Coma Blisters Sans Coma

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Coma blisters (CBs) are self-limited lesions that occur in regions of pressure during unconscious states classically induced by barbiturates. We report a case of CBs sans coma that were histologically confirmed in a 41-year-old woman who developed multiple tense abdominal bullae with surrounding erythema following a transatlantic flight. Interestingly, the patient was fully conscious and denied medication use or history of medical conditions. A clinical diagnosis of CBs was confirmed by histopathologic findings of eccrine gland necrosis, a hallmark of these bullous lesions.

Cutis. 2012;90:137-139.

Case Report

A 41-year-old woman presented to the emergency department with a painful rash on her lower abdomen. She denied pruritus, bleeding, or weeping from the lesions. She had been on a transatlantic flight and returned the previous day. She reported that she wore extremely tight-fitting jeans that pressed into her lower abdomen and remained sedentary for the majority of the flight. Her medical history included abdominoplasty performed 3 years prior. She denied medication use. A review of systems did not reveal any abnormalities. Physical examination revealed a large, horizontal, well-healed scar on her lower abdomen with a bright pink figurate patch with tense bullae above the scar. The patient was given fluocinonide ointment 0.05% and was advised to apply antibiotic ointment to the areas of blister rupture. She returned to the dermatology department 4 days after the initiation of therapy for follow-up with marked improvement of the erythema and bullae, which appeared flaccid with evidence of crusting (Figure 1). Histopathologic examination revealed epidermal necrosis, prominent dermal blood vessels, and eccrine gland necrosis (Figure 2). Based on the patient's history, clinical features, and histologic examination, coma blisters (CBs) were diagnosed.

Comment

Coma blisters are benign self-limited lesions. They commonly occur on areas of the skin that are exposed to unchecked pressure during unconscious states in which individuals do not move to avoid the associated pain. Coma blisters also have been reported to occur in noncomatose patients following medication overdose or diabetic ketoacidosis.[1,2] Coma blisters typically occur within 24 hours after a state of unconsciousness and immobilization.[3] Lesions initially present as blanchable patches with erythema and subsequently develop into violaceous plaques that form into tense blisters within 2 to 3 days. The blisters will spontaneously resolve within 1 to 4 weeks.[4] Treatment primarily is aimed to prevent secondary skin infection with the use of topical antibiotics.

The etiology and pathogenesis of CBs is complex and is not well-understood. Intoxication with

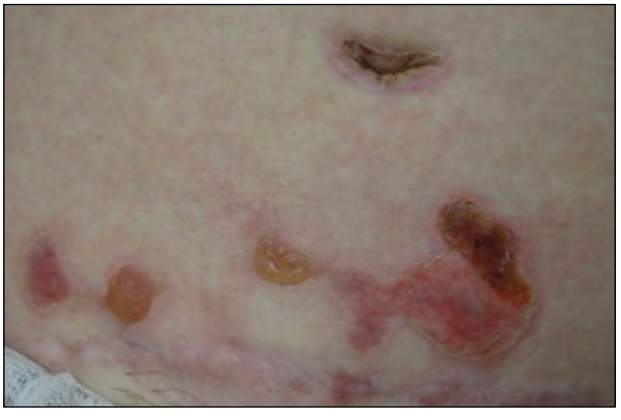

Figure 1. Non–drug-induced coma blisters. Marked improvement of erythema and bullae after 4 days of treatment with fluocinonide ointment 0.05% and antibiotic ointment.

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The authors report no conflict of interest.

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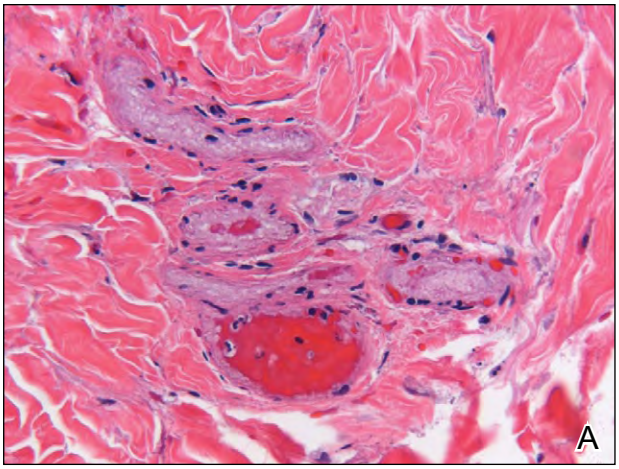

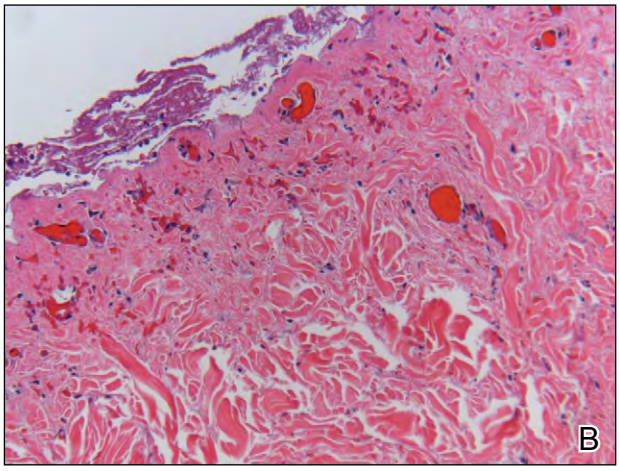

Figure 2. Histopathology of non–drug-induced coma blisters. Punch biopsy revealed eccrine gland necrosis, a characteristic biopsy finding of coma blisters (A) (H&E, original magnification ×40), as well as epidermal necrosis with prominent dermal blood vessels (B)(H&E, original magnification ×20).

Differential Diagnosis of Our Patient's Lesions[a]

Bullous pemphigoid

Coma blisters

Epidermolysis bullosa acquisita

Epidermolysis bullosa simplex

Erythema multiforme bullosum

Weber-Cockayne syndrome

[a]All of the these diagnoses, including the entire range of subepidermal bullous lesions such as bullous lupus erythematosus, were ruled out by clinical presentation or more specifically by biopsy in our patient.

sedative and hypnotic drugs, particularly barbiturates and benzodiazepines,[5] as well as carbon monoxide[6] can produce the unconscious state most commonly associated with CB formation. Coma blisters rarely have been observed in noncomatose patients secondary to medication overdose[1] or diabetic ketoacidosis,[2] indicating a potential role of drugs or metabolic dysregulation in the pathogenesis.

Wenzel and Horn[7] reported eccrine sweat gland drug excretion as a contributing factor to gland necrosis. Biopsies of drug-induced CBs support this mechanism, including findings of necrosis of eccrine sweat ducts, hair follicles, sebaceous glands, and the epidermis.[8] Biopsies of non–drug-induced CBs may resemble drug-induced lesions but may possibly be differentiated by the presence of fibrinoid thrombi in non–drug-induced specimens.[9] Other conditions to consider in the differential diagnosis of CBs are presented in the Table.

Our patient presented with biopsy-confirmed CBs with no history of coma, medication usage, or medical complications. The lesions likely resulted from a combination of prolonged immobilization, abdominal pressure, and a reduction in lymphatic drainage. It is possible that a disruption in lymphatics from the abdominoplasty contributed to the rapid onset of bullae formation in our patient.

Extensive cutaneous pressure during immobilized or semi-immobilized states may induce CBs without coma or medication intoxication. Frequent repositioning or mobilization of a patient is recommended to avoid the formation of these blisters.

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