International Journal of Dermatology

Clinicopathologic challenge

# Lower extremity reticulated hyperpigmentation with bullae

**Myra A. Sendelweck, ME, Ramin Fathi, MD, Nicholas V. Nguyen, MD, and Erin Fuller, MD**

University of Colorado Health, Aurora, CO, USA

**Correspondence**
Nicholas V. Nguyen, MD
Department of Dermatology
University of Colorado Denver
1665 Aurora Ct
Mail Stop F703
Aurora, CO 80045
USA
E-mail: nicholas.v.nguyen@ucdenver.edu

**Conflicts of interest**: None.

doi: 10.1111/ijd.13201

## History

A 37-year-old healthy Caucasian woman presented with hyperpigmented reticulated patches on the bilateral lower extremities of 1 year duration. The lesions originated as red–brown reticulated patches on her anterior shins, which progressively darkened and developed bullae, erosions, and crusting. The lesions were tender, non-pruritic, and not responsive to oral prednisone. Physical examination revealed red–brown to slightly violaceous reticulated and hyperpigmented patches on the knees and anterior legs with crusted papules and small bullae in areas of hyperpigmentation (Fig. 1). The patient had no family history of autoimmune disease. She lived in the foothills of Colorado without central heating and was accustomed to sitting with a space heater.

## Histopathological findings

Histological examination of sections taken from the right leg revealed a necrotic epidermis, and hemorrhage was noted within the stratum corneum. Within the dermis, there was perivascular inflammation consisting of lymphocytes as well as some histiocytic infiltration. Reactive angiomatosis was present, and atypical plump endothelial cells were seen with enlarged nuclei, nuclear hyperchromasia, irregular nuclear contours, and pleomorphism (Fig. 2).

## What is your diagnosis?

**Figure 1** Reticulated and hyperpigmented patches on the lower extremities. Pink 20 × 7 mm irregular blister on the right anterior knee

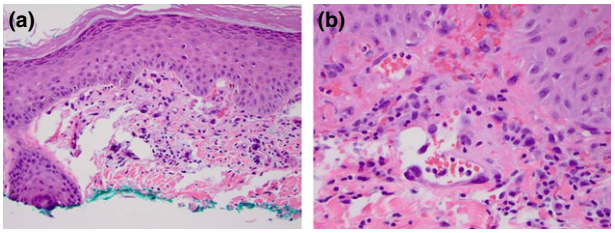


**Figure 2** Histopathology showing epidermal necrosis, slight dermal lymphocytic infiltration, and atypical endothelial cells. (a) Specimen taken from the right knee (hematoxylin–eosin, magnification, ×10) and (b) right lower leg (hematoxylin–eosin, magnification, ×40)

## Diagnosis

Bullous erythema ab igne (EAI).

## Discussion

EAI, Latin for redness from fire, is a disorder resulting from chronic environmental exposure to low levels of thermal radiation. Traditionally, this condition has been identified as an occupational hazard of bakers, metalworkers, and others exposed to a persistent heat source. EAI has become less common with the prevalence of central heating; however, resurgence has occurred with increased utilization of space heaters, laptop computers, heating pads, and heated car seats.[1] The condition typically manifests asymptomatically as transient blanchable macular erythema. Pruritus, mild burning, or pain may also be reported. Continual exposure can progress to dusky hyperpigmentation and fixed reticulated erythema with overlying epidermal atrophy.[2]

Histopathological findings of EAI commonly include necrosis of the epidermis. Mild dermal inflammation consisting of lymphocytes and histiocytes can be present. There have also been reports of secondary reactive angiomatosis within the papillary dermis causing atypical endothelial cells that are plump and exhibit hyperchromatic nuclei,[3] findings that were seen in the histological analysis of this patient. In the bullous variant, the blister cavity is generally subepidermal. Commonly also reported are telangiectasias within the dermal papillae in addition to an increase in elastic tissue, characteristics that were not noted in this case.

Treatment consists of discontinuing exposure to the heat source, which can resolve early cases over several months. More advanced cases may respond to 5-fluorouracil cream to help clear the atypical epithelial cells.[4] There have been promising results in improving the appearance of reticulated skin lesions with Q-switched Nd:YAG laser treatments.[5]

Chronic EAI lesions have been associated with an increased long-term risk of malignant transformation. Actinic keratosis, squamous cell carcinoma, cutaneous marginal zone lymphoma, and Merkel cell carcinoma have been reported arising at the site of longstanding EAI.[6] Early intervention is advised for resolution.

Bullous EAI is a rare variant of EAI, with very few reports in the literature. In this case presentation, the differential diagnosis initially included autoimmune bullous conditions such as bullous pemphigoid, systemic lupus erythematosus, as well as other acquired bullous dermatoses. Distinction was made by the clinical and histologic features described. This case highlights the importance of obtaining a thorough occupational and heat exposure history from patients presenting with bullous dermatoses.

## References

1 Huynh N, Sarma D, Huerter C. Erythema ab igne: a case report and review of the literature. *Cutis* 2011; 88: 290–292.
2 Miller K, Hunt R, Chu J, *et al.* Erythema ab igne. *Dermatol Online J* 2011; 17: 28.
3 Mitsuhashi T, Hirose T, Kuramochi A, *et al.* Cutaneous reactive angiomatosis occurring in erythema ab igne can cause atypia in endothelial cells: potential mimic of malignant vascular neoplasm. *Pathol Int* 2005; 55: 431–435.
4 Sahl WJ, Taira JW. Erythema ab igne: treatment with 5-fluorouracil cream. *J Am Acad Dermatol* 1992; 27: 109–110.
5 Cho S, Jung JY, Lee JH. Erythema ab igne successfully treated using 1,064-nm Q-switched neodymium-doped yttrium aluminum garnet laser with low fluence. *Dermatol Surg* 2011; 37: 551–553.
6 Arrington JH, Lockman DS. Thermal keratoses and squamous cell carcinoma in situ associated with erythema ab igne. *Arch Dermatol* 1979; 115: 1226–1228.