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Eosinophilic Annular Erythema Presenting as Chronic Polycyclic Plaques: A Clinicopathological Case Report

Case Report

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Abstract

Eosinophilic annular erythema (EAE) is a rare chronic figurate dermatosis characterized by recurrent annular plaques associated with tissue eosinophilia. Due to its clinical resemblance to granuloma annulare and other annular dermatoses, accurate diagnosis requires clinicopathological correlation. We report a case of a 60-year-old postmenopausal woman who presented with multiple erythematous annular plaques over the forehead, trunk, upper limbs, and buttocks for two years. The lesions began as papules and gradually enlarged centrifugally with central clearing, eventually coalescing into polycyclic plaques. Mild pruritus was present without systemic symptoms. Histopathological examination revealed an unremarkable epidermis with dense perivascular and interstitial inflammatory infiltrate composed predominantly of lymphocytes and eosinophils extending into the deep dermis and superficial fat. Flame figures were identified, while mucin deposition, collagen degeneration, and palisading granulomas were absent. The patient responded well to topical corticosteroids, systemic corticosteroids, and hydroxychloroquine. This case highlights the importance of recognizing EAE in the differential diagnosis of chronic annular dermatoses and underscores the role of histopathology in establishing the diagnosis.

Introduction

Figurate erythemas represent a heterogeneous group of dermatological conditions characterized by annular, polycyclic, or arciform lesions with centrifugal spread. Eosinophilic annular erythema (EAE) is a rare entity within this group and is often considered a chronic variant of Wells syndrome. Clinically, it closely mimics granuloma annulare (GA) and other annular dermatoses, making diagnosis challenging. Histopathological examination remains crucial for differentiation.[1]

Case Report

A 60-year-old postmenopausal woman presented with a two-year history of multiple circular erythematous lesions over the forehead, trunk, upper limbs, abdomen, and buttocks. The lesions had an insidious onset and gradually progressed in size and number. Initially appearing as small papules, they enlarged centrifugally with relative central clearing and eventually coalesced to form polycyclic plaques. The lesions were mildly pruritic and painless.

There was no history of fever, weight loss, systemic symptoms, drug intake, insect bites, or preceding infection. The patient had no history of atopy, tuberculosis, hypertension, or diabetes. She had received multiple courses of oral itraconazole without improvement.

On examination, multiple well-defined annular plaques ranging from 2 cm to 15 cm in diameter were noted, predominantly over the upper and mid-back with relative peripheral sparing (Figure 1-3). Some lesions showed raised active borders with occasional

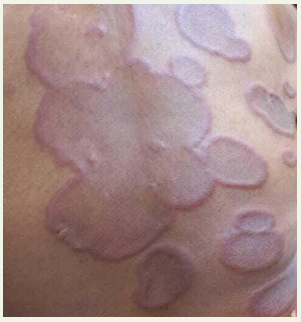

Figure 1: Multiple well-defined erythematous annular and polycyclic plaques over the upper back showing raised, active borders with relative central clearing and coalescence into larger plaques.

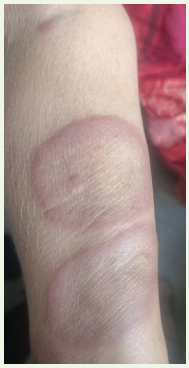

Figure 2: Annular plaque over the forearm demonstrating a smooth surface without scaling, with an elevated, well-demarcated border and central clearing.

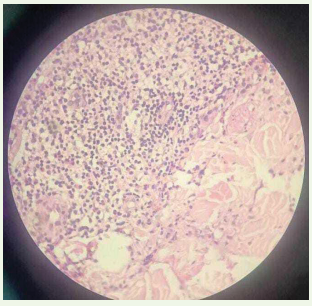

Figure 3: Clinical image showing multiple discrete and coalescing annular plaques over the trunk and extremities with polycyclic configuration and satellite lesions.

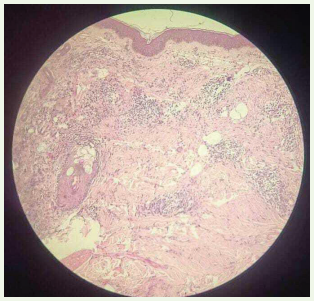

Figure 4: Histopathological examination (Hematoxylin and eosin, ×100) showing unremarkable epidermis with dense perivascular and interstitial inflammatory infiltrate in the dermis.

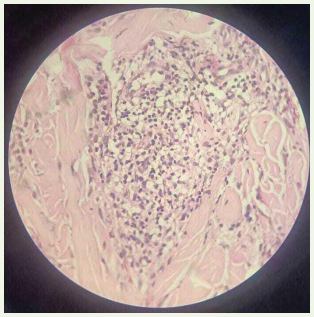

Figure 5: Histopathology (H&E, ×400) demonstrating eosinophil-rich infiltrate with flame figures characterized by deposition of eosinophilic granular material along collagen bundles.

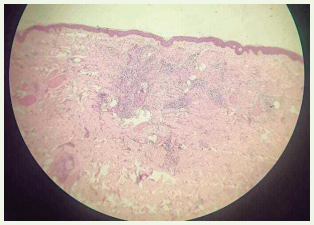

Figure 6: Deep dermal involvement with extension of inflammatory infiltrate into superficial fat lobules (H&E, ×200), without evidence of mucin deposition or granulomatous inflammation.

satellite papules. Scaling was absent. There was no lymphadenopathy, hepatosplenomegaly, sensory deficit, or nerve thickening.

Peripheral blood examination did not reveal eosinophilia, with absolute eosinophil counts within normal limits.

Histopathological examination (Figure 4–6) revealed an unremarkable epidermis. The dermis showed dense perivascular and interstitial inflammatory infiltrate composed predominantly of lymphocytes, eosinophils, and histiocytes extending into the deep dermis and superficial fat. Flame figures were present (Figure 5, 6), characterized by deposition of eosinophilic granule proteins on collagen bundles, appearing as intensely eosinophilic flame-shaped structures representing eosinophil degranulation. There was no mucin deposition, collagen degeneration, or palisading granulomatous inflammation.

Based on clinicopathological findings, a diagnosis of eosinophilic annular erythema was made.

The patient was treated with topical betamethasone dipropionate 0.05% cream applied twice daily for three weeks. Systemic therapy included oral prednisolone 40 mg daily for two weeks, followed by gradual tapering from 30 mg to 7.5 mg. Hydroxychloroquine 200 mg twice daily was administered for three months after baseline ophthalmologic evaluation. Antihistamines were prescribed for symptomatic relief. Significant improvement was observed within three weeks.

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Discussion

Eosinophilic annular erythema is a rare chronic dermatosis characterized by annular plaques with tissue eosinophilia. It is often considered part of the Wells syndrome spectrum, although it differs clinically by its chronic course and absence of acute inflammatory features.[2]

Granuloma annulare is an important differential diagnosis due to its similar clinical presentation. However, histopathology differentiates the two conditions. GA typically shows palisading granulomas, mucin deposition, and collagen degeneration, whereas EAE demonstrates eosinophil-rich infiltrate with flame figures and lacks mucin and granulomatous architecture. [3,4]

Eosinophils may be present in GA in a subset of cases, but they are usually sparse and not associated with flame figures. The presence of prominent eosinophilic infiltrate and flame figures strongly favors EAE.[4]

The pathogenesis of EAE is not fully understood but is thought to involve a hypersensitivity reaction with eosinophilic activation. Various treatments have been described, including corticosteroids, antimalarials, dapsone, and immunomodulators. Our patient responded well to systemic corticosteroids and hydroxychloroquine, consistent with previous reports. [5-7]

Conclusion

Eosinophilic annular erythema is an important differential diagnosis in chronic annular dermatoses. Histopathological evaluation is essential for accurate diagnosis and appropriate management.

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