Erythema Ab Igne Following Heating/Cooling Blanket Use in the Intensive Care Unit

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Erythema ab igne caused by chronic heat exposure presents as a net-like hyperpigmentation of the skin. We report this condition rapidly evolving due to the inadvertent use or dysfunction of an adjustable-temperature blanket in the intensive care unit. The use of heating/cooling blankets, especially in a patient with altered mental status, can result in iatrogenic erythema ab igne.

Erythema ab igne (EAI) is a reticulated hyperpigmented dermatosis typically caused by long-term exposure to heat. Numerous sources of heat including peat, coal, gas, and electric fire, steam radiators, stoves, hot-water bottles, infrared lamps, heating pads, heated reclining chairs, and car heaters have produced EAI. Here we report the first case of EAI associated with intensive care unit (ICU) hospitalization.

Case Report
A 36-year-old African American man with a history of cerebrovascular accident, type I diabetes mellitus, hypertension, and chronic renal failure on hemodialysis, acutely developed aphasia and right-sided weakness. On admission he was hypertensive (blood pressure, 227/128) and hyperglycemic (blood glucose, 597). Brain imaging showed no new ischemic injury. Because the patient’s neurologic examination improved with blood pressure control, blood glucose control, and hemodialysis, his deficits were ascribed to a transient ischemic attack and/or a metabolic imbalance. Fever, delirium, and respiratory distress ensued, necessitating mechanical ventilation and transfer to the ICU on hospital day 1. A left lower lobe pneumonia was subsequently diagnosed and treated with antibiotic therapy. Mechanical ventilation was discontinued on hospital day 5, and he was transferred to step-down care. On hospital day 9, the patient was discharged home.

In the ICU the patient’s fevers were symptomatically treated with acetaminophen and a cooling blanket, the Gaymar Mul-T-Blanket Hyper/Hypothermia Blanket attached to a Blanketrol II heating and cooling source (Cincinnati Subzero Products, Inc.). On hospital day 3, the patient’s mother noted that the blanket was being used as a heating pad wrapped about her son’s torso.

One week after discharge, the patient noted a net-like discoloration on his trunk. He presented to dermatology clinic 2 months after discharge due to the persistence of the discoloration. Vibrant reticular hyperpigmentation was present on the patient’s back (Figure 1, A and B), abdomen, and chest with accentuation at the previous positions of 6-cm circular cardiac monitor adhesive pads (Figure 1C). The hyperpigmentation was apparent on Wood’s light examination, suggesting a superficial distribution of the pigment. A punch-biopsy specimen was obtained from the darkest skin on the lumbar back. Examination of hematoxylin-eosin-stained sections showed basal layer hyperpigmentation (Figure 2). On repeated questioning, the patient denied any pain in the affected areas or any source of heat exposure at home or at work other than the heating pad in the ICU. Eight months after discharge, the reticular pattern was still present, though less vivid.

Discussion
We report the induction of EAI by an adjustable heating/cooling blanket used on an intubated patient during a 5-day ICU stay. The temporal appearance of the rash, approximately 2 weeks after heat exposure, is similar to the most rapidly induced cases of EAI in
Although EAI appears after a minimum of 2 weeks, this case suggests that the source of heat exposure may be transient, and may occur with only one heating episode. Our patient’s altered mental state undoubtedly hindered his ability to moderate the use of the heating blanket and to express discomfort to his caregivers. His mental state may have also abrogated the unpleasant thermal sensations caused by higher temperatures. Unfortunately, our patient cannot remember his stay in the ICU, and the blanket’s exact temperature and duration of use are not documented.

Further support for the rash’s induction in the ICU comes from the enhanced circular patches of EAI on the patient’s chest. These correspond to cardiac monitor adhesive pads in both position and size, suggesting that the pads blocked evaporative cooling of the skin, which in turn aggravated the thermal insult.

The histopathology of EAI displays a wide spectrum of morphology. Increasing ultraviolet light exposure produces increasingly aberrant histology, such as elastosis; likewise, increasing thermal exposure produces increasingly aberrant histology including hyperkeratosis, flattening of the dermo-epithelial junction, dyskeratotic keratinocytes, basal cell degeneration, telangiectatic blood vessels, hemosiderin deposits, prominent melanophages, dermal thinning, course collagen, and dense elastic fibers in the mid- and lower dermis. Squamous cell cancer frequently arises in skin chronically exposed to thermal damage, and multiple case reports corroborate that thermal keratoses in EAI, like solar keratoses, may progress to squamous cell carcinoma. The
lack of major histologic disruption in our patient's biopsy supports his brief heat exposure history and serves as a good prognostic sign.

EAI has frequently been a consequence of patients' warming themselves in cold weather or treating themselves for internal pain with heating pads, as in the case of chronic pancreatitis or back pain. This is the first reported case of iatrogenic EAI. This patient's longstanding discoloration underscores the importance of carefully monitoring heating/cooling blanket use in the ICU.

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REFERENCES