Acute inflammatory edema: A swell concept

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Cellulitis is costly—to the patient and the health care system. Annually, 650,000 hospital admissions costing $3.7 billion are attributed to cellulitis. Cellulitis mimickers (pseudocellulitis), if not recognized, can lead to increased morbidity, inappropriate admissions for antibiotics, and wasted medical resources.

Diagnosing cellulitis is based on the clinical presentation of expanding, ill-defined erythema, warmth, tenderness, and edema.[1] The ALT-70 predictive model (asymmetry, leukocytosis, tachycardia, and age ≥70 years) is a prospectively validated tool that assists in differentiating cellulitis from pseudocellulitis at the time of presentation and at least up to 48 hours later.[2] Radiologic imaging and blood cultures provide scant clinical value for evaluating and treating patients with uncomplicated cellulitis; these studies should only be obtained for those patients who are severely immunocompromised or displaying systemic toxic effects.[3]

Examples of pseudocellulitis include acute lipodermatosclerosis, panniculidities (eg, erythema nodosum), benign pigmented purpuric eruptions, and superficial migratory thrombophlebitis. Drugs, especially gemcitabine, might cause pseudocellulitis.[4] Dermatology consultations likely enhance patient outcomes by improving diagnostic accuracy and facilitating antibiotic stewardship in hospitalized patients with suspected cellulitis. In a study of 175 randomized patients with a presumptive diagnosis of cellulitis, patients were assigned to the control group (the primary medicine team) or the intervention group (with dermatology consultation). At 2 weeks, clinical improvement was significantly higher for those in the intervention group, a reflection of them recognizing pseudocellulitis in 30.7% of patients.[1]

In this issue of the *Journal of the American Academy of Dermatology*, Marchionne et al describe acute inflammatory edema (AIE), a variant of pseudocellulitis.[5] The authors reviewed 15 patients with this diagnosis (9 female and 6 male patients 52-73 years of age). Initial dermatology consults were requested for suspected cellulitis (5 patients), new rash of unknown etiology (9 patients), and an inflamed cyst (1 patient). There was a predilection for patients with high body mass indexes (13 of 15 patients), and those with evidence of fluid overload. AIE presented as bilateral, blanching, erythematous, and edematous plaques, most commonly involving dependent sites, such as the thighs and lower abdomen. Sparing areas of increased pressure on the skin (skin folds or where there was pressure from external objects contacting the skin, such as catheters) was characteristic. Generalized edema or anasarca was often present. Peau d'orange, pseudovesiculation, and edema bulla were observed in a minority of patients. Skin biopsies demonstrated marked papillary edema in all cases; the accompanying infiltrate consisted of neutrophils, lymphocytes, and histiocytes with bubbly cytoplasm (so-called edemaphages). All stains and microbe cultures were negative. Treatment focused on decreasing edema (improving fluid balance, diuretics, dialysis, compression, and repositioning). Antibiotics administered for presumed cellulitis were discontinued. A 3-hit pathogenesis of AIE was proposed: 1) acute volume overload, 2) impaired lymphatic return, and 3) tissue edema leading to connective tissue microtears with resultant inflammation.

I have seen such cases and have been flummoxed by trying to render a precise diagnosis. I commend Marchionne et al in recognizing this entity and providing a precise appellation[5]; diagnosing AIE

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should obviate costly evaluations and unfounded antibiotic therapy.

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