Images in Allergy

Toxic Epidermal Necrolysis in a Child

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A previously healthy 1-year-old boy presented to the Department of Respiratory Medicine with a 10-day history of cough, wheeze, and fever. Despite therapy with antibiotics (cefaclor, 30 mg/kg/d orally, divided into 3 doses) and bronchodilators (procaterol hydrochloride, 2 μg/kg/d, twice daily) for 7 consecutive days at the local primary health care facility, the child's respiratory status progressively deteriorated and he was referred to our institution. A chest radiograph revealed bilateral patchy consolidation. *Streptococcus pneumoniae* urinary antigen was positive. Intravenous amoxicillin-clauvulanic acid, inhaled corticosteroids and bronchodilators, and oral mucolytics were administered for a working diagnosis of community-acquired pneumonia. On the second day of hospitalization, the patient developed an irregular macular rash on the face, neck, and trunk. Beta-lactam antibiotic allergy was suspected. Cetirizine hydrochloride was administered and the antibiotic was replaced with meropenem. In the following 3 days the rash transformed into a confluent maculopapular exanthema with blisters and denudation of the epidermis in large patches that involved large areas (roughly 90%) of the body surface (Figure 1, *A* and *B*). Multiple erosive lesions developed on the oropharyngeal, anal, and ocular mucosa. In addition, he was febrile and dyspneic. Toxic epidermal necrolysis (TEN) was confirmed and the child was transferred to the pediatric intensive care unit for hypoxemia. In addition to noninvasive ventilation and supportive systemic therapy, he was treated with intravenous methylprednisolone and gamma globulin, plasmapheresis, surgical wound care, and ophthalmic lubricant. *Acinetobacter baumannii* and *Klebsiella pneumoniae* were isolated from wound swab cultures and the antibiotic treatment was changed to intravenous tigecycline and polymyxin. During the hospitalization period, fluid balance and creatinine remained stable. The patient's SCORe of Toxic Epidermal Necrosis was 2 (mild) (body surface area involved $>$ 10%, score 1; tachycardia $>$ 120 bpm, score 1).[1] The skin reepithelialized progressively (Figure 1, *C* and *D*) and the patient was discharged after a 14-week stay.

TEN is a potentially life-threatening disorder characterized by widespread epidermal necrosis of skin and mucosa involving greater than 30% of the body surface area.[2] Although the precise pathogenic mechanism is not understood, cutaneous reactions in

FIGURE 1. Irregular macular rash on the face, neck (**A**), and trunk (**B**) that extends in a confluent maculopapular exanthema with blisters and denudation of the epidermis in large patches. (**C** and **D**) Skin reepithelialization during the recovery process.

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TEN lead to keratinocyte apoptosis, which may be induced by drugs or infections or be idiopathic. Medications are the most common reported cause. The culprit antibiotic causing TEN in this patient is not certain, because both cephalosporins and beta-lactamase inhibitors may be etiological factors. TEN usually develops between 4 and 28 days after drug intake; however, in rare cases of rechallenge with a drug with similar structure, the disease appears more rapidly, within hours. This might suggest that the TEN developed in response to coamoxiclav, although the immune-related cytotoxic reaction could have been initiated during the initial antibiotic treatment with cefaclor. The management of TEN is challenging, involving a wide array of costly resources, prolonged hospital stays, and potential mortality.[2] The outcomes depend on prompt discontinuation of the suspected causative drug, antimicrobial treatment, wound care, and supportive therapies.

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