Coma Bullae: Associations Beyond Medications

JOHN PIEDE, MD, AND ERIC WALLACE, DO
Department of Internal Medicine, Wake Forest University, Winston-Salem, NC

A 49-year-old man with a history of anterior scleritis who was receiving maintenance azathioprine therapy presented with ecchymoses and bullous lesions that had developed 2 weeks previously on the bilateral lower extremities. The patient reported that the lesions were intially small blisters that had progressed to large painful hemorrhagic bullae. He had no history of intravenous drug use, high-risk sexual behavior, or tobacco abuse. Findings on physical examination were notable for anterior scleritis, cyanotic fingers, and bilateral 2+ lower extremity edema with numerous well-demarcated bullous lesions that had a hemorrhagic fluid-filled appearance. A complete blood cell count and metabolic profile were normal; urinalysis revealed trace hematuria. Skin biopsy demonstrated vascular thrombi with sweat gland necrosis consistent with coma bullae. Computed tomography of the chest revealed scattered, well-circumscribed pulmonary nodules. Serologic testing confirmed a positive cytoplastic antineutrophilic cytoplasmic antibody titer of 1:160 with anti-proteinase 3 antibodies. Renal biopsy was consistent with Wegener granulomatosis, and treatment was initiated with intravenous methylprednisolone and cyclophosphamide. At 4-month follow-up, the patient's renal function was normal, and he had no further complications.

Coma bullae are commonly associated with use of opiates, tricyclic antidepressants, and antipsychotics. They have also been observed in patients with chronic renal failure, hypercalcemia, diabetic ketoacidosis, and a variety of neurologic conditions. The skin lesions usually resolve in 2 to 4 weeks, but early recognition and treatment of the underlying condition are crucial. Etiology of these pathogenic lesions is broad, should be based on the clinical presentation, and should include autoimmune disorders such as Wegener granulomatosis.