BRIEF ARTICLE

# Eosinophilic Annular Erythema: An Elusive Masquerader in A Leprosy-Endemic Country

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## ABSTRACT

**Introduction:** Eosinophilic annular erythema (EAE) is a rare eosinophilic dermatosis, originally described in children, characterized by multiple annular, erythematous plaques with centrifugal growth pattern. Its manifestations closely resemble other figurate erythemas and can mimic conditions like leprosy. We present a case exploring the diagnostic complexities in an elderly woman first suspected of having leprosy.
**Case:** A 67-year-old female presented with a 2-month history of round, erythematous rash on the abdomen, neck, chest, and both lower limbs, initially considered to be leprosy. Physical examination revealed multiple annular erythematous plaques with hypoesthesia in some areas. Further tests did not reveal acid-fast bacilli or fungal elements. Total IgE levels were elevated. Serial skin biopsies showed mild spongiosis with predominantly eosinophilic infiltrates. Treatment with oral and topical corticosteroids resulted in clinical improvement.
**Conclusion:** The clinicopathological correlation plays a vital role in confirming the diagnosis in this case. EAE should be suspected in patients with nonspecific annular lesions and established based on eosinophilic dominance in histopathology.

## INTRODUCTION

Eosinophilic annular erythema (EAE) is a rare eosinophilic dermatosis characterized by recurrent, pruritic, erythematous annular plaques, predominantly affecting the trunk and proximal extremities.[1] EAE has been associated with various systemic conditions, including chronic kidney disease, diabetes mellitus, hepatitis C, autoimmune thyroid diseases, malignancies, and *Helicobacter pylori* infection.[2] The diagnostic complexities lie in its clinical resemblance to other figurate erythema lesions. This case report highlights the diagnostic challenges in an elderly woman initially suspected as leprosy. Leprosy can present as well-defined, erythematous plaques with an annular configuration, resembling other erythematous figurate lesion. Although leprosy is endemic in Indonesia, annular erythematous skin lesions should prompt consideration of other conditions, including EAE.

## CASE REPORT

A 67-year-old woman was referred to our center with a suspicion of leprosy for further evaluation. She presented with circular

erythematous rashes on her neck, trunk, and lower limbs that had persisted for two months. Physical examination revealed multiple annular erythematous plaques on the neck, chest, abdomen, and lower extremities (**Figure 1**). A few hypoesthetic lesions were observed on the lower limbs; however, no nerve enlargement was noted, and sensory and motor functions remained normal. Slit skin smears revealed no acid-fast bacilli, and direct potassium hydroxide examination demonstrated no fungal elements. Initially, borderline lepromatous leprosy was considered; however, the neurologic and microbiologic examinations were non-confirmatory, prompting a skin biopsy.

The initial skin biopsy from the neck and abdomen revealed non-specific spongiotic dermatitis with an abundance of eosinophils. We also conducted blood serum laboratory tests, which showed positive anti-nuclear antibodies (ANA) with a coarse speckled pattern (titer 1/320), increased total IgE level (4,313 IU/mL, reference range <100 IU/mL), and lactate dehydrogenase levels at the upper limit (210 U/L, reference range 125 – 220 U/L). Some lesions improved after receiving cetirizine and topical steroids; however, the patient continued to develop new, increasingly pruritic lesions, leading to another skin biopsy from the right thigh, which yielded similar findings to the previous biopsy (**Figure 2**). Based on clinicopathologic correlation, a diagnosis of EAE was made.

The patient was treated with methylprednisolone 8 mg three times daily and 0.1% mometasone furoate cream. After two weeks, no new lesions appeared, the existing lesions subsided (Figure 1C-D), and itching decreased, leading to a gradual tapering of the methylprednisolone dosage. The patient was then referred to the Internal Medicine Department for a systemic evaluation related to EAE. Ultrasound and contrast-enhanced thoracoabdominal computed tomography scans revealed multiple solid nodules in the right thyroid lobe, leading to a fine-needle aspiration biopsy (FNAB) showing atypia of undetermined significance. Unfortunately, the patient was lost to follow-up.

## DISCUSSION

The patient was referred to our center with multiple annular, partially hypoesthetic lesions that were initially presumed to be leprosy. However, sensory examinations are subjective and may yield false-positive results in various inflammatory dermatoses. In this case, objective assessment revealed no nerve thickening or impairment, and the absence of granulomas in the skin biopsy ruled out leprosy.[3] Many dermatoses can present as figurate erythema, necessitating careful clinical examination and skin biopsies to pinpoint the etiology. **Table 1** summarizes the differential diagnosis of figurate erythematous lesions.[3-8]

The lesions on the neck and left calf resemble erythema annulare centrifugum (EAC), but no trailing scale was found. The distinctive "coat-sleeve pattern" seen in typical EAC histopathology was not present in this case.[5] Serial skin biopsies consistently revealed mild spongiosis with a mix of inflammatory cells, predominantly eosinophils. Both EAE and urticarial vasculitis (UV) are classified as figurate erythema lesions dominated by eosinophils.[6] UV arises due to inflammation of small blood vessels in the skin, characterized by chronic urticarial lesions persisting for > 24 hours.[7] Histopathology examination found no leukocytoclastic vasculitis, thus ruling out a diagnosis of UV. The clinical presentation and pathology results favored the diagnosis of EAE.

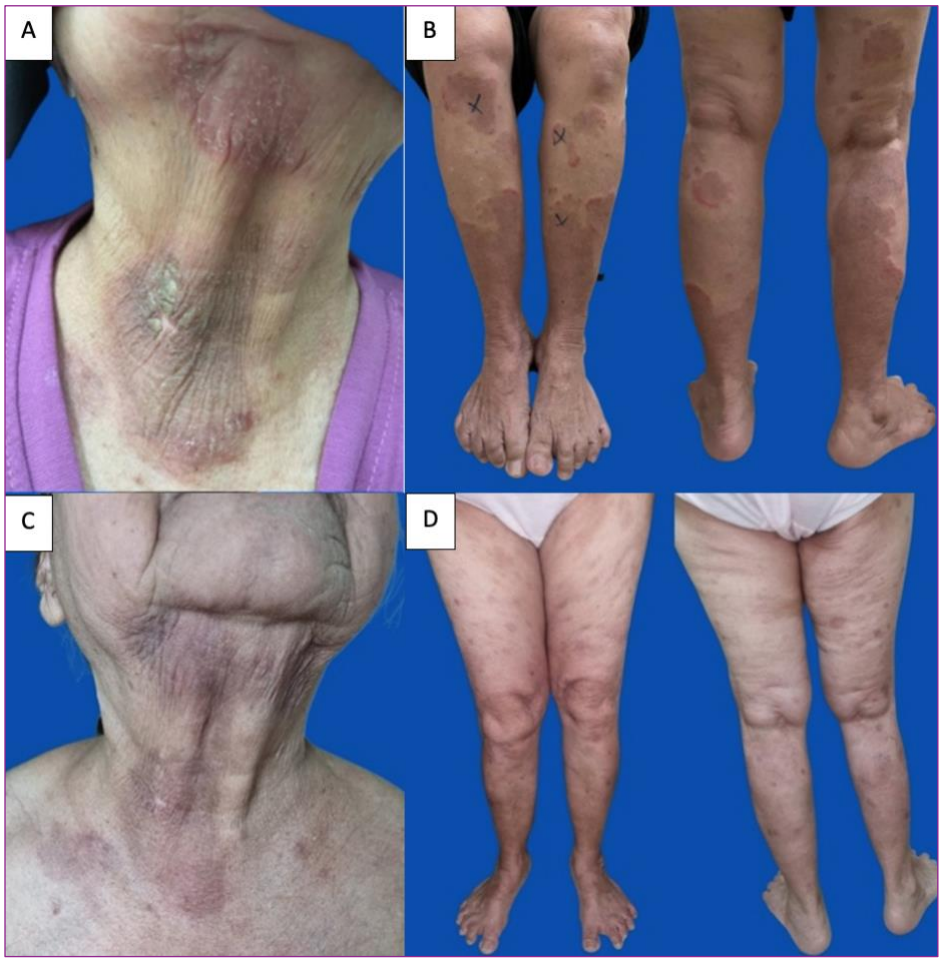


**Figure 1. (A-B)** Multiple annular erythematous plaques at the initial visit (the "X" in picture B denotes the lesion site tested for acid-fast bacilli) and **(C-D)** significant improvement after two-weeks of oral and topical corticosteroids.

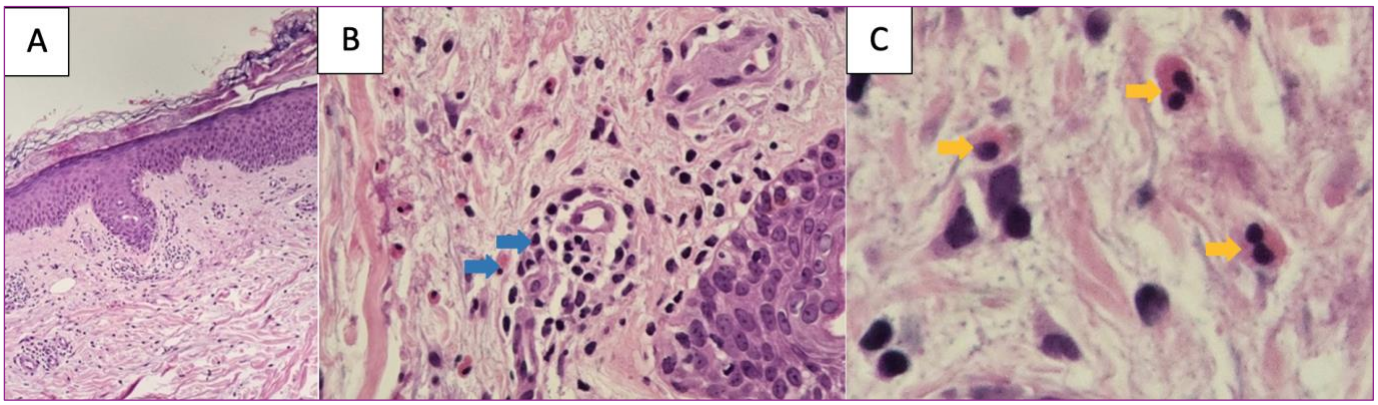


**Figure 2.** Histopathology demonstrates **(A)** mild spongiosis along with perivascular and interstitial mixed inflammatory infiltrate (HE, 100x), **(B)** mixed inflammatory infiltrate (blue arrow) perivascular (HE, 400x); and **(C)** eosinophil dominance (yellow) among the inflammatory cells (HE, 1000x).

**Table 1.** Differential Diagnoses of Figurate Erythematous Lesions

| Diagnosis | Clinical Presentation | Histopatological Findings |
|---|---|---|
| *Non-infection* | | |
| Eosinophilic annular erythema[1,7] | Multiple annular, erythematous plaques with centrifugal growth pattern. | Perivascular and interstitial mixed infiltrate with abundant eosinophils, without flame figures. |
| Erythema annulare centrifugum[3,4,5] | Annular patches, spreading centrifugally with central clearing, most commonly on the trunk and proximal extremities.<br>• Superficial EAC: trailing scale inside erythematous borders, pruritic.<br>• Deep EAC: "cord-like border" without scaling, non-pruritic. | • Superficial EAC: Spongiosis, parakeratosis, perivascular dermal lymphocytic infiltrates gathered in a "coat-sleeve pattern".<br>• Deep EAC: No epidermal changes, mononuclear infiltrate at the mid and lower dermis. |
| Granuloma annulare[3,4] | Annular plaques with indurated borders, non-scaly, asymptomatic – mild pruritus, typically on the extremities. | Lymphohistiocytic granuloma with connective tissue degeneration, mucin deposition, and palisaded or interstitial infiltrate |
| Plaque psoriasis[4] | Round or ovoid erythematous plaques, sharply demarcated, with silvery white scales. | Hyperkeratosis, acanthosis, uniform elongation of rete ridges, hypogranulosis, Munro microabscess, spongiform pustules of Kogoj, suprapapillary thinning. |
| Subacute cutaneous lupus erythematosus[3,4] | Annular or papulosquamous plaques, with or without scale, on sun-exposed areas. Predominantly in women. | Epidermal atrophy, vacuolar basal cell degeneration, interface dermatitis, mucin deposition, edema. |
| Urticarial vasculitis[6] | Indurated wheal (well-demarcated erythematous border and central pallor), lasting > 24 hours, with pain or burning sensation. | Leukocytoclastic vasculitis, inflammatory infiltrate perivascular: eosinophil (predominant), neutrophil, and/or lymphocytes. |
| Well's Syndrome[8] | Erythematous plaques with edema, evolving into annular or arched nodules, preceded by pruritus and burning sensation. | Eosinophil degranulation, flame figures, and granulomatous inflammation. |
| *Infection* | | |
| Tinea corporis[3] | Pruritic, annular, erythematous patch or plaque with central clearing, centrifugal growth, and polycyclic border. | Biosy is not routinely performed, but may show the presence of branching hyphae in the stratum corneum with periodic-Acid schiff or methenamine silver stains. |
| Tuberculoid leprosy[3] | Erythematous to hypopigmented, sharply demarcated macules or plaques, anesthesia/hypo-esthetic, | Epithelioid granulomas with abundant peripheral lymphocytes. |

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|---|---|---|
| | with or without scaling. May involve sensory/motor loss and palpable nerve. | |
| Secondary syphilis[4] | Erythematous macules or maculopapules or papulosquamous, white scaly ring on the surface (biett collarette), symmetric, involves the palms and soles. | Dermal inflammatory infiltrate with lymphocytes and plasma cells and a lichenoid lesion at the dermal-epidermal junction. |

EAE is often considered part of the Wells syndrome (WS) spectrum due to its similar histopathological features.[1] Clinically, WS manifest as erythematous plaques with edema evolving into annular or arched nodules preceded by itching and burning sensation.[8] The histopathological feature of "flame figures," characteristic of WS, and peripheral hyper-eosinophilia are rarely found in EAE, consistent with this case.[1,9]

The FNAB from solid nodules in the right thyroid lobe showed atypia of undetermined significance. Certain malignancies, such as renal clear cell carcinoma, metastasis of prostate adenocarcinoma, and thymoma, have been documented in EAE patients, yet there are no reports of thyroid malignancy in EAE cases. As the workup remains incomplete, other potential systemic causes are yet to be identified.

Clinical improvement was observed following a two-week treatment with oral and topical corticosteroids. Topical corticosteroids (betamethasone or clobetasol for 4–8 weeks) are the primary therapy for EAE due to their reasonably high cure rate, easy accessibility, and minimal side effects.[9] However, the relapse rate with corticosteroid therapy (both topical and oral) is quite high, making hydroxychloroquine and dapsone potential alternative therapies.[10,11]

## CONCLUSION

Clinicopathological correlation is crucial for establishing the diagnosis. EAE should be considered in patients with nonspecific annular lesions and confirmed histopathologically by the presence of predominant eosinophilic infiltrates. It is essential to assess various systemic conditions that may underlie EAE, as they can impact prognosis and management.

**Conflict of Interest Disclosures:** None

**Funding:** None

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