Mehmet Fatih Orhan
M.D.
Department of Pediatrics
Ataturk University Faculty of Medicine,

Hasan Kahveci
M.D.
Department of Pediatrics
Ataturk University Faculty of Medicine,

Handan Alp
Prof., M.D.
Department of Pediatrics
Ataturk University Faculty of Medicine,
halp@atauni.edu.tr

Mustafa Buyukavci
Assocc. Prof., M.D.
Department of Pediatric Oncology
Ataturk University Faculty of Medicine,
buyukavci@hotmail.com

Dear Editör,

A case of heliotrope rash associated with acetylsalicylic acid

(Asetilsalisilik asite bağlı heliotrop döküntü görülen bir çocuk olgu)

Heliotrope rash is a reddish-purple, violaceous discoloration of the upper eyelids. It is one of the most typical cutaneous manifestations of juvenile dermatomyositis (JDM) (1). However, the eruptions of upper eyelid mimicking the heliotrope rush, may rarely be observed as an adverse effect of some drugs (2). In this case, we reported a child presented with heliotrope rash after use of acetylsalicylic acid (ASA).

A 19 month-old girl presented with two-week history of fever, 10 days of multiple erythematous rash on her body and discoloration of her upper eye-lids. She had received two pills of ASA for her fever in days preceding cutaneous lesions. Physical examination revealed edema and heliotropic rash on her eye-lids, and multiple erythematous lesions on her skin (Fig. 1). Laboratory investigations revealed white blood cell count of 2.4×10^3 /mcL (PNL 80 %), erythrocyte sedimentation rate of 80 mm/hour, C-reactive protein of 7.9 mg/dL (N<0.8), and positive antinuclear antibody (ANA) test. Aspartate aminotransferase, alanine aminotransferase, creatine kinase (CK) and CK-MB were normal. Skin biopsy was reported as leukocytoclastic vasculitis. Clinical and laboratory abnormalities improved 15 days after prednisolone (1 mg /kg/day) treatment. She was followed up for eight months without any symptoms.

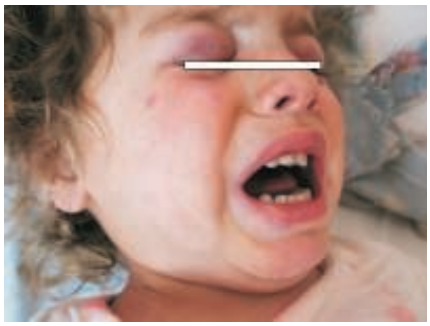

Figure 1. Edema and heliotropic rash on her eye-lids.

JDM is an inflammatory multi-system disease of unknown etiology with classic involvement of the skin and striated muscles. In the diagnosis of JDM, Bohan and Peter criteria including characteristic cutaneous changes, symmetric weakness of the proximal muscles, elevation of the serum level of the skeletal muscle enzymes and typical findings from electromyography and muscle biopsy are used. Typical skin involvement includes heliotrope rash, facial erythema, Gottrons sign and nailfold capillary abnormalities. Rarely, typical skin changes may occur without evidence of myositis. In practice, a diagnosis of JDM requires the presence of the pathognomonic rash and three of the other criteria (3). In our case, the lack of the proximal muscle weakness and the normal serum levels of the skeletal muscle enzymes were inconsistent with the diagnosis of JDM.

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Corresponding Author:
Doç. Dr. Mustafa Buyukavci
Department of Pediatric Oncology
Ataturk University Faculty of Medicine,
Erzurum - Turkey

Phone: 90 - 442 2316891
Email: buyukavci@hotmail.com

Leukocytoclastic vasculitis is defined as necrotizing vasculitis affecting small blood vessels usually caused by immune complex deposition. This is the predominant reaction in Henoch-Schönlein purpura and connective tissue diseases such as systemic lupus erythematosus. However, it is sometimes observed due to drug hypersensitivity (4). In literature, we could not found any case of leukocytoclastic vasculitis occurred following ASA use. In our case, the presence of the medication at disease onset, maculopapular rash and specific histologic changes on biopsy material were consistent with the diagnosis of hypersensitivity vasculitis according to the American College of Rheumatology criteria (4).

In conclusion, heliotrope rash was thought to be due to leukocytoclastic vasculitis associated with ASA in this case. We would like to emphasize that the previous medication must be investigated in children presented with heliotrope rash.

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