The Molting Man: Anasarca-Induced Full-Body Desquamation

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PRACTICE **POINTS**

- The appearance of anasarca-induced desquamation can be similar to staphylococcal scalded skin syndrome and Stevens-Johnson syndrome.
- Histopathologic evaluation of this condition shows desquamation localized to the stratum corneum without epidermal necrosis.
- Careful evaluation, including bacterial culture, is required to rule out an infectious cause.
- Early diagnosis of anasarca-induced desquamation reduces the potential for providing harmful empiric treatment, such as systemic steroids and intravenous antibiotics, especially in patients known to have comorbidities.

Blisters and subsequent desquamation of the skin in the presence of acute edema is a well-known clinical phenomenon. In this case report, we describe a new variant that we have termed *anasarca-induced desquamation* in a 50-year-old man with molting of the entire cutaneous surface after acute edema, in a setting of 40-lb weight gain over 5 days. Laboratory workup for infectious causes and punch biopsies of skin lesions ruled out Stevens-Johnson syndrome and staphylococcal scalded skin syndrome, which have a similar clinical presentation to anasarca-induced desquamation. In patients with diffuse superficial desquamation in the setting of acute edema, anasarca-induced desquamation is worth investigating to avoid the use of corticosteroids and intravenous antibiotics in this inherently benign condition.

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Edema blisters are a common but often underreported entity most commonly seen on the lower extremities in the setting of acute edema.[1] Reported risk factors and associations include chronic venous insufficiency, congestive heart failure, hereditary angioedema, and medications (eg, amlodipine).[1,2] We report a newly described variant that we have termed *anasarca-induced desquamation* in which a patient sloughed the entire cutaneous surface of the body after gaining almost 40 pounds over 5 days.

Case Report

A 50-year-old man without a home was found minimally responsive in a yard. His core body temperature was 25.5 °C. He was profoundly acidotic (pH, <6.733 [reference range, 7.35–7.45]; lactic acid, 20.5 mmol/L [reference range, 0.5–2.2 mmol/L]) at admission. His medical history was notable for diabetes mellitus, hypertension, alcohol abuse, and pulmonary embolism. The patient was resuscitated with rewarming and intravenous fluids in the setting of acute renal insufficiency. By day 5 of the hospital stay, he had a net positive intake of 21.8 L and an 18-kg (39.7-lb) weight gain.

Dermatology was consulted for skin sloughing. Physical examination revealed nonpainful desquamation of the vermilion lip, periorbital skin, right shoulder, and hips without notable mucosal changes. Two 4-mm punch biopsies of the shoulder revealed an intracorneal split with desquamation of the stratum corneum and a mild dermal lymphocytic infiltrate, consistent with exfoliation secondary

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to edema or staphylococcal scalded skin syndrome (Figure 1). No staphylococcal growth was noted on blood, urine, nasal, wound, and ocular cultures throughout the hospital stay.

As the patient's anasarca improved with diuretics and continuous renal replacement therapy, the entire cutaneous surface—head to toe—underwent desquamation, including the palms and soles. He was managed with supportive skin care. The anasarca healed completely with residual hypopigmentation (Figures 2 and 3).

Comment

Anasarca-induced desquamation represents a more diffuse form of a known entity: edema blisters. Occurring most commonly in the setting of acute exacerbation of chronic venous insufficiency, edema blisters can mimic other vesiculobullous conditions, such as bullous pemphigoid and herpes zoster.[3]

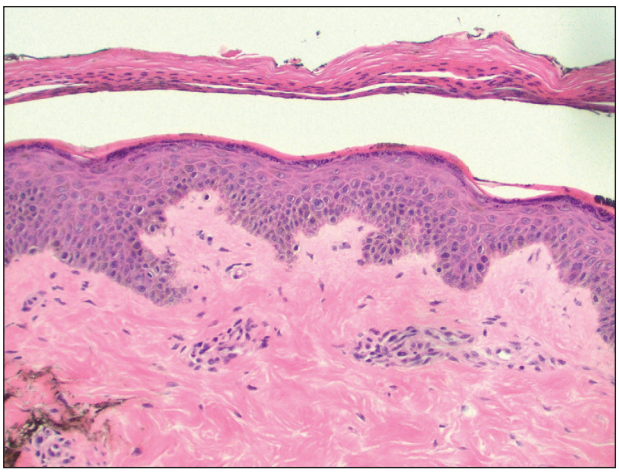

FIGURE 1. A punch biopsy of the right shoulder revealed an intracorneal split with desquamation of the stratum corneum and a mild dermal lymphocytic infiltrate (H&E, original magnification ×20).

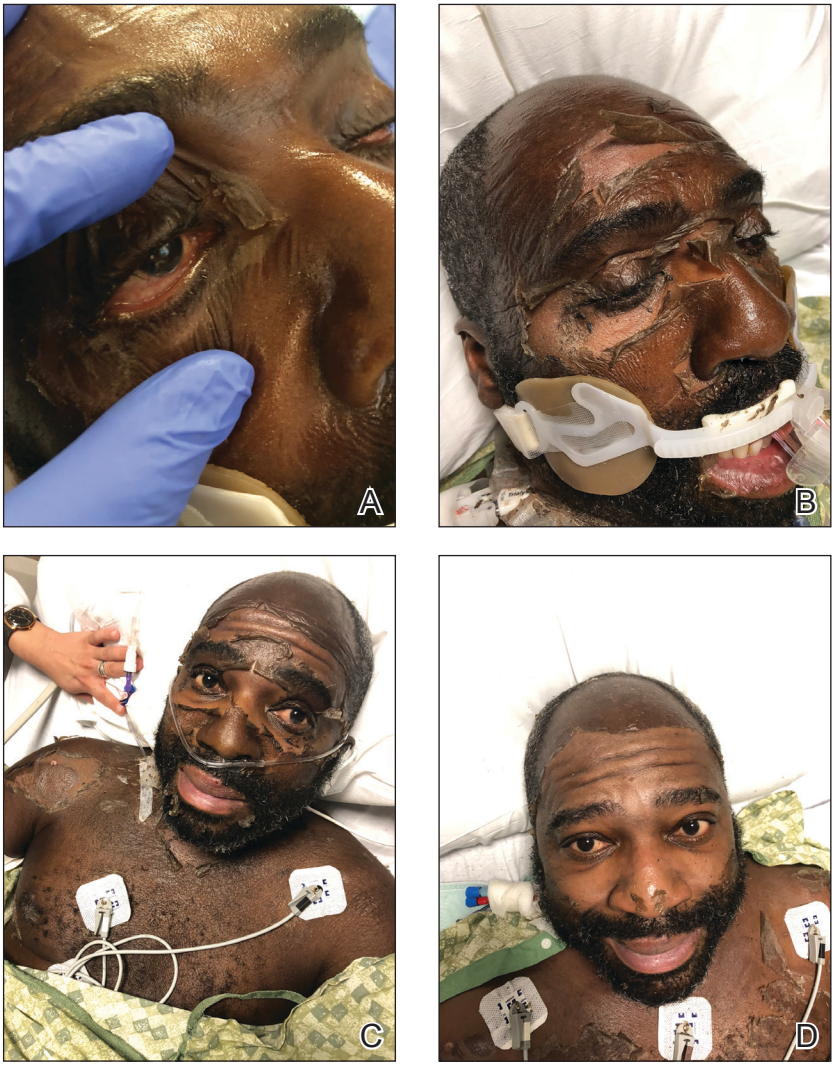

FIGURE 2. Progression of facial desquamation. A, Day 5 of hospital stay. B, Day 6. C, Day 7. D, Day 10.

FIGURE 3. Desquamation of the right palmar surface on day 10 of the hospital stay.

Pathogenesis of Edema Blisters—Edema develops in the skin when the capillary filtration rate, determined by the hydrostatic and oncotic pressures of the capillaries and interstitium, exceeds venous and lymphatic drainage. The appearance of edema blisters in the acute setting likely is related to the speed at which edema develops in skin.[1] Although edema blisters often are described as tense, there is a paucity of histologic data at the anatomical level of split in the skin.

In our patient, desquamation was within the stratum corneum and likely multifactorial. His weight gain of nearly 40 lb, the result of intravenous instillation of fluids and low urine output, was undeniably a contributing factor. The anasarca was aggravated by hypoalbuminemia (2.1 g/dL) in the setting of known liver disease. Other possible contributing factors were hypotension, which required vasopressor therapy that led to hypoperfusion of the skin, and treatment of hypothermia, with resulting reactive vasodilation and capillary leak.

Management—Treatment of acute edema blisters is focused on the underlying cause of the edema. In a study of 13 patients with edema blisters, all had blisters on the legs that resolved with treatment, such as diuretics or compression therapy.[1]

Anasarca-induced desquamation is an inherently benign condition that mimics potentially fatal disorders, such as Stevens-Johnson syndrome, staphylococcal scalded skin syndrome, and toxic shock syndrome. Therefore, patients presenting with diffuse superficial desquamation should be assessed for the mucosal changes of Stevens-Johnson syndrome and a history of acute edema in the affected areas to avoid potentially harmful empiric treatments, such as corticosteroids and intravenous antibiotics.

Conclusion

Anasarca-induced desquamation represents a more diffuse form of edema blisters. This desquamation can mimic a potentially fatal rash, such as Stevens-Johnson syndrome and staphylococcal scalded skin syndrome.

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