Erythematous Rash on the Face and Neck

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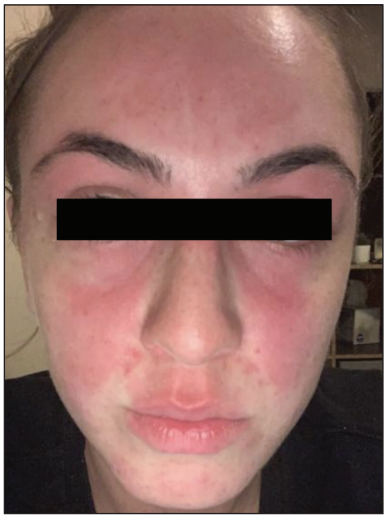

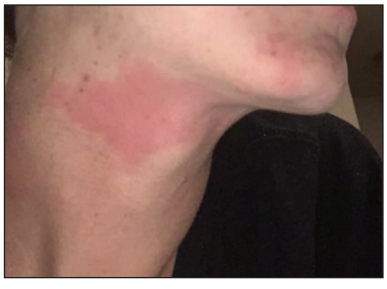

A 23-year-old woman with atopic dermatitis and seasonal allergic rhinitis presented to the dermatology department with an erythematous pruritic rash of 1 year's duration involving the forehead, periorbital and submental skin, and neck. The patient's atopic dermatitis was stable and had been well controlled with dupilumab and topical triamcinolone as needed for flares. The patient denied any other symptoms including fever, fatigue, and muscle weakness. Physical examination of the hands and nails revealed no abnormalities. She was treated with topical triamcinolone acetonide 0.1% without improvement. Short-term prednisone tapers fully resolved the rash, but it recurred within 5 days after discontinuation of prednisone. Results of testing for rheumatoid factor, antinuclear antibodies, complete blood count, comprehensive metabolic panel, C-reactive protein, erythrocyte sedimentation rate, and antistreptolysin O antibodies were unremarkable.

WHAT IS YOUR **DIAGNOSIS?**

a. allergic contact dermatitis
b. atopic dermatitis flare
c. dermatomyositis
d. erythematotelangiectatic rosacea
e. systemic lupus erythematosus

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THE **DIAGNOSIS:** Allergic Contact Dermatitis

In our patient, the erythematous pruritic rash on the face and neck, the lack of systemic symptoms, and her history of atopic dermatitis suggested a diagnosis of allergic contact dermatitis (ACD). She underwent patch testing with standard, fragrance, and cosmetic panels in addition to 6 of her personal care products. Her first patch test, which was read on day 2, showed a positive reaction to isopropyl myristate (IPM), a penetration enhancer used in cosmetics, topical medications (eg, tretinoin), and cosmeceuticals. The reading on day 5 showed a 2+ reaction to IPM, which was found in several of her personal care products, including her shampoo, leave-in conditioner, and eczema-calming cream. Isopropyl myristate is used in these products because of its ability to enhance their penetration into the skin and also can be found in commercially used products such as hand sanitizers. The patient was given information on this allergen and how to identify and avoid triggers. At follow-up, the ACD had resolved with avoidance of IPM.

Contact dermatitis is an inflammatory skin condition that is triggered by contact with a specific causative agent. There are 2 types of contact dermatitis: irritant and allergic; the irritant type is more common (approximately 80% of cases worldwide).[1] Allergic contact dermatitis is a type IV (delayed-type) hypersensitivity reaction; common causative agents include shampoos, moisturizers, makeup, certain metals (eg, nickel), fragrances, latex, and certain plants (eg, poison ivy).[2] In cases of ACD, a new reaction can develop from exposure to a product that the patient has used for years. It manifests clinically as erythema, pruritus, scaling, and vesicle formation.[1] Certain populations, such as those with atopic dermatitis, are more prone to developing ACD due to a breakdown of the skin barrier, frequent use of topical products, and immune dysregulation.[1,2] Patch testing performed by dermatologists and allergists is the gold standard for diagnosing ACD.[1,3]

Annually, allergists, dermatologists, and primary care physicians see thousands of cases of contact dermatitis.[1] Early recognition and appropriate treatment can help reduce the severity and duration of symptoms and improve patient outcomes. The main treatment for ACD is identification of the causative agent followed by patient education on how to identify and avoid triggers.[2] Once patch testing has been completed, patients can be given access to the American Contact Dermatitis Society's Contact Allergen Management Program (CAMP) database (https://www.contactderm.org/resources/acds-camp) to help them identify and avoid products that contain triggering allergens.

Topical corticosteroids are the first-line pharmacologic treatments for atopic dermatitis.[4] When our patient presented with the facial rash, her atopic dermatitis had been well controlled with both dupilumab and topical triamcinolone. The lack of response to previously successful therapies in a new area of involvement made a flare of atopic dermatitis less likely. For flares of ACD after exposure, topical corticosteroids and topical calcineurin inhibitors can help. If needed due to severity, oral corticosteroids also can be used.[1]

Dermatomyositis is an inflammatory myopathy that has several skin manifestations, including a heliotrope rash and poikiloderma.[5] While our patient's rash covered the periorbital area, she did not have other classic skin findings of dermatomyositis, such as nail-fold capillary changes or poikiloderma in a shawl or holster distribution.[6] She also lacked signs of systemic involvement including myositis and elevated C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), and creatine kinase levels.[5]

Erythematotelangiectatic rosacea is characterized by telangiectasias and transient flushing and erythema on the central face.[5] Rosacea typically is triggered by temperature changes, alcohol consumption, sun exposure, spicy foods, and stress[5] and would be expected to involve the nose, which was not observed in our patient. The fixed nature of our patient's patches and the absence of telangiectasias also argued against this diagnosis.

The classic cutaneous finding of systemic lupus erythematosus is a malar rash, which appears as erythematous patches or thin plaques across the bridge of the nose and over the cheeks, sparing the nasolabial folds.[5] Systemic lupus erythematosus is associated with laboratory abnormalities, such as positive antinuclear antibodies and elevated CRP and ESR levels.[5] Our patient had notable sparing of the nose, negative antinuclear antibodies, and normal CRP and ESR levels, making systemic lupus erythematosus unlikely. Systemic lupus erythematosus also can manifest with photosensitivity,[7] and involvement of the submental skin in our patient argued against a photosensitive eruption.

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