's chronic nature and its association with occupational UV exposure present ongoing challenges. Patch and photo patch testing play a pivotal role in identifying specific allergens and tailoring management strategies. Moreover, educating patients about the importance of consistent photoprotection and adherence to treatment regimens is fundamental to improving outcomes.[16-17] The insights from this study, as summarized in Table 1, emphasize the critical need for integrating emerging therapeutic modalities into routine clinical practice. Future research should focus on unravelling the molecular interplay between CAD and ACD, exploring the role of autoimmunity, and evaluating the long-term efficacy and safety of targeted treatments. By bridging these knowledge gaps, we can refine therapeutic strategies, ultimately improving the quality of life for patients with CAD.

This case series contributes significantly to the understanding of CAD and its management, offering a foundation for further studies and supporting the development of evidence-based clinical guidelines.

Table 1: Comprehensive Clinical and Pathological Profile of 15 Chronic Actinic Dermatitis Cases

No.	Age	Sex	Interval (Onset to Diagnosis)	Occupation	Histopathology	MED A	MED B	Treatment
1	51	M	4 years	Construction	NA	Y	Y	Clobetasol, Fluticasone, Betamethasone Valerate, Hydrocortisone, Pimecrolimus
2	28	M	2 years	NA	NA	e	Y	Desoximetasone
3	75	M	1 year	NA	NA	e	Y	Fluticasone
4	50	M	2 months	Postman	Superficial dermatitis	e	Y	Desoximetasone, Hydrocortisone
5	59	M	1 month	NA	NA	Y	Y	Clobetasol, Fluticasone, Hydrocortisone
6	59	M	3 years	Salesman	Chronic cheilitis	e	Y	Azathioprine (50 mg BID, tapered to QOD for >2 years)
7	71	M	10 years	NA	NA	Y	Y	Azathioprine (50 mg BID for 2 weeks), lost to follow-up
8	40	M	3 years	Clerk	NA	e	Y	Clobetasol

No.	Age	Sex	Interval (Onset to Diagnosis)	Occupation	Histopathology	MED A	MED B	Treatment
9	45	M	1 year	Construction	NA	Y	Y	Azathioprine (50 mg QD for 3 weeks, discontinued due to nausea/vomiting), Clobetasol
10	50	M	1 year	Salesman	NA	Y	Y	Clobetasol, Fluticasone
11	61	M	3 years	Carpenter	Hypersensitivity reaction	e	Y	Clobetasol, Desoximetasone, Hydrocortisone, Betamethasone Dipropionate
12	69	M	5 years	Farmer	NA	Y	Y	Azathioprine (50 mg QD for 2 weeks), lost to follow-up
13	74	M	10 years	NA	Chronic dermatitis	Y	e	Prednisolone (10 mg QD for 24 days, tapered to 10 mg QOD for 16 weeks), lost to follow-up
14	66	M	1 month	Businessman	NA	Y	Y	Clobetasol, Fluticasone, Hydrocortisone
15	82	M	1 year	NA	NA	Y	Y	Clobetasol, Desoximetasone, Fluticasone, Betamethasone Dipropionate

Conclusion

This case illustrates the complex interaction of environmental, immunological, and occupational factors in the development and progression of Chronic Actinic Dermatitis (CAD). The patient's clinical signs, diagnostic process, and response to a customized treatment plan highlight the importance of early detection, thorough assessment, and personalized management strategies for effectively managing CAD. The substantial improvement observed with tofacitinib suggests that targeted therapies hold promise in achieving lasting remission, especially in cases that are resistant to conventional treatments. Additionally, this case emphasizes the essential role of photoprotection and patient education in slowing disease progression and improving overall quality of life. Given the challenges and high morbidity associated with CAD, this report contributes to the increasing body of evidence supporting a collaborative, multidisciplinary approach to care. Ongoing research into innovative treatment options, autoimmune mechanisms, and tailored interventions will be key in advancing CAD management and optimizing outcomes.

List of Abbreviation
CAD: Chronic Actinic Dermatitis
ANA: Antinuclear Antibody
AZT: Azathioprine
BD: Betamethasone Dipropionate
BID: Twice a day
BV: Betamethasone Valerate
C: Clobetasol
D: Desoximetasone
DLQI: Dermatology Life Quality Index
F: Fluticasone
JAK: Janus Kinase
MED A: Minimal Erythema Dose to UVA
MED B: Minimal Erythema Dose to UVB
NA: Not Applicable
P: Pimecrolimus
QD: Each Day
QOD: Every Other Day
LFU: Loss of Follow-up
Tof: Tofacitinib (a Janus kinase inhibitor)
UV: Ultraviolet Radiation
UVA: Ultraviolet A
UVB: Ultraviolet B

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Conflict of Interest
The authors declare that there are no conflicts of interest pertaining to this case report.

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Contribution to authors:
All of the listed authors have reviewed and approved the manuscript.

Data Availability
Any questions regarding the availability of the study's supporti ng data should be addressed to the corresponding author, who can provide it upon justifiable request.

Ethics Approval and Consent to Participate
Written informed consent was obtained from the patient prior to the publication of their medical information and images. All personal identifiers have been carefully omitted to ensure the patient’s privacy and confidentiality are fully respected.

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