

Prolonged Treatment of Eosinophilic Erythema Annulare with Chloroquine

Dear Editor,

Eosinophilic annular erythema (EAE) is a rare figurate dermatitis of unknown etiology with prominent tissue eosinophilia.

A 59-year-old male patient presented with a one-month history of itchy, polycyclic, annular, and partially serpiginous lesions involving the back, the gluteal region, and the extremities (Figure 1, a, b). There was no medical history of drug intake. High potency local steroids and antihistamines were prescribed, but without adequate therapeutic results. Extensive laboratory work-up including serological infectious disease testing was performed and was within normal ranges. Histopathological examination of a biopsy taken from a lesion on the gluteus showed perivascular lymphocytic infiltrate around superficial and deep vascular plexus with admixture of eosinophils that was found interstitially (Figure 2, a,b) and within the lobules of subcutaneous fat. The overlying epidermis was unremarkable. There were no signs of flame figures and granulomatous inflammation. Based on the clinical and histopathological findings, a diagnosis of

EAE was established. The patient was given 40 mg of prednisone orally which resulted in partial improvement, but the lesions relapsed soon after the dose was tapered down to 20 mg. Chloroquine was started at a dose of 4 mg/kg daily for 10 days, then 250 mg daily for next the 10 weeks, resulting in complete clearance of all the lesions, which was sustained for over 2 years of follow-up.

It is still matter of debate whether EAE is a clinical subtype of Wells syndrome (WS) presenting with an annular or figurate pattern or is a distinct entity. In recently published paper, El-Khalawany et al. argued that EAE is a peculiar clinical variant of WS, because flames figures, blood and tissue eosinophilia, and granulomatous infiltrate can be observed in well-developed and long-standing lesions (1). The etiology of EAE is still unknown, although it has been suggested that it occurs as a result of a hypersensitivity reaction to an unidentified allergen (2). EAE has been associated with *Helicobacter pylori*, *Borrelia burgdorferi*, and hepatitis C virus infection, diabetes mellitus, chronic



Figure 1. (a) Polycyclic, annular and serpiginous lesions on the back and gluteal region. (b) Multiple annular erythematous lesions on the patient's trunk.

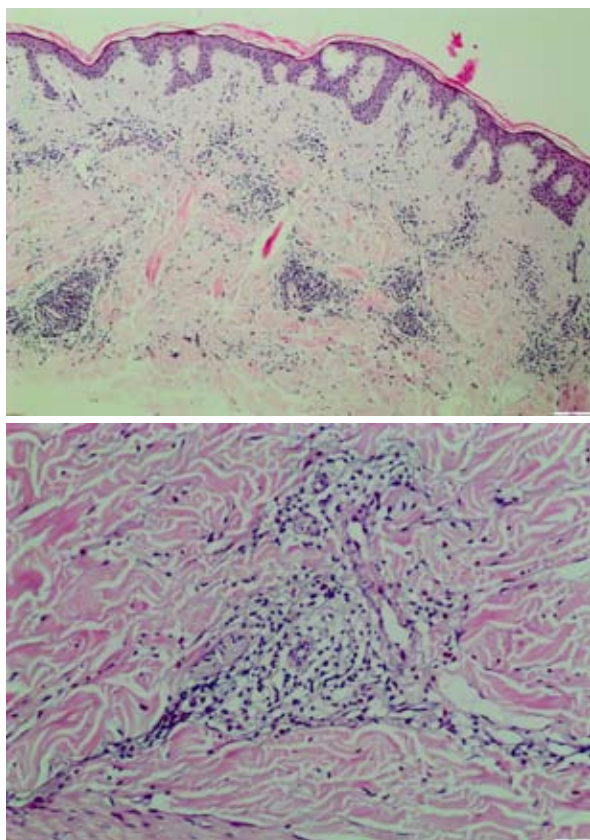


Figure 2. (a) Unremarkable epidermis. Superficial, mid-dermal dense infiltrate of lymphocytes around the vascular plexus. Extensive solar elastosis of the papillary dermis; hematoxylin and eosin $\times 10$. (b) Perivascular infiltrate of lymphocytes with admixture of eosinophils also found interstitially; hematoxylin and eosin $\times 40$ -

kidney disease, thymoma, autoimmune pancreatitis, autoimmune hypothyroidism, and internal malignancies (clear cell renal carcinoma, metastatic prostate adenocarcinoma) (3,4). Clinically, EAE is characterized by asymptomatic or mildly pruritic urticarial papules and plaques in annular configuration, mainly on the trunk and proximal extremities (5). Histologically, as in our patient, EAE is characterized by the appearance of a superficial and deep perivascular inflammatory infiltrate composed of lymphocytes and abundant eosinophils and absence of epidermal change (5). There is no standard treatment for EAE. Systemic steroids and antimalarials are the usual first-line options (5). Other treatment options include dapsone, indomethacin, cyclosporine, and UVB therapy (1,3,5). Response to antimalarials is usually observed within the first 2-4 weeks (2). However, as in our case, it may take several weeks for patients to respond to antimalarial treatment, and complete regression may even take longer (3).

We believe that EAE should be treated with antimalarials over a longer time period in order to avoid frequent relapses.

References:

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