



**Figure 3** Dermoscopy (original magnification  $\times 15$ ) performed with DermLite Pro (3Gen, LLC, San Juan Capistrano, CA, USA) coupled to a Sony DSC-T200 camera of 8.1 MP: central fibrous tissue and peripheral red blood cells. In the center, a long, yellow structure formed by 2 finger-like projections corresponds to the caudal segment with the posterior or respiratory spiracles of a *Dermatobia hominis* larva.

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## Chemical Burn from an Airbag

### Quemadura química por airbag

*To the Editor:*

Airbags are a safety mechanism proven to be effective in reducing the severity and fatality of traffic accidents.<sup>1</sup> Nevertheless, airbag deployment has induced multiple

injuries; superficial abrasions, contusions, lacerations, and thermal and chemical burns are the most common.<sup>2-4</sup>

A 21-year-old man was seen by the dermatologist on duty for facial burns and eye injury secondary to the sudden deployment and rapid inflation of the airbag in the vehicle he was driving. The injuries consisted of a well-delimited plaque on the forehead, with a very superficial, erosive appearance, sparing the deeper parts of the forehead



**Figure** A, Front physical abrasion secondary to airbag trauma associated with chemical and thermal burns on the eyelid, cheek, and lateral aspect of the nose caused by the mixture of high-temperature gases from the interior of the airbag. B, Palpebral edema, conjunctival chemosis, and corneal erosions secondary to the alkali chemical burn (alkali keratitis). C, Marked clinical improvement after 10 days of antibiotic and corticosteroid therapy.

skin folds, and with no blisters or exudate, suggesting traumatic abrasion related to the frontal impact of the airbag when it inflated rapidly (Figure, A). The right side of the face presented a large, well-delimited, edematous plaque with no solution of continuity or associated signs of friction, an exudative appearance, and with the presence of small blisters; the lesion was considered to be a burn due to first and second-degree thermal or chemical contact (Figure, A). Ophthalmologic examination showed marked edema of the eyelids with conjunctival chemosis secondary to eye trauma (Figure, B) and diminished visual acuity secondary to retinal trauma. The patient also presented edema and small superficial erosions of the cornea that, in association with the conjunctival hyperemia and eyelid and facial burns, suggested chemical keratitis. Orbital and facial bone fractures were ruled out. The injuries were thoroughly cleansed with water, and the patient was prescribed topical and systemic antibiotics and corticosteroids plus an eye antibiotic. There were no further complications, and the patient was therefore discharged. The burns showed significant improvement at follow-up 10 days later (Figure, C).

Most airbag injuries are minimal and consist of superficial skin lesions on the face, neck, and upper limbs.<sup>2</sup> However, serious injuries directly related to explosive inflation of the airbag have been observed: lacerations and other skin lesions, eye trauma, airbag-induced asthma, cranial, cervical, or limb fractures, and even death.<sup>2,5</sup> Airbags are made of nylon and are housed in the center of the steering wheel. When the sensors detect rapid deceleration, an electrical mechanism is activated which triggers a series of chemical reactions that ignite a sodium azide ( $\text{NaN}_3$ ) pellet. The pellet reacts with potassium nitrate ( $\text{KNO}_3$ ) leading to the very

rapid production of a large amount of high-temperature nitrogen gas, as well as sodium hydroxide and other gases, causing the bag to inflate within 20 hundredths of a second (300 km/h). Moments after the airbag inflates, the gas produced begins to dissipate through the small pores of the fabric. Deflation occurs in less than 2 seconds, allowing the occupants to move. The high-temperature gases (sodium hydroxide,  $\text{CO}_2$ , and other metal oxides) produce a corrosive alkaline aerosol able to cause alkali chemical skin burns and thermal burns.<sup>3-6</sup> Thermal burns can also be caused by a direct mechanism when the skin comes into contact with high-temperature gases or by an indirect mechanism, secondary to clothing burning and melting.<sup>6</sup> Irritant contact dermatitis due to the solid residues—talc and sodium hydroxide—has also been reported. Additionally, the corrosive effect of the gases has been implicated in alkali keratitis and other corneal lesions,<sup>7</sup> and direct eye trauma induced by the airbag can cause retinal damage and a temporary or permanent loss of visual acuity.<sup>10</sup>

Fortunately, most airbag burns are minor, superficial, and only require symptomatic treatment, but there are some reports of airbag burns that required aggressive debridement and skin grafts.<sup>8,9</sup> After cleansing chemical and thermal burns with abundant water, treatment usually consists of topical and systemic antibiotic and corticosteroid therapy, followed by close monitoring due to the higher number of complications. Friction burns are less challenging and do not require such careful monitoring.<sup>5,6,8,9</sup>

In conclusion, airbag burns are common but usually very mild. Dermatologists lack familiarity with this condition, which is often associated with keratitis and other eye injuries. The multiple mechanisms of the injuries produce diverse types of burns, sometimes in the same patient. Thermal and chemical burns require a different, more aggressive therapeutic approach than simple abrasions.

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## Pityriasis Rubra Pilaris With Focal Acantholytic Dyskeratosis During Treatment With Imiquimod 5% Cream

### Pitiriasis rubra pilaris acantolítica durante tratamiento con imiquimod 5% crema

*To the Editor:*

A 56-year-old man was in his second week of treatment with imiquimod 5% cream for superficial basal cell carcinoma on his back when he developed influenza-like symptoms accompanied by a desquamative erythematous eruption that started on his head and quickly extended

caudally. Examination revealed erythroderma with small islets of healthy skin, incipient peau d'orange on the palms and soles, orange-colored erythema of the face, mild ectropion, and small keratotic follicular papules on the chest (Figure 1). The mucosas were not affected. Two skin biopsies—one from the abdomen and the other from a keratotic papule on the chest—revealed identical findings, namely, alternating areas of parakeratosis and focal dyskeratosis with acantholysis, discreet spongiosis, and a band-like lymphocytic infiltrate in the superficial dermis (Figure 2). Laboratory results were normal and HIV serology was negative. Treatment with imiquimod was stopped and acitretin (35 mg/d) started, leading to complete resolution of the lesions after 2 months. The patient refused to undergo an allergy workup to rule out a possible role for



**Figure 1** A, Shiny erythematous skin on the legs. B, Erythroderma with sparing of skin islets (arrows). C, Orange keratoderma with a waxy appearance on the palms and soles. D, Keratotic follicular papules on the anterior thorax.