Answer to Dermacase *continued from* **page 901**

4. Inverse psoriasis

Psoriasis is a chronic inflammatory skin condition occurring in genetically predisposed individuals. It is typically, although not exclusively, induced by various environmental stimuli. The pathogenesis is complex and multifactorial, involving complicated immunologic and biochemical abnormalities that consequently result in deviant epidermal differentiation and vascular proliferation.[1] Psoriasis affects both men and women equally; it can appear at any age, but mostly manifests between the ages of 15 and 30 years. Approximately 90% of patients have plaque psoriasis, which is typically characterized by discrete, symmetric, well-demarcated, erythematous plaques covered with silvery-white scales; the most common sites of predilection include the extensor surfaces of the extremities, especially the elbows and knees, along with the scalp, lumbosacral region, buttocks, and genital areas. These preferential areas might be explained by an isomorphic response (ie, Köbner phenomenon)—the induction of new psoriatic lesions on normal skin after traumatic events. Several other distinctive clinical variants (inverse, guttate, pustular, and erythrodermic psoriasis) might also occur.[2]

Inverse psoriasis (IP), also called intertriginous or flexural psoriasis, is an uncommon variant that can be easily overlooked, leading to delayed or inappropriate treatment secondary to misdiagnosis. It mainly affects infants and young children, although it can manifest at any age.[3] Patients commonly report pruritus and sometimes stinging or even painful sensations due to fissurization of the skin lesions. The characteristic clinical feature of IP is the preferential involvement of prominant skin folds, such as the axillar, submammary, genitofemoral, intergluteal, and retroauricular areas. Inverse psoriasis usually manifests as smooth, shiny, well-demarcated, bright red plaques. Unlike the conventional plaque psoriasis, IP displays much less scaling and thickness, but it still retains the sharply demarcated erythema. It is believed that physical stimuli (such as friction and sweat), as well as various infections (including dermatophytosis and candidiasis), can trigger the appearance of IP.[4] The observed pattern of lesions occuring in IP is similar to the isomorphic response of plaque psoriasis. Considering the ambiguous clinical features, accurate diagnosis can be achieved by histopathologic examination. The typical pathologic findings include confluent parakeratosis, Munro microabscesses, spongiform pustules of Kogoj, hypogranulosis, suprapapillary thinning of the epidermis, and regular acanthosis, along with dilated capillaries in the dermal papillae and perivascular lymphocytic infiltration.[5]

Differential diagnosis

Inverse psoriasis mimics many intertriginous skin eruptions; these include common conditions, such as intertriginous cutaneous candidiasis and dermatophytosis, and less commonly encountered diseases, such as keratosis follicularis and benign familial chronic pemphigus. Intertriginous cutaneous candidiasis is predisposed to occur in patients who are obese, who wear occlusive clothing, who have diabetes, or who work in certain occupational environments.[6] It is characterized by itchy, erythematous, often confluent macerated patches, with satellite vesicopustules in the flexural areas. The diagnosis can be confirmed by potassium hydroxide examination or culture of skin scrapings showing the presence of yeast. Treatment involves keeping the skin dry in conjunction with the use of topical antifungal medications, such as nystatin or clotrimazole.

Dermatophytosis is a common superficial fungal infection. It also commonly affects the intertriginous areas, including the interdigital webs, groin, and intergluteal and axillary regions, and might sometimes bear resemblance to IP.[7] However, it typically presents as an annular, centrifugal, raised border. The central area of the lesion is usually scaly, but might at times be clear in appearance. Direct observation of spores and branching hyphae during potassium hydroxide examination is a direct measure for rapid diagnosis. Topical antifungal agents, such as terbinafine or butenafine, are often curative.

Keratosis follicularis, also known as Darier disease, is a late-onset autosomal dominant genodermatosis caused by loss-of-function mutations in the adenosine triphosphatase–2A2 gene, which provides instructions for producing an enzyme that regulates the levels of positively charged calcium ions inside the endoplasmic and sarcoplasmic reticula.[8] The onset of keratosis follicularis occurs between the first and second decades of life, similar to psoriasis. Typical lesions are greasy, keratotic, skin-coloured to yellow-brown papules on the seborrheic areas, such as the scalp, forehead, ears, upper chest, and back. Not uncommonly, the axillar, genitofemoral, and anogenital areas can be affected as well. Palmoplantar keratotic papules and nail abnormalities, including red and white longitudinal bands and V-shaped nicks at the free margins of nails, can be identified frequently. No curative treatment has been established at this time; the goals of pharmacotherapy are aimed at reducing morbidity and preventing complications. Topical treatment using retinoids, low- or mid-potency steroids, and antibiotics has been tried with variable success.

Benign familiar chronic pemphigus, also called Hailey-Hailey disease, is a late-onset blistering genodermatosis resulting from loss-of-function mutations in the adenosine triphosphatase–2C1 gene, which regulates the secretory pathway that supplies

calcium and manganese to the Golgi apparatus.[9] The clinical manifestation is characterized by itching or burning vesicles; painful erosions; and erythematous, erosive, scaling plaques, all of which typically occur in the flexural areas—the neck, axillae, submammary regions, and groin. Examination of the fingernails, which might reveal longitudinal white streaks, can be a helpful adjunct to achieving a correct diagnosis. The clinical course waxes and wanes, with transient improvement after the application of mid-potency topical steroids and topical antibiotics.

Treatment

The treatment of IP is somewhat different from that of plaque psoriasis owing to the occlusive nature of the intertriginous areas. According to recent evidence-based studies on the management of this particular subtype of psoriasis, the recommended first-line short-term therapy is low- to mid-potency topical steroids.[10] However, long-term therapy for IP is recommended, namely calcipotriene or topical immunomodulators (tacrolimus or pimecrolimus), to avoid the side effects related to the long-term application of steroids.[3,10]

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Competing interests
None declared

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