This is a unique case of juvenile dermatomyositis presenting as an acquired Blaschko-linear dermatosis. The diagnosis was delayed because of the atypical presentation and only became apparent with worsening of the linear eruption as the patient developed the classic clinical manifestations of dermatomyositis. Other skin diseases that may present as an acquired Blaschko-linear dermatosis include psoriasis, lichen planus, lichen nitidus, lupus erythematosus, chronic graft-versus-host disease, drug eruptions, and atopic dermatitis.[1-3]

A case of linear calcification in a boy with dermatomyositis was recently described in which loss of heterozygosity was suggested as the pathogenetic mechanism.[4] However, Lipsker and Lenormand[5] point out that molecular evidence for loss of heterozygosity in polygenic inflammatory disease is missing. According to their categorization, our case would be classified as type 2b cutaneous mosaicism, wherein both Blaschko-linear and non-segmental distributions occur with varying skin manifestations and/or systemic involvement of the disease.[5] This unusual presentation of juvenile dermatomyositis further expands the number of diseases associated with an acquired inflammatory Blaschko-linear dermatosis.

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Eosinophilic annular erythema: An expression of the clinical and pathological polymorphism of Wells syndrome

To the Editor: Eosinophilic annular erythema (EAE) is a rare figurate dermatitis with a prominent tissue eosinophilia. The clinical features and the absence of "flame figures" make its relationship with Wells syndrome (WS) controversial.[1,2] We report 4 patients who shared clinical and histopathologic features of EAE. All were male adults, 35 to 85 years of age (mean age, 56 years), and had itching, figurate, polycyclic, or annular lesions on the trunk and extremities of 4 to 12 months' duration (Fig 1, *A* and *B*). Histopathologic examination revealed a dense lymphocytic infiltrate with abundant eosinophils without "flame figures" (Fig 2, *A*). Mucin deposition was seen in two biopsy specimens. Oral indomethacin, prednisone, or hydroxychloroquine were temporarily effective. In two patients, a second biopsy specimen disclosed typical "flame figures" surrounded by a granulomatous infiltrate (Fig 2, *B*). One patient died of a clear cell renal carcinoma.

To date, only 4 cases of EAE have been reported.[3] Clinical lesions featured two patterns: a figurate/centrifugum-type pattern reminiscent of erythema annulare centrifugum and an urticarial/annular-type pattern resembling granuloma annulare or erythema multiforme. Histologically, a dense superficial and deep perivascular lymphohistiocytic infiltrate with abundant eosinophils in the absence of flame figures is seen. Blood eosinophilia and mucin deposits were present in only one case.[2] Temporary clearance was obtained with oral chloroquine, indomethacin, or prednisone, but patients promptly relapsed after stopping treatment.

Substantially, EAE should be distinguished only from annular erythema of infancy (AEI) and WS.

AEI has been reported in only 6 infants.[4] Although morphologically and histologically alike, EAE is distinguished by the early age at onset, the spontaneous resolution, and the scarcer eosinophilic infiltration.

WS typically presents as a tender cellulitis-like plaque. Annular and figurate lesions similar to those of EAE, however, may occur in WS as well.[5] WS may be associated with arthropod bites, infections and infestations, drug intake, Churg-Strauss syndrome, and neoplasms. Laboratory tests reveal blood eosinophilia and increased eosinophil cation protein and interleukin 5 levels. Although not specific, the "flame figures" remain its distinctive histopathologic feature, caused by the deposition, in the acute stage, of the eosinophil major cationic protein onto collagen fibers.

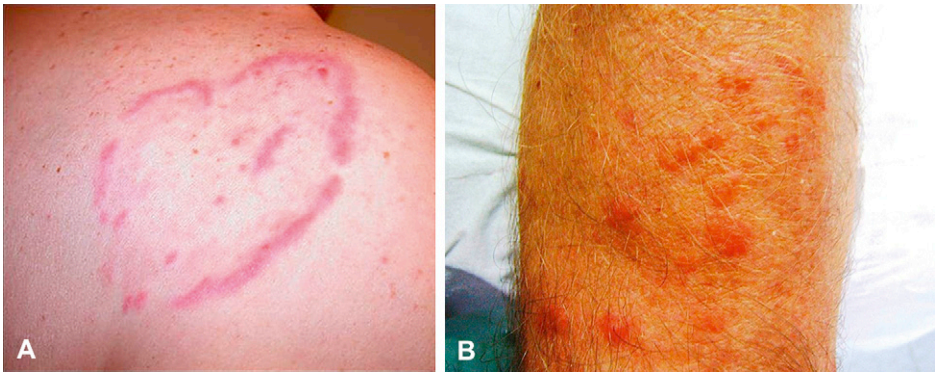

Fig.1. Patient 1. **A**, Annular polycyclic and gyrate lesion on trunk. **B**, Patient 2. Multiple, annular, indurate plaques, localized on upper arms.

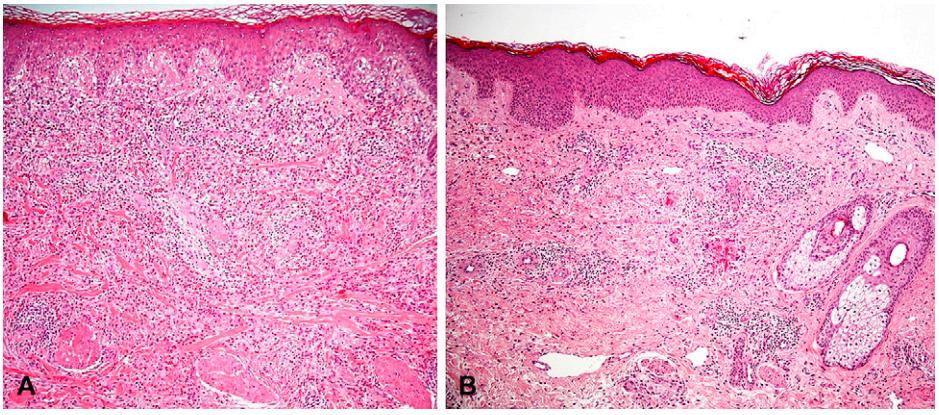

Fig.2. Patient 2. **A**. Dense, diffuse, dermal infiltrate made of numerous eosinophils, lymphocytes, and histiocytes. Neither flame figures nor granulomatous inflammation were seen. **B**, Second biopsy specimen shows a relatively dense perivascular and interstitial infiltrate rich in eosinophils in superficial and deep reticular dermis with "flame figures". (**A** and **B**, Hematoxylin-eosin stain; original magnification: ×40.).

To our knowledge, ours is the first finding of "flame figures" in EAE, seen after repeated biopsies. Kahofer, Grabmaier, and Aberer[1] took 6 skin biopsies during 9 years, and Howes, Girgis, and Kossard[2] took biopsies at the initial consultation and 2 years later without finding "flame figures."

As for clinical associations, an autoimmune thyroid disease and chronic borreliosis affected one of the previous cases of EAE, and one of our patients had a renal carcinoma.

Systemic steroids are considered the first line of treatment in idiopathic WS, while antimalarials have been suggested as the best treatment for EAE.In our cases, both oral steroids and hydroxychloroquine produced prompt clearing of lesions. However, both patients relapsed early after stopping treatment.

Although Weedon[6] has stated that "EAE may not be a variant of WS", we believe that EAE is a clinical subset of WS presenting with an annular or figurate pattern. The distinction between EAE and WS is arbitrary and may reflect the wide clinical polymorphism of WS and its histological dynamism.

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