Grover Disease (Transient Acantholytic Dermatosis) and Piebaldism

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A 35-year-old white man with lifelong stable white lesions on the anterior trunk and extremities presented with a pruritic papular eruption limited to the white patches. Results of a histologic examination led to a diagnosis of Grover disease (transient acantholytic dermatosis). To our knowledge, this article is the first to report an association between Grover disease and piebaldism or other depigmented disorders. We review the literature and speculate on the association between these conditions.

Case Report

A 35-year-old white man with lifelong stable white lesions on the anterior trunk and extremities presented with a 2-month history of a slightly pruritic eruption within the white patches. He reported not having had lesions on the normal-colored skin. His family history included a similar pigmentary disorder. The patient was not receiving any medications. The depigmented patches found on the abdomen and lower chest and bilaterally on the extremities were characteristic of piebaldism. Within these patches were several erythematous scaly papules 2 mm in diameter (Figure 1). Similar lesions were not found on the normally pigmented skin. Findings from a histologic examination included acantholysis, dyskeratosis, and a lymphocytic infiltrate characteristic of Grover disease (Figure 2).

Comment

Although the exact cause of Grover disease is unknown,[1-5] the condition has been associated with many factors, particularly heat and sweating in bedridden patients.[6-8] Hu et al[7] suggested that obstruction of sweat glands leads to escape of sweat urea into the epidermis, where acantholysis is the result.[7] Grover disease has been reported in association with sun exposure; UVB radiation exposure[4]; and sulfadoxine-pyrimethamine,[9] interleukin-4,[10] and ribavirin[11] intake. Grover disease has been reported in association with several dermatologic diseases.[4] Grover and Rosenbaum[3] reported associations between Grover disease and asteatotic eczema, atopic dermatitis, and allergic contact dermatitis and concluded that nonspecific irritation and inflammation of the skin might precipitate Grover disease by the Köbner phenomenon.

Piebaldism is an autosomal-dominant condition with stable depigmentation of the skin but without extracutaneous manifestations.[12] Piebaldism results from mutations in the KIT protooncogene, which encodes a plasma membrane receptor for mast/stem cell growth factor—a survival and migratory factor for melanocytes.[13] These mutations disrupt the normal migration of melanocytes from the neural crest during embryonic development.[14] Although Grover disease is associated with a variety of cutaneous diseases, an association with piebaldism[12-17] or other hypopigmented diseases has not been reported to our knowledge. Guttate leukoderma was reported in a black patient with Grover disease.[18] Loss of intercellular contact might affect melanin production or the transfer of melanin to the keratinocytes and thereby result in decreased pigmentation.[18] Hypopigmentation has been noted in Darier disease, a condition involving focal acantholytic dyskeratosis similar to Grover disease.[19-21] Cornelison et al[20] proposed that the decreased pigmentation is a postinflammatory phenomenon, whereas Berth-Jones and Hutchinson[21] suggested that leukoderma might precede or even supersede vesiculation and thus represent subclinical acantholysis.

In our patient's case, any of several possible mechanisms may be involved in the development of Grover disease and in its limited distribution to depigmented patches. First, the disease seems to be associated with solar UVB radiation exposure.[4] With piebaldism, depigmented areas might be more

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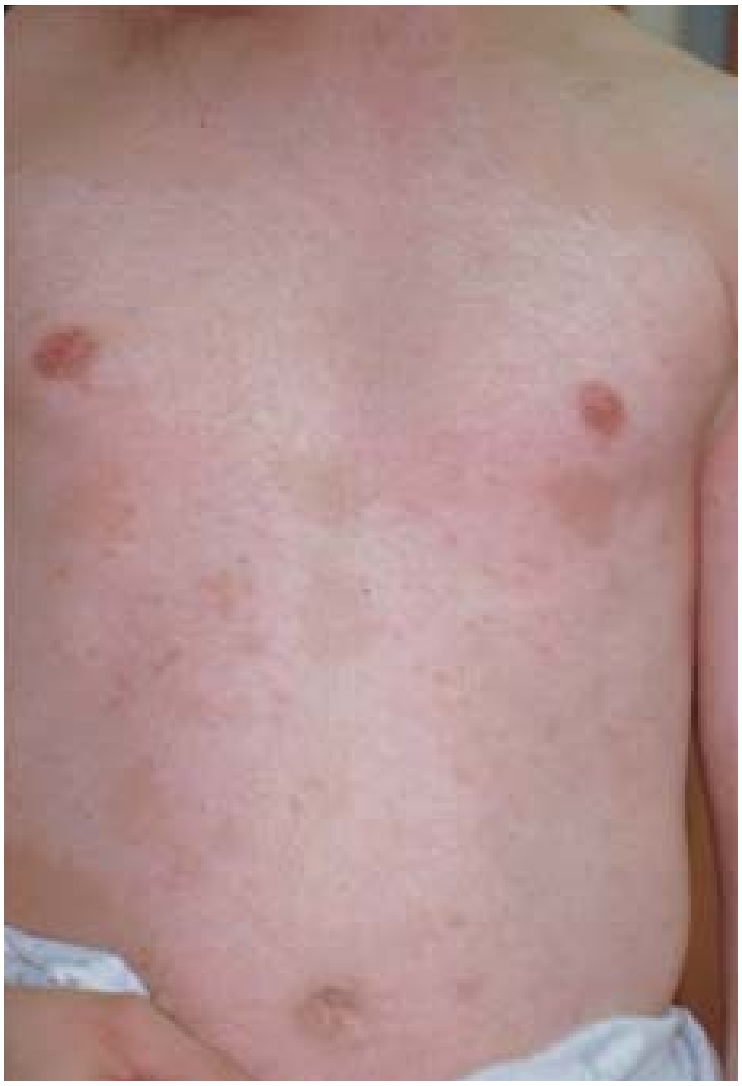

Figure 1. Papules of transient acantholytic dermatosis within the depigmented patches of piebaldism.

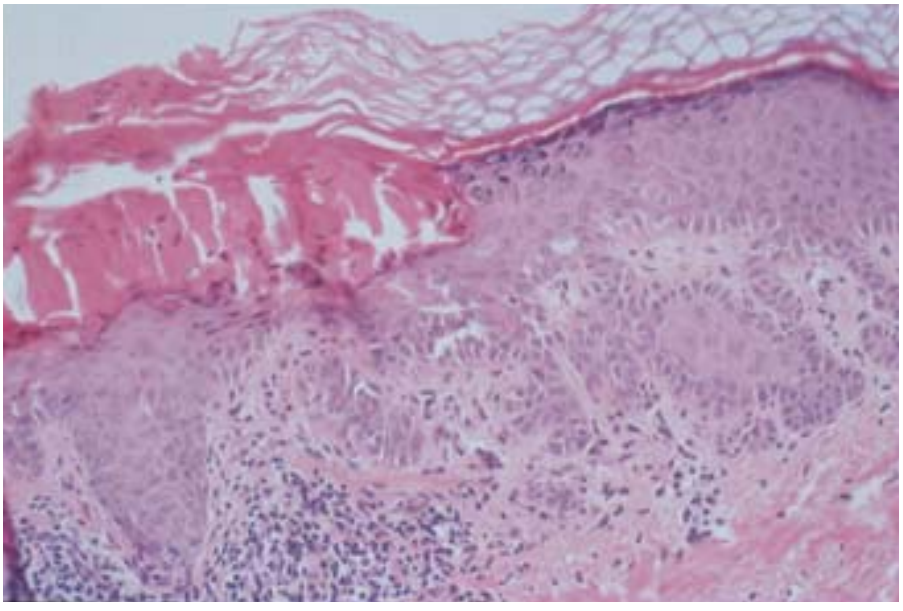

Figure 2. Findings from a histologic examination of a biopsy specimen included focal intraepidermal acantholysis, dyskeratosis of superficial epidermal cells, parakeratosis, and a superficial lymphocytic infiltrate (H&E, original magnification ×200).

sensitive to UVB radiation, and this higher sensitivity might facilitate the development of Grover disease in these areas. Second, the Köbner phenomenon has been implicated in several cutaneous diseases (eg, vitiligo, Grover disease). Whether piebald areas are more likely to be affected by the Köbner phenomenon and consequently to be more prone to acantholysis is unclear. Third, sweating has been strongly associated with Grover disease. Fourth, in piebald areas deprived of melanocytes, which are of neuroectodermal origin, an associated abnormal sympathetic regulation of sweat glands predisposes to increased sweating. Our patient reported no excessive sweating in the depigmented patches.

In summary, we report a case of Grover disease in which lesions were limited to depigmented patches of piebaldism. Similar observations in further studies may help explain the etiology of this association.

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