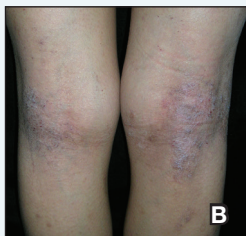


Atopic Dermatitis: New Insights and Expanded Treatment Options

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A Atopic dermatitis flare on the wrist in a 24-year-old man of African descent that occurred during work with cement.

B Atopic dermatitis with lichenification on the popliteal fossae in a 20-year-old Hispanic woman demonstrating hyperpigmentation.

C Atopic dermatitis with postinflammatory hypopigmentation in an infant of African descent. The hypopigmented patch on the wrist likely was exacerbated by topical steroid use. Additional faint patchy hypopigmented areas may represent associated pityriasis alba.

Photographs courtesy of Richard P. Usatine, MD.

Atopic dermatitis (AD) is a chronic skin condition generally characterized by pruritic and erythematous papules and plaques.¹ While AD commonly manifests in childhood, 1 in 4 patients living with AD report adult onset of the disease.² The clinical presentation and prevalence of AD vary across age groups, skin tones, and racial and ethnic groups. Globally, AD is estimated to have a prevalence of 2.6%; however, rates vary widely by region.¹ Morphology and distribution of AD lesions also vary by population; therefore, defining one classic presentation of AD is not sufficient in diverse patient populations.³

Epidemiology

The prevalence of AD ranges from 0.2% to 24.6% worldwide, with higher rates in Africa and Oceania and lower rates in India and Northern and Eastern Europe.¹ In the United States, AD affects all racial and ethnic groups; however, prevalence and severity are increased in Black children compared with White children.⁴ In one prospective cohort study, Hispanic children and non-Hispanic Black children aged 3 years and younger had greater odds of AD persisting into mid-childhood (approximately age 7 years) compared with non-Hispanic White children.^{5,6}

Key Clinical Features

Clinical features of AD are heterogeneous and may include differences in color, morphology, and distribution. Brown, hyperpigmented, gray, and/or violaceous plaques may predominate in patients with skin of color (SOC) compared with the erythematous plaques commonly described in lighter skin tones.^{1,3} Established scoring systems for AD rely on erythema as a key diagnostic feature, but because erythema can be difficult to detect in darker skin tones, disease

severity may be underestimated and diagnosis may be delayed in this population.⁴

Atopic dermatitis in SOC may manifest as lichenoid plaques,⁷ prurigo nodules,^{7,8} lichenification,¹ and follicular accentuation.⁹ Lichen planus–like AD is a distinct variant characterized by lichenoid plaques with a predilection for the extensor surfaces and face in patients with darker skin tones^{1,8} occurring in approximately 9% of patients in one study.¹⁰

Other key clinical features of AD in patients with SOC include pityriasis alba,¹⁰ increased risk for postinflammatory pigment alteration (including hyperpigmentation and/or hypopigmentation),¹ and greater trunk and extensor involvement.^{1,11}

Worth Noting

The scientific landscape for AD has grown rapidly, increasing our understanding of its pathophysiology, treatment, and social impact. Nonsteroidal treatments available for pediatric and adult patients with AD have increased in recent years, including crisaborole (approved for use in those ages ≥ 3 months), tacrolimus (≥ 2 years), and pimecrolimus (≥ 2 years). Injectable options include dupilumab (≥ 6 months), lebrikizumab (≥ 12 years), nemolizumab (≥ 12 years), and tralokinumab (≥ 12 years). Oral options include abrocitinib (≥ 12 years) and upadacitinib (≥ 12 years).¹² Topical options include roflumilast 0.15% cream (≥ 6 years)¹² and 0.05% cream (≥ 2 -5 years),¹³ ruxolitinib 1.5% cream (≥ 2 years),¹⁴ and tapinarof 1% cream (≥ 2 years).¹²

For some patients, postinflammatory pigment alteration associated with AD has a higher impact on quality of life than the AD itself.⁷ In a study of 260 US adults with AD, the emotional impact of pigmentary changes was greatest in Black patients, with 53.3% reporting that pigment changes bothered them “a lot” or “very much.”¹⁵

Genome-wide association studies have not identified a single determinant that explains racial and ethnic differences in susceptibility to AD.⁴ Instead, social determinants of health are thought to play a role in the difference in AD prevalence and severity across groups in the United States.¹⁶

Health Disparity Highlight

In an analysis of 20 US metropolitan cities, urban and inner-city residence was associated with approximately 1.7-fold increased odds of AD.⁴ Among pediatric patients with moderate to severe AD, Black children were more likely to be exposed to tobacco smoke¹⁷ and traffic-related air pollution.¹⁸ Low socioeconomic status and low income also have been associated with moderate¹⁶ and severe¹⁹ AD. At the same education level, Black individuals in the United States receive less income than their White counterparts and have markedly less wealth at equivalent incomes.²⁰

In utero exposure to maternal stress is associated with AD.⁴ Increased IgE levels have been recorded in children who develop AD, with Black children having the highest IgE levels overall compared to other children.¹⁸

An analysis of medical records from an urban medical center in Baltimore, Maryland, from 2013 through 2018 showed that Black patients with AD were less likely to receive topical corticosteroids, topical calcineurin inhibitors, a topical phosphodiesterase 4 inhibitor, and a biologic compared to White patients with AD.²¹

Since the disproportionate burden experienced by patients with AD is not physiologic, it is imperative to address these systemic complexities and address the barriers impacting treatment availability to improve health outcomes for all patients living with AD.

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