Molluscum Contagiosum and Atopic Dermatitis

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A 13-year-old boy with a history of chronic atopic dermatitis, which had mainly affected the antecubital and popliteal areas, presented with a diffuse papular eruption in the lower abdomen that had persisted for approximately 9 months. The atopic dermatitis, which had affected him since early childhood, had been treated intermittently with topical tacrolimus ointment, 0.03%.

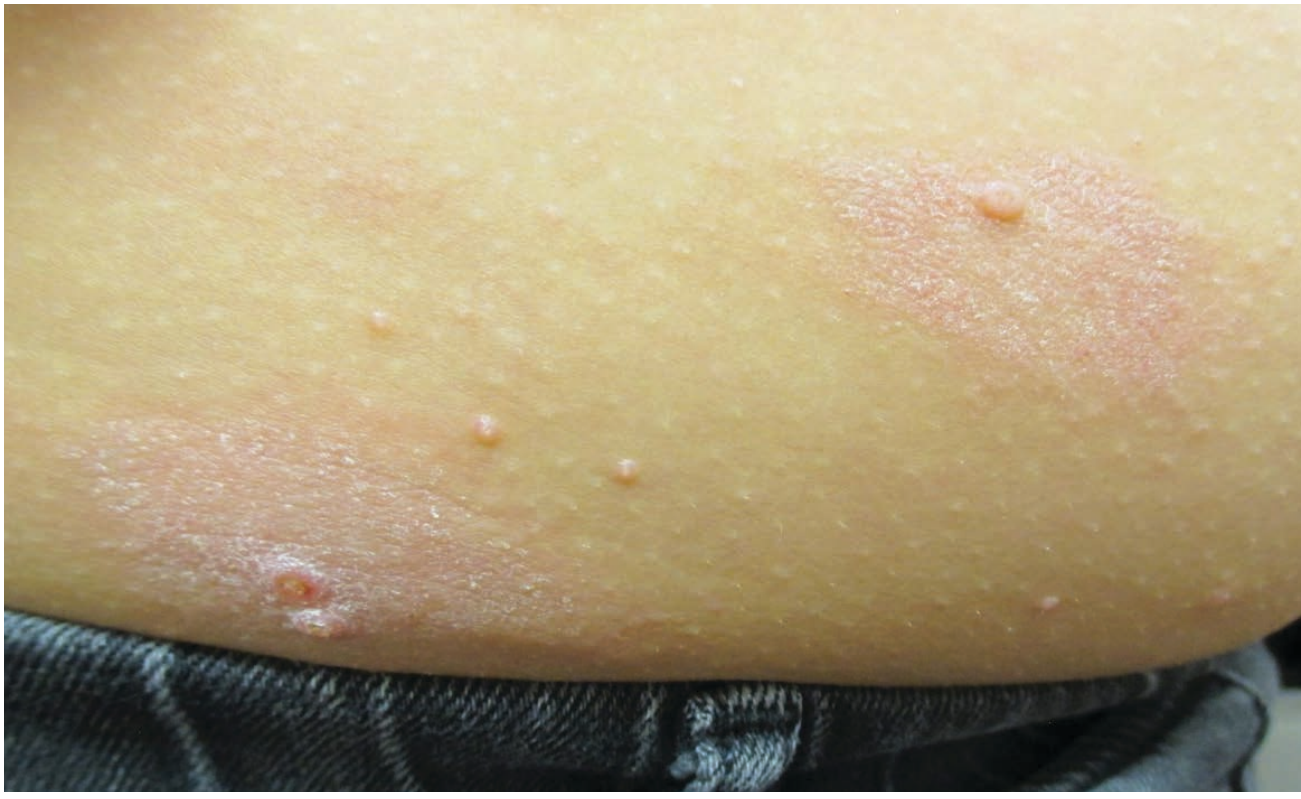
On physical examination, reddish-brown, excoriated, scaly, and lichenified lesions of atopic dermatitis were present in the antecubital and popliteal fossae. The papules on the abdomen were discrete, smooth, flesh-colored, and dome-shaped. Some of the lesions were umbilicated with a surrounding eczematous reaction. The patient's lesions were diagnosed as molluscum contagiosum.

The boy was advised to use a ceramide-based moisturizer twice a day on the entire body. His dermatologist applied cantharidin to the molluscum contagiosum lesions every 2 weeks. The molluscum and eczema disappeared after 3 treatments.

Molluscum contagiosum, a common viral cutaneous infection in childhood, is caused by a poxvirus of the *Molluscipox* genus. Elementary school-aged children are more commonly affected.[1] The virus is transmitted through close physical contact, autoinoculation, and fomites (eg, bath sponges, towels), especially if the skin is wet.

Typically, molluscum contagiosum presents as discrete, smooth, flesh-colored, dome-shaped, waxy papules with central umbilication from which a plug of cheesy material can be expressed. Lesions usually are 1 to 5 mm in diameter, and they usually number fewer than 20. They often appear in clusters or in a linear pattern. The lesions often are asymptomatic but sometimes may itch.[1] Surrounding eczema is common, as illustrated in the case described here. Approximately 10% of affected patients develop eczematous dermatitis around the molluscum contagiosum lesions.[2]

Atopic dermatitis is a chronically relapsing dermatosis characterized by pruritus, erythema, vesiculation, exudation, excoriation, crusting, scaling, and sometimes lichenification. Atopic dermatitis affects 10% to 20% of school-aged children.[3] Its pathogenesis involves complex interactions between



susceptible genes, immunologic factors, skin barrier defects, infections, neuroendocrine factors, and environmental factors.

Patients with atopic dermatitis have impairment of the barrier function of the skin and immunologic alteration. The loss of skin barrier function and alteration in cell immunity make the stratum corneum susceptible to microbial colonization.[3] Also, the barrier disruption in skin with dermatitis provides a portal of entry for microorganisms. As such, individuals with atopic dermatitis are susceptible to viral, bacterial, and fungal infections. Secondary bacterial infection, most commonly with *Staphylococcus aureus* and group A β-hemolytic streptococcus (*Streptococcus pyogenes*), is the main complication of atopic dermatitis.[3] Eczema herpeticum caused by herpes simplex virus is a potentially dangerous complication. Chickenpox can severely exacerbate atopic dermatitis and present as a generalized pruritic rash. Eczema vaccinatum, caused by variola virus, historically follows smallpox vaccination or exposure to atopic dermatitis in individuals with smallpox or who have been vaccinated with smallpox. Children with atopic dermatitis also are prone to molluscum contagiosum, verruca vulgaris (warts), and superficial fungal infections.[3]

Molluscum contagiosum is more common in individuals with atopic dermatitis, and its lesions tend to be more persistent, more extensive, and more disseminated in this patient population. Agromayor and colleagues reported that 49% of children in Spain under 10 years of age who were infected with molluscum contagiosum also had atopic dermatitis.[4]

The use of topical corticosteroids or immunomodulators (ie, tacrolimus and pimecrolimus) may be contributory; these agents lower the cell-mediated immunity locally and provide a favorable environment for molluscum contagiosum infection.[5]

The perilesional eczematous reaction may be due to a local sensitization to molluscum contagiosum elementary bodies or a soluble product of their metabolism.[6] Such sensitization occurs only in a minority of cases of molluscum contagiosum. The localized eczematous reaction also may be related to scratching of the lesions in susceptible individuals with atopic dermatitis. ■

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