

Space Heater–Induced Bullous Erythema Ab Igne

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PRACTICE POINTS

- Consider erythema ab igne (EAI) as a potential differential diagnosis in bullous eruptions.
- Space heaters, heating pads, and even laptop computers should be considered as potential causes of EAI.

To the Editor:

Erythema ab igne (EAI) is a reticular erythematous hyperpigmentation of skin repeatedly exposed to moderate heat.¹ It usually is asymptomatic, though some patients report itching or burning at the site.² Historically caused by exposure to coal stoves or open fires, EAI has become increasingly common among individuals using space heaters, heating pads, or laptop computers near bare skin.^{2,3} Although EAI itself is benign and usually resolves with the removal of the exposure, it remains of clinical importance because of its association with underlying chronic disease, as chronic pain often is managed with frequent heating pad or hot water bottle use.² Additionally, accurate diagnosis is important given the future risk for malignancy, as chronic changes of EAI have been reported to lead to squamous cell carcinoma or rarely Merkel cell carcinoma.² Erythema ab igne is not traditionally associated with the formation of bullae; however, we present a case of bullous EAI that we believe highlights the importance of including this condition in the differential diagnosis of bullous disorders.

A 55-year-old man was admitted for presumed cellulitis of the bilateral legs. The patient had developed hyperpigmented discoloration of the medial surface of both legs with subsequent formation of tense bullae over the last 2 months. The dermatology department was consulted,

as there was concern for bullous pemphigoid. The patient's medical history was notable for hypertension, hyperlipidemia, diet-controlled type 2 diabetes mellitus, and hepatitis C virus with cirrhosis. The patient denied pruritus, pain, or known exposure of the legs to potential irritants prior to developing the lesions; however, with additional questioning he did report frequently sitting in front of a space heater with bare legs. Physical examination revealed multiple areas of reticulated erythematous hyperpigmentation with several overlying bullae (Figure 1). Many of the bullae were unroofed with full-thickness ulceration. Biopsies were taken for hematoxylin and eosin staining (Figure 2) and direct immunofluorescence.

Basic hematologic and metabolic laboratory test results as well as blood cultures were negative. Wound culture was positive for methicillin-resistant *Staphylococcus aureus*. Histologic examination showed interface dermatitis with subepidermal vesicle (Figure 2). Scattered necrotic keratinocytes were present in the adjacent epidermis, and focal subtle vacuolar alteration of the dermoepidermal junction was seen (Figure 3). Sparse perivascular mononuclear cells and scattered melanophages were present in the dermis. Direct immunofluorescence showed no diagnostic immunopathologic abnormality. Focal weak nonspecific vascular positivity for IgG and C3 was seen, but IgA and IgM were negative. Although not specific, these changes were compatible with EAI in the clinical context provided. The diagnosis of bullous EAI with superimposed staphylococcal infection was made.

Although rare, there have been reports of a bullous variant of EAI. Flanagan et al⁴ described 3 cases of bullous EAI with histopathology similar to our case. All 3 biopsies showed subepidermal separation with a mild perivascular dermal lymphocytic infiltrate. Direct immunofluorescence was negative in 2 cases but showed nonspecific weak

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The authors report no conflict of interest.

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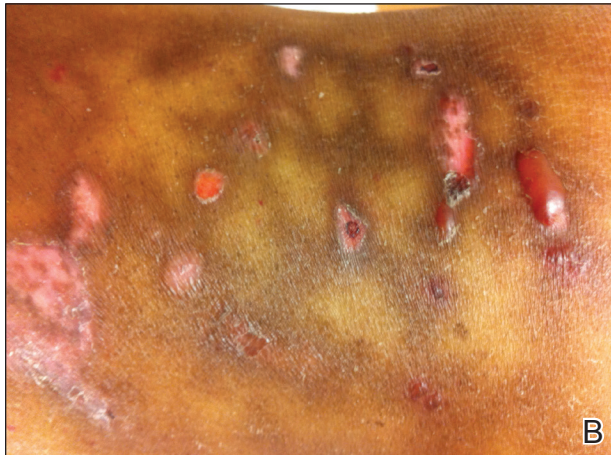


FIGURE 1. Bullous erythema ab igne. Bilateral legs (A) with multiple areas of reticulated erythematous hyperpigmentation with several overlying bullae (B).

patchy deposition of IgM along the dermoepidermal junction.⁴ Although our case was negative for IgM, there was a similar weak nonspecific distribution of IgG. Kokturk et al⁵ described a case of bullous EAI in a man with repeated exposure to a space heater. The lesions showed subepidermal separation of the epidermis; increased elastic fibers; dilated dermal capillaries; melanophages in the upper dermis; and a mild, superficial, perivascular-lymphocytic infiltrate. Direct immunofluorescence showed no immune deposits.⁵ Several earlier cases of bullae associated with EAI have been reported in the literature but were thought to be bullous lichen planus superimposed on EAI.⁶ Our case, which exhibited similar historical, physical, and histopathologic findings, strengthens the argument for a defined bullous variant of EAI.

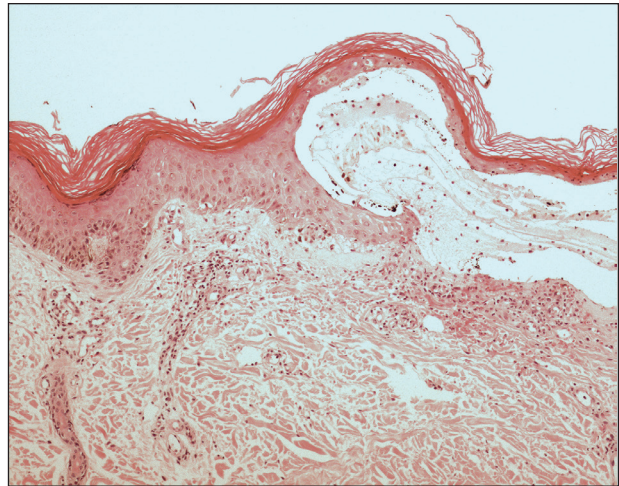


FIGURE 2. Bullous erythema ab igne. Interface dermatitis with subepidermal vesicle (H&E, original magnification $\times 100$).

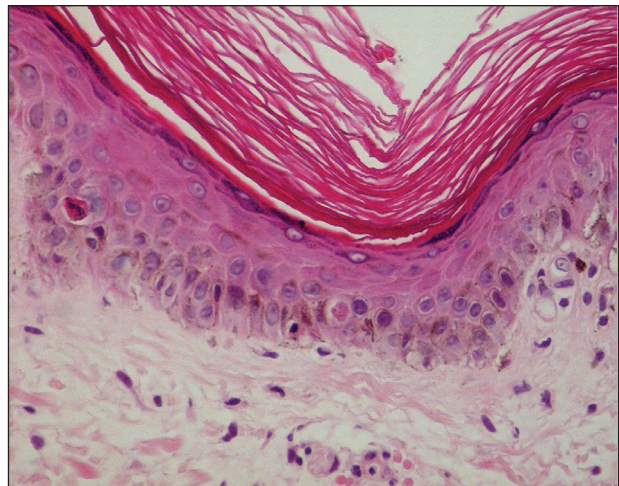


FIGURE 3. Bullous erythema ab igne. Scattered necrotic keratinocytes in the adjacent epidermis and focal subtle vacuolar alteration of the dermoepidermal junction (H&E, original magnification $\times 400$).

REFERENCES

1. Baruchin AM. Erythema ab igne—a neglected entity? *Burns*. 1994;20:460-462.
2. Arnold AW, Itin PH. Laptop computer–induced erythema ab igne in a child and review of the literature [published online October 4, 2010]. *Pediatrics*. 2010;126:E1227-E1230.
3. Tan S, Bertucci V. Erythema ab igne: an old condition new again. *CMAJ*. 2000;162:77-78.
4. Flanagan N, Watson R, Sweeney E, et al. Bullous erythema ab igne. *Br J Dermatol*. 1996;134:1159-1160.
5. Kokturk A, Kaya TI, Baz K, et al. Bullous erythema ab igne. *Dermatol Online J*. 2003;9:18.
6. Horio T, Imamura S. Bullous lichen planus developed on erythema ab igne. *J Dermatol*. 1986;13:203-207.