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# Not All Rashes Are Allergic: Keratoderma Blennorrhagicum-Like Rash Masquerading as Contact Dermatitis

**Maxwell Y. Li, MD[1,2], Jason Kolfenbach, MD[3], and Alan Schocket, MD[1]**

A 56-year-old Caucasian man was referred to the allergy clinic for evaluation of palmoplantar dermatitis. The patient's rash developed one year prior to presentation. He described erythema, pruritus, and hyperkeratosis of the involved skin with the eventual development of deep fissuring. He had a history of onychomycosis of his toenails but no history of fungal skin rash. He was exposed to solvents, mineral spirits, and gasoline through his occupation in home renovation and wearing neither nitrile nor cotton gloves alleviated his symptoms. Prior evaluations were carried out by primary care and dermatology. He had been treated with topical emollients, topical steroids (including potent agents such as clobetasol) for suspected atopic dermatitis, and topical antifungal agents as well (although KOH prep was negative). All prior treatments failed to resolve his severe palmoplantar rash.

His medical history included hypertension, obesity, fatty liver disease, uveitis, bilateral total hip arthroplasty, and a history of childhood allergic rhinitis for which he underwent allergen immunotherapy and was quiescent at the time of evaluation.

The physical examination was significant for moderately erythematous, hyperkeratotic, well-defined plaques on the palmoplantar surfaces of the hands and feet without dorsal involvement. Fissuring was seen at the fingertips and the plantar surface of the feet (Figure 1(A) to (D)) without associated pustulosis. There were no obvious nail pitting, oil spots, nor onycholysis, and scalp examination was normal. External examination of the eyes and oral examination were both normal. No appreciable synovitis was documented on peripheral joint examination, but the patient appeared "stiff" with ambulation as well as when stepping down from the examination table.

Patch testing result revealed a weak positive reaction to gold sodium thiosulfate and an irritant reaction to thimerosal. Common sensitizers of allergic contact dermatitis in the construction worker were evaluated. The patient did not have reactions to potassium dichromate found in cements, biocides such as isothiazolinones, rubber chemical, and metal allergens (ie, chrome, thiurams, carbamates, mercaptobenzothiazole) accounting for foot dermatitis from work boot materials, and epoxy resin. These results, along with the patient's history, led to decreased suspicion for either contact or atopic dermatitis. Given the patient's history of uveitis (which upon review of the chart was recurrent and associated with HLA-B27 positivity), the hyperkeratotic and plaque-like appearance of his lesions, and the concern for possible axial spine disease based on examination, formal radiographs were obtained (Figure 2) and a referral to rheumatology was initiated.

His rheumatologic evaluation confirmed decreased range of motion at the spine with an abnormal occiput to wall test of 8 cm, abnormal Schober's test (10–12 cm increase with flexion and no reversal of lumbar lordosis), and decreased excursion in lateral bending. The constellation of findings was consistent with a diagnosis of axial spondyloarthritis (axial SpA). His skin findings were thought to represent keratoderma blenorrhagicum versus palmoplantar-variant psoriasis (PPP) and were suspected to be a manifestation of his axial SpA. He denied a history of genitourinary or gastrointestinal infection, and urinary testing for gonorrhea and chlamydia was negative. Given failure of two prior

[1]Division of Allergy and Clinical Immunology, University of Colorado Anschutz Medical Campus, Aurora, Colorado
[2]Division of Allergy and Clinical Immunology, National Jewish Health, Denver, Colorado
[3]Division of Rheumatology, University of Colorado Anschutz Medical Campus, Aurora, Colorado

**Corresponding Author:**
Maxwell Y. Li, National Jewish Health, 1400 Jackson Street, Denver, CO 80206, USA.
Email: maxwelllimd@gmail.com

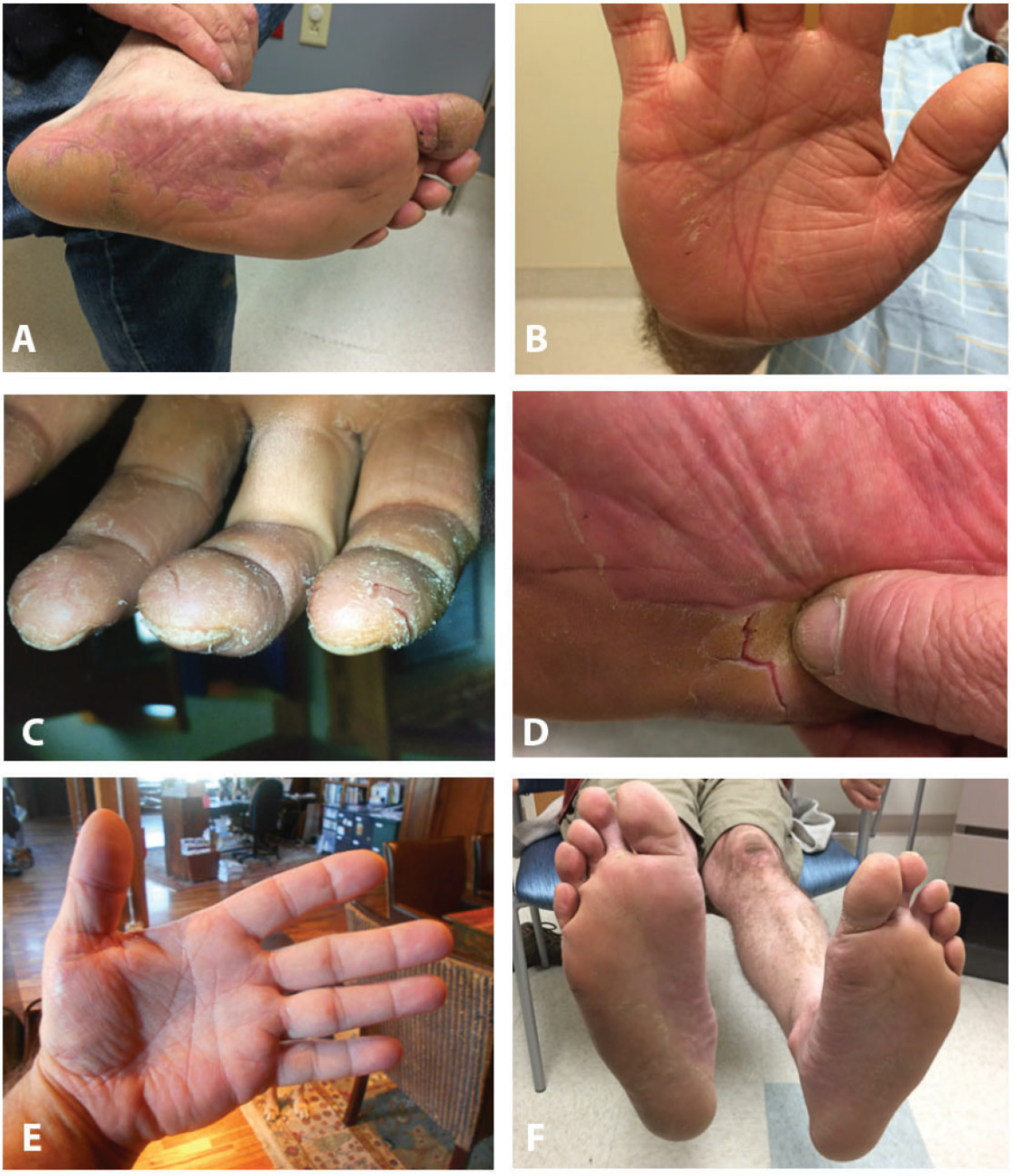


**Figure 1.** A–D, Cutaneous manifestations at baseline, with palmoplantar hyperkeratosis and fissuring at the fingertips and plantar surface. E, Response to anti-TNF therapy (adalimumab 40 mg every two weeks) at two months. F, Response to therapy for skin involvement at the feet was delayed and required an increased dosing regimen (adalimumab 40 mg weekly; photo taken three months following dosing regimen increase).

nonsteroidal anti-inflammatory drugs (NSAIDs) to relieve symptoms of back stiffness, he was started on adalimumab in the hopes of achieving benefit for his musculoskeletal and dermatologic manifestations. After four injections of adalimumab, the patient experienced significant improvement in cutaneous manifestations at the palms (Figure 1(E)). Clinical involvement at the feet proved more recalcitrant and required an increase in the

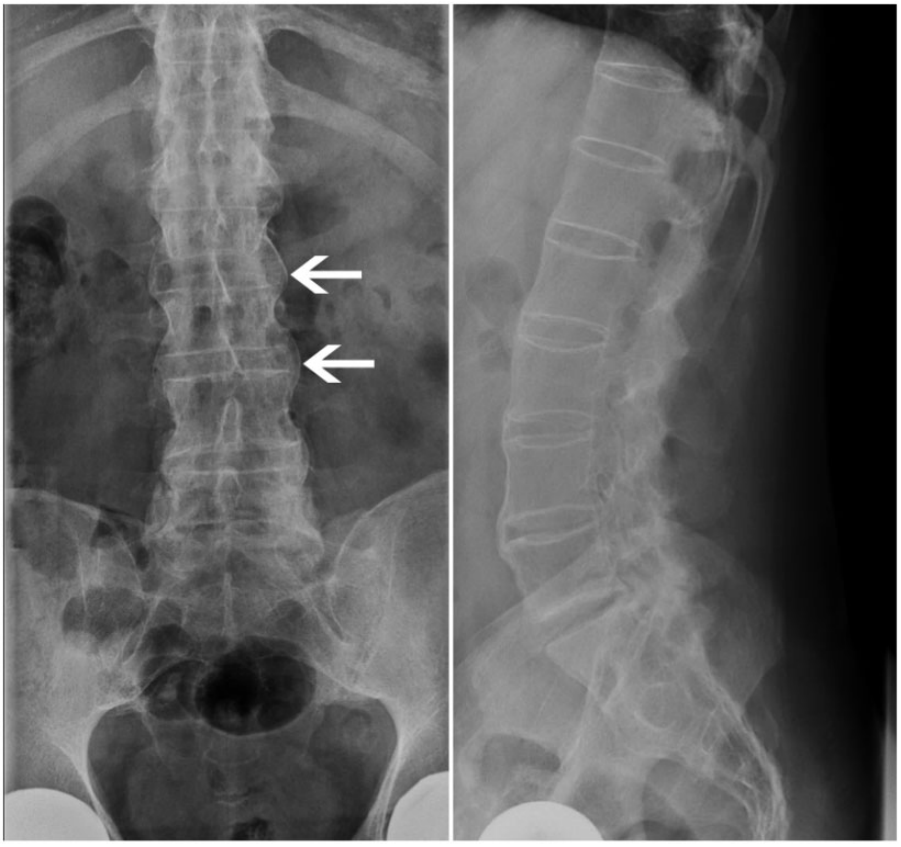

**Figure 2.** Radiographic findings of axial SpA. Plain radiographs demonstrate bilateral sacroiliac joint fusion as well as thin (marginal), syndesmophyte formation throughout the lumbar spine (arrows) resulting in the formation of the "bamboo spine."

dosage of the adalimumab to weekly, resulting in clinical improvement (Figure 1(F)).

## Discussion

In recent years, the term spondyloarthropathy has largely been replaced by the disease categories of axial and peripheral SpA. This nomenclature emphasizes the inflammatory nature of the disease (hence the focus on SpA) and the knowledge that patients with axial versus peripheral joint involvement tend to have different genetic backgrounds (HLA-B27 association with axial disease) as well as clinical manifestations. Rheumatologic conditions that may present as axial SpA include ankylosing spondylitis (AS), psoriatic arthritis, inflammatory bowel disease associated arthritis, reactive arthritis, and undifferentiated SpA. In the last decade, revised classification criteria for axial SpA have been developed, as older criteria commonly relied upon X-ray evidence of disease that took many years to develop.[1,2] The Assessment of Spondyloarthritis International Society (ASAS) has developed new classification criteria which incorporate modern imaging techniques (such as magnetic resonance imaging, which is known to be more sensitive than plain radiographs for early involvement) as well as extra-articular manifestations, as part of the classification scheme. Examples of extra-articular manifestations in the new criteria include uveitis, HLA-B27 positivity, psoriasis, and inflammatory bowel disease.

Under the new classification criteria, axial SpA includes two subtypes: nonradiographic axial spondyloarthritis (nr-axSpA) and radiographic axial SpA (or AS). Both meet the criteria for axial SpA as proposed by ASAS, except the former does not have radiographic evidence of sacroiliitis. The new classification of nr-axSpA allows for earlier diagnosis and treatment of patients with evidence of disease but without classic radiographic findings. However, there is ongoing debate concerning whether these subtypes are separate entities or a spectrum of the same disease.

Studies have shown similarities and differences between these two groups. The estimated prevalence of the two subtypes is similar at 0.35% based on retrospective data from U.S. rheumatology practices.[3] Other similarities shared between the two subtypes include genetic predisposition (HLA-B27), presence of peripheral arthritis and extra-articular manifestations, and patient-

reported quality of life scores.[4,5] In addition, similar outcomes and adherence to anti-tumor necrosis factor (TNF) therapy has also been reported in the two subtypes.[6] This strengthens the postulate that the two subtypes may represent different phases of the same disease. In contrast, patients with nr-axSpA are younger, more often female, have lower levels of C-reactive protein, and have milder disease as defined by clinician-derived global assessment scores, compared to AS.[4–6] These data suggest that nr-axSpA may be a separate and milder disease than AS. Additional study of nr-axSpA is needed to further define this subgroup of patients and determine its relationship to radiographic axial SpA.

The treatment of axial SpA includes various nonpharmacologic and nonbiologic therapies as first-line treatment. A recent meta-analysis showed that regular exercise was associated with small improvements in disease activity, and NSAIDs use was linked with improved symptoms and decreased radiographic progression compared to placebo.[7] Biologic disease modifying antirheumatic drugs, including TNF inhibitors and anti-IL17 directed therapies (secukinumab), represent good treatment options for patients with an inadequate response to NSAID monotherapy. TNF inhibitors are effective for both subtypes of axial SpA, with a number needed to treat to achieve an ASAS40 response ranging from 2.6 to 5.2 for AS and 2.3 to 5.4 for nr-axSpA.[8]

In our case, the patient demonstrated many of the criteria outlined by the ASAS group and was therefore classified as axial SpA. The presence of bilateral sacroiliac joint fusion, bilateral, marginal (thin) syndesmophytes, as well as the absence of known inflammatory bowel disease, preceding infection, nor psoriasis, aligns best with a traditional diagnosis of AS. The presence of cutaneous disease (specifically keratoderma blenorrhagicum versus PPP) raised the question of underlying reactive arthritis or psoriatic arthritis, respectively. However, these conditions classically lead to unilateral sacroiliac joint involvement as well as unilateral, thick syndesmophytes ("jug-handle" syndesmophytes). In addition, no clear preceding infection could be identified in our case.

Ultimately, the cutaneous findings in this case were strongly suspected to be associated with the underlying diagnosis of axial SpA. This clinical suspicion was supported by the remarkable cutaneous response to anti-TNF therapy, despite being recalcitrant to numerous topical approaches. Patients with AS are not commonly reported to have associated cutaneous manifestations, but it is important to recognize that SpA are a collection of rheumatologic diseases, often with shared, but overlapping clinical features.

This case highlights the importance of a thorough history and examination in patients with refractory dermatitis and reminds the clinician to reconsider the diagnosis when the response to therapy is not as expected. Taking the time to outline prior treatment failures and exposure history, documenting a history of uveitis, and observing examination findings suggestive of musculoskeletal disease, all led to the correct diagnosis for this patient. As Sir William Osler astutely pointed out over 100 years ago, "Always listen to the patient, they might be telling you the diagnosis."

## Clinical Implications

Consider other diagnoses in the differential of atopic and allergic contact dermatitis if the patient is not responsive to conventional therapies.

## Ethical Approval

Not applicable, this is a de-identified case report consisting of a case description without intervention nor data collection for research purposes.

## Statement of Human and Animal Rights

This article does not contain any studies with human or animal subjects.

## Statement of Informed Consent

There are no human subjects in this article and informed consent is not applicable.

## Declaration of Conflicting Interests

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