ONLINE EXCLUSIVE

Pruritus since childhood

The itchy rash had been previously diagnosed as psoriasis. But treatment provided minimal relief. So what was causing the itch?

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A 48-YEAR-OLD WOMAN experiencing homelessness presented to our clinic with a 4-week history of an intensely pruritic rash on her upper back and bilateral upper extremities. She reported that she had experienced exacerbations and remissions of the rash in similar locations for the past several years and during childhood. Factors that exacerbated the rash included being outdoors and being exposed to heat. Her pruritus was intensified by scratching the skin and was significantly worse at night. Previous doctors had diagnosed her with psoriasis and prescribed a short trial of hydrocortisone cream and oral antihistamines, but they provided minimal relief.

The patient indicated that the itching interrupted her sleep and her skin's appearance made it difficult to get a job. The physical exam revealed excoriated and erythematous papules and patches on her upper back, the extensor and flexor aspects of her bilateral forearms, and the dorsal surface of her bilateral wrists, hands, and fingers (**FIGURE 1**). Her skin was dry and scaly with pigmentary changes and skin thickening (**FIGURE 2**). She denied any other systemic symptoms. Her hair and nails were normal, she had no palpable lymph nodes, and she was afebrile. She reported suffering from seasonal allergies, but wasn't aware of a family history of skin disorders.

- WHAT IS YOUR DIAGNOSIS?
- HOW WOULD YOU TREAT THIS PATIENT?

FIGURE 1
Excoriated, erythematous papules and patches on arm

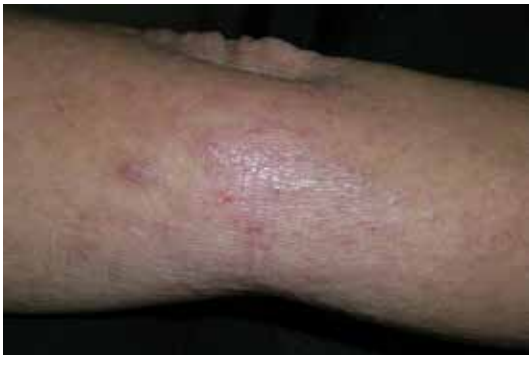
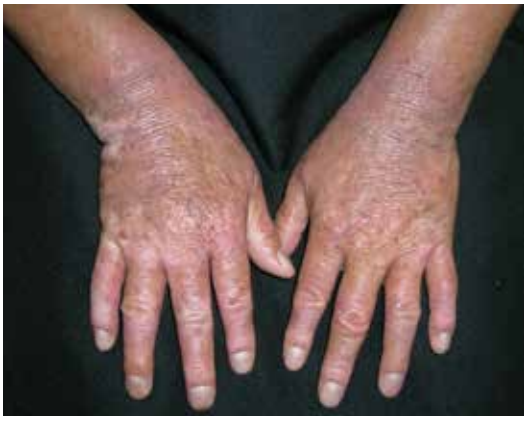


FIGURE 2
Lichenification and hyperpigmentation of hands



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Approximately 70% of patients with atopic dermatitis have a positive family history of an atopic disease, such as eczema, asthma, or allergic rhinitis.

Diagnosis: Chronic atopic dermatitis

Although the patient was told she had psoriasis by previous doctors, we diagnosed her condition as atopic dermatitis based on its clinical appearance. There is no single test that can establish a diagnosis of atopic dermatitis. While serum total IgE levels are often elevated, testing is not currently recommended.

The United Kingdom working group on atopic dermatitis published diagnostic criteria based on clinical history and physical exam that include pruritic skin in addition to the presence of 3 or more of the following: skin crease involvement, chronically dry skin, symptom onset before 2 years of age, and visible evidence of dermatitis involving flexural surfaces.[1] Our patient fulfilled all but one condition, as she wasn't sure if her symptoms began before age 2.

Atopic dermatitis is a chronic and inflammatory cutaneous disease that affects approximately 10% to 12% of children and less than 1% of adults in the United States.[2] Approximately 90% of cases present before the age of 5 and the literature demonstrates a slight female predominance.[3,4]

Disease severity is classified as mild, moderate, or severe.[5] Mild disease is characterized by dry skin and minimal itching with little impairment of the patient's physical and psychological wellbeing. Moderate disease includes frequent pruritus and erythema with or without secondary skin changes and a moderate impact on physical and mental health. In severe disease, extensive secondary skin changes exist and the patient's daily activities, sleep, and mental health may be severely impaired.

Etiology is multifactorial. Causes of atopic dermatitis include abnormalities in the epidermal stratum corneum and tight junctions, a heightened type-2 helper T-cell response to environmental antigens, innate immunity defects, and altered microbial skin flora.[6,7]

Genetic influences appear to play a substantial role in disease development. Approximately 70% of patients have a positive family history of an atopic disease such as eczema, asthma, or allergic rhinitis.[8] Genetic defects are believed to be related to defective proteins and lipids in the epidermis that lead to disruption of the epidermal barrier and subsequent cutaneous inflammation.[6,7]

Clinical presentation: Lesion distribution varies with age

Intense pruritus and dry scaly skin occur in both children and adults, although the distribution of lesions may vary with age. Children typically exhibit erythematous patches with papules and crusting on the face, scalp, extremities, or trunk. In adults, lesions are primarily located on the hands and feet, but may also present on the face, wrists, forearms, and flexural areas.[3]

Adults also frequently present with secondary skin changes such as thickened skin, pigmentation changes, lichenification, and excoriated papules due to chronic rubbing or scratching.[3] Our patient presented with significant lichenification and hyperpigmentation of the skin that was most prominent on the wrists and forearms.

Additional clinical features consistent with atopic dermatitis include a personal history of allergic conditions and a disease course characterized by exacerbations and remissions. Exacerbations may be caused by heat exposure, dry climates, anxiety, rapid temperature variations, contact with certain chemical substances, or microbial infections.[8]

Differential Dx includes psoriasis and scabies

The differential diagnosis of chronic atopic dermatitis consists of allergic or irritant contact dermatitis, plaque psoriasis, seborrheic dermatitis, scabies, and drug eruptions. Early diagnosis of atopic dermatitis is imperative to prevent sleep disturbances, chronic secondary skin changes, scarring, and the development of skin infections.

Allergic or irritant contact dermatitis is a cutaneous inflammation occurring after contact with an allergen or irritant. The lesions include erythematous, scaling areas with marked borders that are commonly pruritic. Acute cases often present with vesicles and bullae, while lichenification with cracks and fissures are common among chronic cases. Patch testing may be performed if the diagnosis is suspected.

Plaque psoriasis is characterized by areas of dry, erythematous, and well-demarcated plaques with silver scales that are most commonly found on the knees, elbows, scalp, and lower back. Systemic manifestations can include joint pain and joint swelling. Nail pitting and onycholysis are also common. While our patient had skin thickening, it was from the lichenification that is common in atopic dermatitis.

Psoriasis and atopic dermatitis are often confused. Psoriasis has discrete plaques on extensor surfaces and is often associated with nail changes, while the thickening of the skin that comes with chronic itch and scratching of atopic dermatitis is often less well circumscribed and found on flexor surfaces. Family physicians are frequently the first to encounter patients with atopic dermatitis and psoriasis and must be able to distinguish these conditions, as their treatments differ.

Seborrheic dermatitis is a chronic, relapsing inflammatory condition characterized by pruritic, erythematous, greasy, scaly patches on sebum-rich skin such as the scalp, face, and upper trunk. Seborrheic dermatitis is a clinical diagnosis.

Scabies is a pruritic skin condition caused by *Sarcoptes scabiei* var *hominis*. Characteristic linear burrows often appear as serpiginous, gray, threadlike elevations in the webbed spaces of the fingers, scrotum, areolae, elbows, axillae, feet, and wrist flexors. Secondary lesions from scratching or inflammation include excoriations, erythema, and hyperpigmentation. The diagnosis is made clinically and confirmed by dermoscopy, when available. Alternatively, mites or eggs may be observed on skin scrapings using light microscopy.

Drug eruptions should be considered in individuals taking medications who develop acute, symmetric cutaneous eruptions that may be morbilliform, urticarial, papulosquamous, pustular, or bullous in nature.

Treatment depends on severity, area of involvement, and patient's age

Components of atopic dermatitis treatment include skin hydration, negative stimuli avoidance, pharmacologic modalities, and patient education. Improved skin hydration can be achieved by applying thick emollients containing little to no water at least twice daily and after bathing.

Topical corticosteroids are added when emollient use alone fails. Potency selection is based upon the patient's age, involved body region, and the severity of skin inflammation.[8] In order to reduce cutaneous atrophy, only low-potency corticosteroids should be applied to the face, groin, and axillae. Patients with mild disease may apply desonide 0.05% or hydrocortisone 2.5% cream or ointment once or twice daily for 2 to 4 weeks.[8] Patients without improvement or with moderate disease may need medium- to high-potency steroids such as fluocinolone 0.025% or triamcinolone 0.1%.

Patients with atopic dermatitis on the face, eyelids, neck, and skin folds or those who do not obtain relief from combined emollients and topical corticosteroids may benefit from topical calcineurin inhibitors such as pimecrolimus or tacrolimus.[9] However, these agents should be utilized only for short periods of time and avoided in immunocompromised patients and those younger than 2 years of age.[9]

Patients with uncontrolled moderate to severe refractory disease may consider a trial of phototherapy or cyclosporine if phototherapy is ineffective or unavailable.[10] A meta-analysis has shown that once remission is achieved, intermittent therapy with moderate- to high-potency corticosteroids or tacrolimus may be effective in reducing subsequent flares.[11]

In all cases, sedating antihistamines such as diphenhydramine or hydroxyzine can be utilized for pruritic relief, particularly at night. Additionally, signs suggestive of infection should prompt antibiotic treatment with agents that provide coverage for *Staphylococcus* and *Streptococcus* species. Lastly, patients must be adequately educated on stimuli avoidance (eg, hot water, wool) and counseled on the medical and psychological issues that often accompany atopic dermatitis.

Our patient was placed on triamcinolone 0.1% for 4 weeks and her condition improved. JFP

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Exacerbations of atopic dermatitis may be caused by heat exposure, anxiety, rapid temperature variations, contact with certain chemical substances, or microbial infections.

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