Staphylococcal Scalded Skin Syndrome in a Healthy Adult

Síndrome da Pele Escaldada Estafilocócica num Adulto Saudável

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A 64-year-old man presented to the emergency room (ER) in February 2021 with fever, otorrhoea, redness, and discharge in the right eye. The patient was a smoker, had been diagnosed with essential hypertension, and was a hepatitis B inactive carrier. He did not take any medication.

His vital signs were normal, and the laboratory investigation showed an elevated leucocyte count and an elevated C-reactive protein (CRP) of 10.0 mg/dL. After observation by an otolaryngologist and an ophthalmologist, he was diagnosed with acute otitis and conjunctivitis and was treated with oral amoxicillin, clavulanic acid, and topical chloramphenicol. At the time, he also showed an erythematous rash in the groin and was treated with topical fluconazole.

Nine days later, the patient returned to the ER due to a significant worsening of the dermatological findings (Figs. 1 and 2). He had a generalized rash with reddish desquamative lesions covering more than 70% of his body surface, which caused severe itching and pain throughout his body. The vital signs were stable, the CRP was lower than in the previous ER admission (3.4 mg/dL), and no other significant findings were found. Nikolsky's sign (blistering of skin when applied pressure) was evident while inserting a central venous catheter.[1] Due to the exuberance of the lesions, computed tomography of the pelvis was performed to rule out Fournier gangrene, which revealed "diffuse liquid infiltration of the subcutaneous tissue, without liquid or air collections" (Fig. 3). Blood cultures were negative.

The patient was put on antibiotics and corticosteroid therapy and was admitted to the ward. A skin biopsy was performed and revealed subcorneal cleavage with no significant inflammatory infiltrate.

The diagnosis of staphylococcal scalded skin syndrome (SSSS) was considered since *Staphylococcus aureus* is a frequent agent in bacterial conjunctivitis.[2] Despite negative blood cultures, SSSS was diagnosed due to its toxin-mediated pathogenesis. The disease is caused by exfoliative toxins produced by *Staphylococcus aureus,* which act on desmosomal proteins in the epidermis, leading to subcorneal splitting of the skin.[1] This explains why bacteremia is not required for SSSS to manifest. The significance of the skin biopsy lies in confirming this pathophysiological mechanism by showing subcorneal cleavage without inflammatory infiltrates, supporting the diagnosis.

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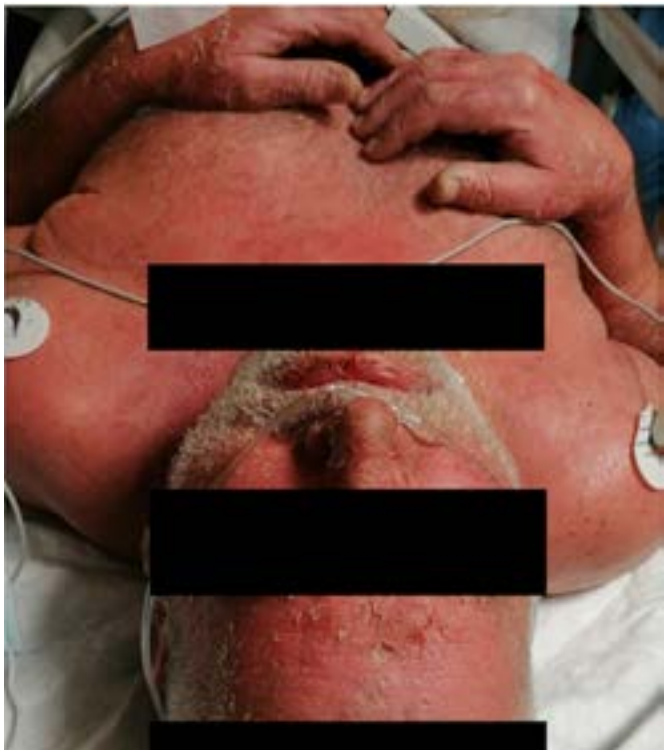

Figure 1. Red desquamative lesions of the face and lips.

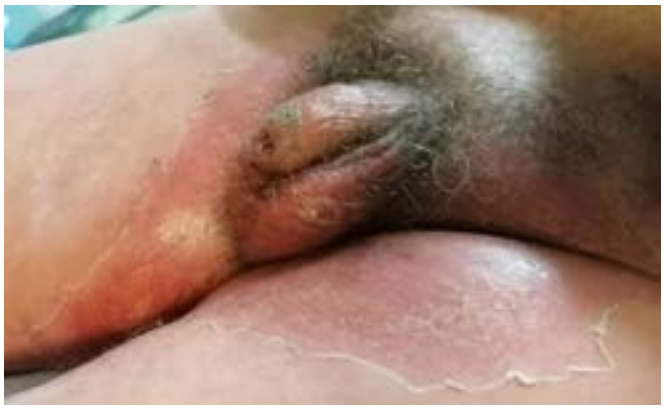

Figure 2. Red desquamative lesions of the perineum.

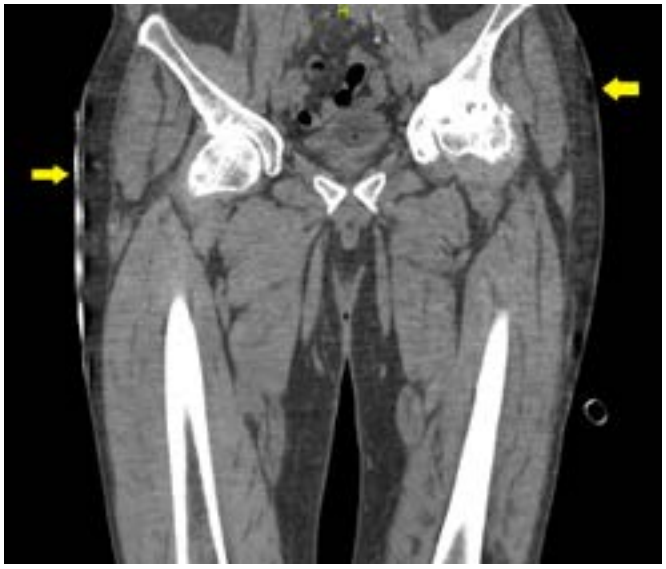

Figure 3. Diffuse liquid infiltration of the subcutaneous tissue of the external surface of both thighs.

The incubation period from *Staphylococcus aureus* infection to SSSS usually ranges from 1 to 10 days – in this case, mild manifestations were already present during the first ER visit and worsened in the following days.[1] This entity is rare in adults, and the risk factors related to immunosuppression, which our patient did not present.[3]

The hypotheses of Stevens-Johnson syndrome and toxic epidermal necrolysis were also considered but deemed less likely because the signs and symptoms began at a time when the patient was not receiving any medication, and both these entities are usually triggered by a drug.[4]

Treatment included the use of antibiotics, specifically flucloxacillin and clindamycin, which were chosen to target methicillin-sensitive *Staphylococcus aureus* and to inhibit toxin production.[3] Corticosteroids were administered to reduce inflammation and alleviate symptoms associated with the extensive skin damage. Although their use in SSSS remains controversial due to concerns about potential immunosuppression, they were deemed appropriate in this case to manage the severe skin manifestations.

The skin manifestations improved, the lesions became increasingly less painful and more desquamative, especially in the palms, and eventually disappeared. The patient was discharged after 10 days of flucloxacillin and 9 days of clindamycin. Although mortality rates can be up to 60% in adults with SSSS, our patient's evolution was benign.[1]

Ethical Disclosures

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