Acute Generalized Exanthematous Pustulosis Induced by Iopromide

İopromide Bağlı Akut Generalize Ekzantematöz Püstülozis

To the editor:
Acute generalized exanthematous pustulosis (AGEP) is a rare, but well-recognized condition that is usually attributed to drugs (1). AGEP, which is also known as toxic pustuloderma or pustular drug eruptions, is characterized by acute onset of widespread pustular eruptions (1, 2). We present a case of AGEP that was triggered by an iopromide-containing radiocontrast media. A 67-year-old woman presented to our clinic complaining of a 5 day history of malaise, pruritus, and a skin eruption that she said began 4 days after the administration of iopromide-containing radiocontrast media for a coronary angiography. The patient had hypertension, diabetes mellitus, coronary artery disease, and asthma. The patient had no history of psoriasis. Her daily medications included acetylsalicylic acid, diltiazem, furosemide, budesonide, formoterol fumarate, salbutamol/ipratropium combination therapy, and montelukast sodium. Cutaneous examination showed erythematous macules and plaques on the abdomen, back, and bilateral lower and upper extremities. Pustules were noted over her abdomen and lower extremities (Figure 1). There was no involvement of her palms, soles, or mucosae. The patient was afebrile. Laboratory findings showed leukocytosis with 87.8% neutrophils. Biopsy specimens showed neutrophil clusters in the stratum corneum and papillary edema (Figure 2). Treatment with an antihistaminic, systemic, and topical steroid was started, and the eruption had markedly improved with desquamations within 1 week.

AGEP is generally attributed to drugs (in over 90% of the cases) (1, 3). β-lactam antibiotics, macrolides, tetracyclines, doxycycline, vancomycin, isoniazid, quinolones, hydroxychloroquine, diltiazem, itraconazole, nystatin, terbinafine, nonsteroidal anti-inflammatory drugs, and antiepileptic drugs are associated with AGEP. Viral infections, mercury hypersensitivity, and spider bites have also been implicated (1, 2, 3, 4). AGEP usually develops acutely and is characterized by numerous non-follicular sterile pustules on an erythematous background. The eruption mostly develops on the face or...
intertriginous areas, disseminates rapidly, and resolves after discontinuation of the causative drug treatment (5). Sometimes, additional manifestations include marked edema of the face, purpura, blisters, or target-like lesions. The skin symptoms of AGEP are frequently accompanied by fever and leukocytosis. Mucous membranes, usually the oral mucosa, are involved in 20% of the cases. The lesions usually heal completely in 15 days (2).

A drug-specific T-cell-mediated mechanism, which is evidenced by positive findings on patch tests and lymphocyte transformation tests, plays an important role in the pathogenesis of AGEP (2, 5). The histopathological findings in AGEP include papillary edema, neutrophil clusters in the dermal papilla, perivascular eosinophils, and intraepidermal or subcorneal pustules (2, 5).

The major differential diagnosis is acute pustular psoriasis. However, no psoriasis history, a history of recent exposure to drugs, and the short course of AGEP (less than 15 days) are important clues (2). The treatment of AGEP is symptomatic and rarely requires the use of systemic corticosteroids. To our knowledge, there are few reports of radiocontrast-induced AGEP in the literature, and this is the first case of iopromide-induced AGEP to be reported (5).

REFERENCES