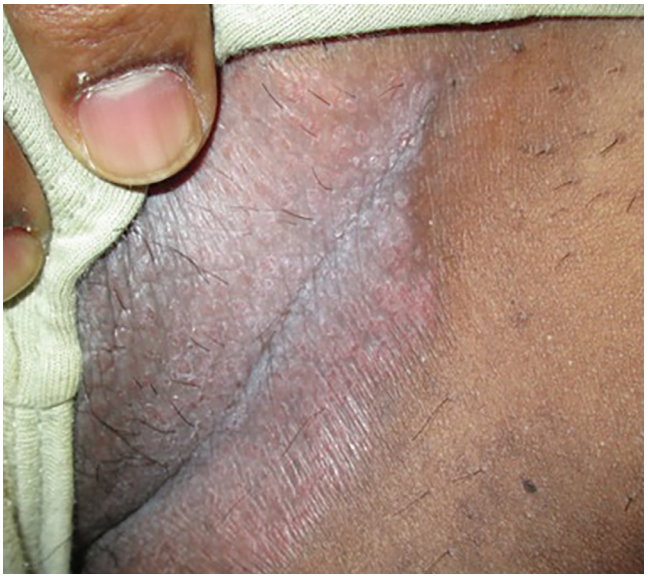
What's Causing This Man's Pruritic Rash?

Alexander K.C. Leung, MD—*Series Editor*
Benjamin Barankin, MD

A 40-year-old East Indian man presented with a chronic, recurrent, erythematous rash limited to the inguinal and axillary areas for the past 3 years. The rash was pruritic and at times slightly macerated, and there was no scaling. He treated himself with some over-the-counter medications recommended by his pharmacist, but it was of minimal benefit. Medical history was significant for diabetes mellitus. He did not consume alcohol or smoke. His 65-year-old father had psoriasis.



PHYSICAL EXAMINATION

On examination, there were well-demarcated, thin erythematous plaques in the inguinal and axillary regions. The skin surface was smooth, atrophic, non-scaly, and slightly shiny. He also had pitting of the fingernails. The rest of the physical examination was unremarkable.

What's Your Diagnosis?

A. Extra-mammary Paget disease
B. Fungal intertrigo
C. Glucagonoma syndrome
D. Inverse psoriasis
E. Langerhans cell histiocytosis
F. Seborrheic dermatitis

Answer: INVERSE PSORIASIS

Diagnosis of inverse psoriasis is usually clinical, based on the characteristic features, and family history of psoriasis and typical psoriatic lesions in other areas may aid in the diagnosis. A mycologic examination of skin scraping using potassium hydroxide may at times be necessary to differentiate it from fungal intertrigo. A skin biopsy should be considered if the diagnosis is in doubt. In this case, the patient's presentation was consistent with histological findings of inverse psoriasis, also known as flexural or intertriginous psoriasis.

An anatomic variant of psoriasis, "inverse psoriasis" is characterized by the involvement of skin-fold areas such as the inguinal folds, axillary folds, inframammary folds, retroauricular area, and gluteal cleft rather than the more common psoriatic involvement of the trunk, extensor surfaces of the extremities, and scalp.[1] Histologic examination of a classic lesion shows confluent parakeratosis with a loss of the granular layer, epidermal hyperplasia, elongation of rete ridges with thinning of the suprapapillary plates, and dilated capillaries in the dermal papillae with perivascular lymphocytic infiltration.[2,3] Munro microabscesses (intracorneal deposits of neutrophils) and Kogoj micropustules (neutrophil deposits in the stratum spinosum), if present, are diagnostic.[2]

EPIDEMIOLOGY

Estimates of prevalence of psoriasis range from 1% to 2%.[2] Inverse psoriasis presents in 3% to 7% of psoriatic patients.[1,4] The condition most commonly manifests between 15 and 30 years of age.[3] Both sexes are affected equally.[3] The condition is more common in Caucasian individuals than in black or Asian individuals.[2] The prevalence is greatest in northern, colder climates, and the disease is often more severe in the colder months. A family history of psoriasis in a first-degree relative is present in about 30% of patients with

childhood-onset psoriasis.[5] The concordance rate in monozygotic twins is approximately 70%, compared with 20% in dizygotic twins.[5]

ETIOPATHOGENESIS

Like plaque-type psoriasis, inverse psoriasis is characterized by hyperproliferation of epidermal keratinocytes and hyperkeratosis as well as a lymphocytic infiltration that consists mainly of T lymphocytes. Activation of T lymphocytes, migration of T lymphocytes to the skin, and T lymphocyte-mediated production of cytokines such as interferon gamma, interleukin-2 (IL-2), and tumor necrosis-factor alpha (TNF-alpha) are important in the pathogenesis.[5] Interferon gamma inhibits apoptosis of keratinocytes, IL-2 stimulates growth of T lymphocytes, and TFN-alpha increases proliferation of proinflammatory cytokines and adhesion molecules. The adhesion molecules further stimulate T lymphocytes to produce cytokines. Psoriasis is associated with various histocompatibility antigens, especially HLA-Cw6. Predisposing factors include the use of chloroquine, lithium, or beta blockers, withdrawal of corticosteroid in a susceptible individual, emotional stress, alcohol or tobacco consumption, trauma (Köebner phenomenon), friction, obesity, and infection.[2,3,5,6]

CLINICAL MANIFESTATIONS

Inverse psoriasis is characterized by smooth, sharply demarcated erythematous plaques with minimal or no scales.[3] In contrast to the common plaque form, the lesion tends to be shiny and moist due to the ambient humidity of the inverse location.[2] The lesions are usually symmetrically distributed. Some lesions may also have fissures, maceration, and rhagades. They often itch, sting, and/or burn.[3,4] Typically, inverse psoriasis affects the skin-fold areas such as the inguinal folds, axillary folds, inframammary folds, genital folds, retroauricular area, and gluteal cleft.[4]

Some patients may also have seronegative inflammatory arthritis and/or nail involvement such as pitting, discoloration, onycholysis, or onychodystrophy.[5] Patients with palmar and/or plantar psoriasis have an increased chance of having inverse psoriasis compared with the much more common plaque psoriasis.[2,4]

COMPLICATIONS

Inverse psoriasis has a significant negative impact on the quality of life. The lesions are cosmetically unsightly and socially embarrassing for some patients and can cause emotional distress in more severely affected individuals.[2] This form of psoriasis is more prone to maceration and secondary bacterial and/or fungal infection.

TREATMENT

Precipitating and exacerbating factors should be minimized or avoided. Optimal skincare requires constant attention to hydration and lubrication, and efforts to minimize itching. Low- to mid-potency topical corticosteroids are the first-line, short-term treatment of choice.[7] Local adverse effects such as skin atrophy, striae, and telangiectasia, occur with increased frequency which prevent the prolonged use of topical corticosteroids in the skin-fold areas.[7] Topical medications such as, calcipotriol or calcipotriene, and immunomodulators such as tacrolimus and pimecrolimus, while more expensive and not as rapid or efficacious as topical corticosteroids, are associated with fewer adverse effects and are recommended in the maintenance phase of treatment.[1,2,5,7] Application of irritant topical agents should be avoided. Narrowband UVB phototherapy or photochemotherapy psoralen UVA, either alone or in combination with other topical remedies, should be considered for widespread or severe psoriasis.[2,4,5] Systemic treatment is usually reserved for moderate to severe and generalized forms of psoriasis that are resistant to other therapies.[5] Combination, sequential, or rotational systemic therapy, with methotrexate, acitretin, or cyclosporine, can achieve additive or synergistic efficacy at lower dosages and with less risk of adverse events.[2,5] Medications such as adalimumab, apremilast, etanercept, infliximab, and ustekinumab which block molecular steps in the pathogenesis, have less potential for side effects but are more expensive.[1,2] Preliminary studies have shown some efficacy of botulinum toxin A in the treatment of inverse psoriasis[6,8]; further large-scale studies are necessary to confirm their findings.

OUTCOME OF THE CASE

Upon diagnosis by his dermatologist, the patient was treated with betamethasone valerate 0.1% cream nightly for 1 week, followed by topical tacrolimus 0.1% twice per week indefinitely. This treatment cleared up his psoriasis and resolved the associated itch, and appeared to keep the psoriasis at bay. ■

Alexander K.C. Leung, MD, *is clinical professor of pediatrics at the University of Calgary and pediatric consultant at the Alberta Children's Hospital in Calgary, Alberta, Canada.*

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