Barrier Disease Beyond Eczema: Management of Juvenile Plantar Dermatosis

There are few standard treatment options for juvenile plantar dermatosis, a poorly understood and understudied condition. Barrier repair therapies offer a new management option.

By Joseph Bikowski, MD

Understanding of the structure and function of the epidermal barrier has expanded greatly over the course of the last two decades.¹ Primary functions of the epidermal barrier are to protect against the entry of foreign substances (antigens, irritants, and microbes). In addition to these primarily defensive roles, the epidermis traps moisture and regulates hydration, and it synthesizes vitamin D.² The epidermal barrier is not a passive structure. As keratinocytes mature to horny corneocytes, the epidermis is in a constantly active self-proliferating phase. In addition to physically blocking entry of most foreign substances, the barrier coordinates an immunologic defense against pathogens that manage to bypass the barrier.

The structure of the stratum corneum has been described as a bricks-and-mortar structure. The “bricks” are covalently bonded corneocytes arranged in compact, overlapping layers to hold moisture in while keeping allergens, pathogens, and environmental toxins (such as UV radiation) out. The “mortar” consists of ceramides, cholesterol, and lipids. Together, these elements form lipid bilayers that fill the spaces between the corneocytes. This extracellular matrix provides necessary permeability of moisture to the stratum corneum.

**Take-Home Tips.** Juvenile plantar dermatosis tends to be chronic; typical interventions may be palliative but not curative. The differential diagnosis includes keratolyis exfoliativa and tinea pedis. Topical corticosteroids are a standard treatment. An anti-inflammatory barrier repair therapy may represent a suitable option for primary or adjunctive treatment in children with JPD.
A majority of research related to epidermal barrier function has focused on classic atopic dermatitis, which has come to be seen as the quintessential disease of barrier dysfunction. However, research continues to show that impaired barrier function contributes to a host of inflammatory dermatoses, including rosacea, acne, and psoriasis. The following provides a closer look at the diagnosis and management of a challenging cutaneous condition influenced by barrier dysfunction: juvenile plantar dermatosis or wet-to-dry foot syndrome.

Also known as wet-to-dry foot syndrome or sweaty socks syndrome, juvenile plantar dermatosis (JPD) is a poorly understood and understudied condition whose presentation may mimic that of numerous other common dermatoses. Due to a lack of published data on the condition, its prevalence is not well known. However, the condition may be somewhat common. There is some evidence that JPD may be a presentation of childhood atopic dermatitis and that it can persist into adulthood.

**Clinical Presentation**

Although juvenile plantar dermatosis was first described more than three decades ago, a literature search returns relatively few publications on the condition. Taken together, information from these few publications suggests that the condition tends to be chronic with an extended course of two to four years and that typical interventions may be palliative but not curative.

JPD typically presents as an erythematous rash of the weight-bearing plantar aspects of the feet; the distal one-third of the plantar surface of the feet and toes tends to be involved more frequently. The postal two-thirds of the plantar surface are not involved. Areas of involvement generally appear smooth and shiny with a high incidence of painful fissuring and cracking. In some cases, the skin of the affected areas desquamates. Although generally linked with hyperhidrosis, ironically, juvenile plantar dermatosis may be associated with anhidrosis.

The desquamation of the skin can mimic keratolysis exfoliativa, but this typically presents initially as pin-size white dots that coalesce. Furthermore, keratolysis exfoliativa tends to affect the palms of the hands more often than the feet and is often asymptomatic.

The differential diagnosis of JPD also includes tinea pedis. However, tinea pedis typically involves the fourth and fifth toe web spaces, whereas JPD generally spares the toe webs. Although tinea
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Varus rarely affects small children, clinicians should not assume based on a patient’s younger age that a questionable presentation is juvenile plantar dermatosis rather than tinea pedis. A potassium hydroxide (KOH) preparation can confirm or rule out tinea.

JPD may be misdiagnosed as classic atopic dermatitis (AD) of the foot, given the young age of affected individuals. While there is evidence of an association between JPD and AD, they are distinct diagnoses. In a 10-year follow-up of patients diagnosed with JPD, researchers found that 52 percent of JPD patients were atopic, and that JPD was often associated with the development of hand eczema in adulthood. About 26 percent of individuals who had JPD as children experienced hand eczema as adults.

Pathogenesis
The etiology and pathogenesis of JPD are not well understood, though it is generally accepted that excessive moisture of the feet contributes. There is no evidence for a fungal component to the disease. While there is no conclusive evidence implicating bacterial mediators, hypotheses suggest that bacterial colonization may be a factor in the pathogenesis. There is histopathological documentation of inflammation at the junctions of sweat-gland ducts and acrosyringia in affected individuals.

Irritants/allergens are not shown to contribute to the etiology of JPD. In the case of recalcitrant JPD, patch testing may be indicated; data show that in one study population, half of patients diagnosed with non-atopic plantar dermatoses ultimately had positive patch reactions to at least one tested item.

The process of skin fissuring and desquamation in JPD is not fully understood, but it may be likened loosely to the formation of syneresis cracks in dried mud puddles (See Sidebar). As water evaporates out of the puddle, shrinkage occurs, leading to the formation of cracks and fissures in the mud crust.

In JPD, when the foot is exposed to moisture over extended periods of time (either through hyperhidrosis or occlusion of the foot via footwear made of non-breathable synthetic materials), high levels of surface moisture develop. However, it is well established that exposure to water does not induce skin hydration; in fact, persistent water exposure is shown to disrupt epidermal barrier function. Furthermore, there is recent evidence that transepidermal water loss (TEWL) increases as temperature increases, a finding that may be relevant because non-breathable footwear may be associated with higher foot temperatures.

As the disrupted epidermal barrier permits excessive evaporation of subcutaneous moisture beyond normal evaporation of moisture on the surface of the skin skin surface water loss (SSWL), a cycle of further degradation and dysfunction is instigated. With lack of hydration, desiccated corneocytes on the epidermal layer shrink. It is likely that in the presence of depleted lipids and epidermal proteins, corneocyte adhesion is diminished, and fissures develop.

Syneresis Cracks

Derived from the Greek synairesis, meaning to contract, syneresis is the physical process of separation of liquid from a gel through contraction, and is even used in food chemistry to describe the making of jelly. The term is used as an adjective to describe the cracks that commonly form in desiccated mud puddles, which are also sometimes called sun cracks. As water evaporates from the muddy composite, fine sediment contracts and the "shrinkage cracks" form.
Another theory posits that in the hot, humid environment of the shoe, sweat becomes “trapped” in the skin, producing corneocyte edema and exaggerated shearing stress.7

Treatment Strategies
There is no specific treatment for JPD. Management strategies include avoidance of excess moisture through the selection of breathable footwear and the avoidance of footwear when possible. This may help to reduce the amount of sweating and also minimizes the effects of occlusion and friction that promote cutaneous peeling and cracking. Topical corticosteroids are frequently used to diminish acute inflammation but do not seem to directly affect the pathogenesis. Therefore recurrence is common upon discontinuation of corticosteroid therapy.

Given that inflammation and barrier dysfunction are primary components of JPD, an anti-inflammatory barrier repair therapy may represent a suitable option for primary or adjunctive treatment in children with the condition.

In clinical experience, twice-daily application of a barrier repair cream (EpiCeram, Promius Pharma) was associated with significant improvement in the appearance of the foot at the end of two weeks. Peeling and cracking were significantly reduced, and the patient reported reduced discomfort (Figs. 1a, 1b).

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