Clinical Images

Presentations of Cutaneous Disease in Various Skin Pigmentations: Chronic Atopic Dermatitis

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Abstract

Description

Atopic dermatitis is a chronic inflammatory skin disorder classically affecting flexural areas of the body. It is present in children and adults, including those with darker skin pigmentation. Chronic lesions are hyperpigmented plaques that are dry, cracked, and/or scaly often with lichenification. Differential diagnoses include psoriasis, seborrheic dermatitis, ichthyosis, and pityriasis rosea. This article will showcase clinical images with varying presentations of chronic atopic dermatitis in a range of age groups and skin colors according to the Fitzpatrick scale.

Keywords

atopic dermatitis; hyperpigmentation; skin pigmentation; skin of color; Fitzpatrick scale; genetic skin diseases; eczema

Introduction

Atopic dermatitis (AD), commonly known as eczema, is a chronic inflammatory skin disorder.[1] AD can affect any age group but commonly develops during infancy and early childhood.[2] Chronic lesions are clinically characterized as hyperpigmented plaques (raised, >1 cm) that are dry, cracked, or scaly, often with lichenification.[1,3] Morphological subtypes of AD can exist due to varying skin tones, as characterized by the Fitzpatrick scale (**Figure 1**).[3] Additional background information on the Fitzpatrick scale and a description of the classification of skin types are discussed in further detail in the article "Presentations of Cutaneous Disease in Various Skin Pigmentations: An Introduction".[4]

Case Presentation

Figure 2 shows a 40-year-old man with Fitzpatrick type I (always burns, never tans) skin. His case demonstrates chronic AD along the flexural surfaces around his knees and his face, com-

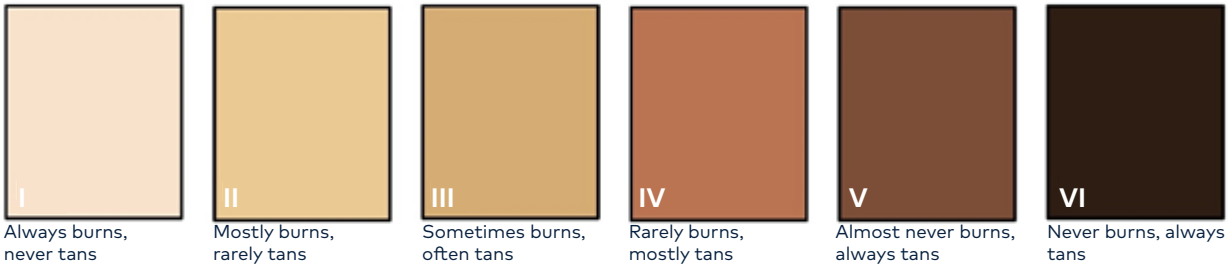

Figure 1. The Fitzpatrick scale provides a classification system for an individual's skin type based on the ability to burn and/or tan when exposed to ultraviolet light. It is used to approximate the degree of skin pigmentation.

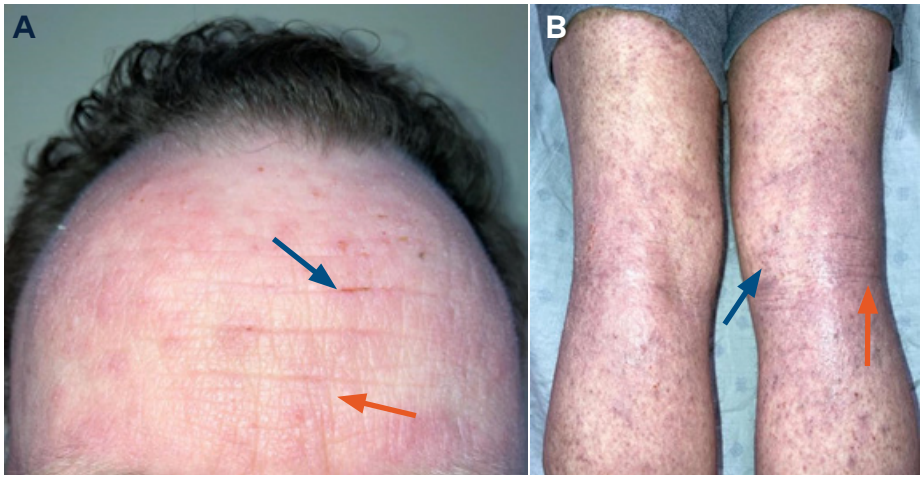

Figure 2. Fitzpatrick I (always burns, never tans). A 40-year-old man demonstrating chronic atopic dermatitis. There are poorly defined papules (raised, <1 cm) and plaques on the (**A**) face and (**B**) flexural regions of the popliteal fossa. Erythema is easily seen as bright pink and red. Erosions and serous crusting are scattered throughout. Chronic features of lichenification (orange arrows) are evident as accentuated skin lines on plaques of the face and popliteal fossa. The whole forehead is lichenified except for a small area of sparing on the superior mid aspect. Linear erosions (blue arrows) consistent with excoriation (superficial skin abrasion secondary to scratching) illustrate the pruritic nature of this condition. (**B**) Background sun damage, consisting of mottled hyper- and hypopigmentation with telangiectasias, is shown.

mon locations for this eruption to be found. Additionally, the erythema which is commonly associated with AD is very easily appreciated on his light skin tone. His lesions are overall poorly-defined, and it is difficult to clearly distinguish where the lesions start and end. The lesions demonstrate common characteristics seen in AD, such as areas of raised papules and plaques, as well as areas of serous crusting (dried exudate). The chronicity of his disease is demonstrated with the lichenification, or thickening, of the lesions, especially seen along the skin lines of his forehead. Lastly, the pruritic nature of the disease is demonstrated with the linear configuration of erosions (skin loss involving the epidermis only) across his forehead. The patient also has evidence of sun damage, with areas of both hyper- and hypopigmentation and telangiectasias, which are unrelated to the eruption of AD.

Figure 3 demonstrates a 13-year-old boy with Fitzpatrick type III (sometimes burns, often tans) skin. In this case, the AD is localized around his eyes, which is common in children. Once again, the erythema is easy to appreciate on his light skin tone. Additionally, areas of excoriation and lichenification can also be seen on

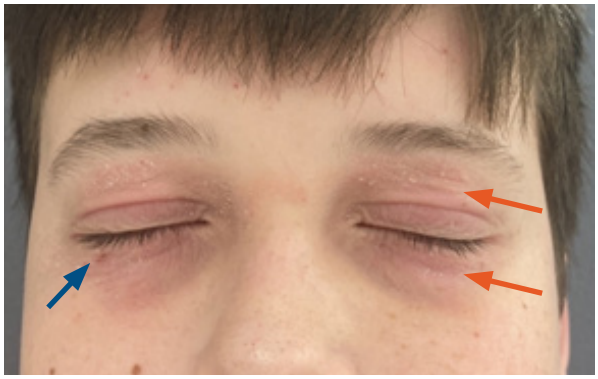

Figure 3. Fitzpatrick III (sometimes burns, often tans). A 13-year-old boy demonstrating chronic atopic dermatitis around the periorbital skin. The erythema is dark pink and red in this skin type. There are poorly defined scaly plaques. Areas of excoriation (blue arrow) and chronic features of lichenification are evident on plaques of the upper and lower eyelids (orange arrows). The accentuated lower eyelid creases are consistent with Dennie-Morgan lines (a minor criterion for diagnosis of atopic dermatitis).

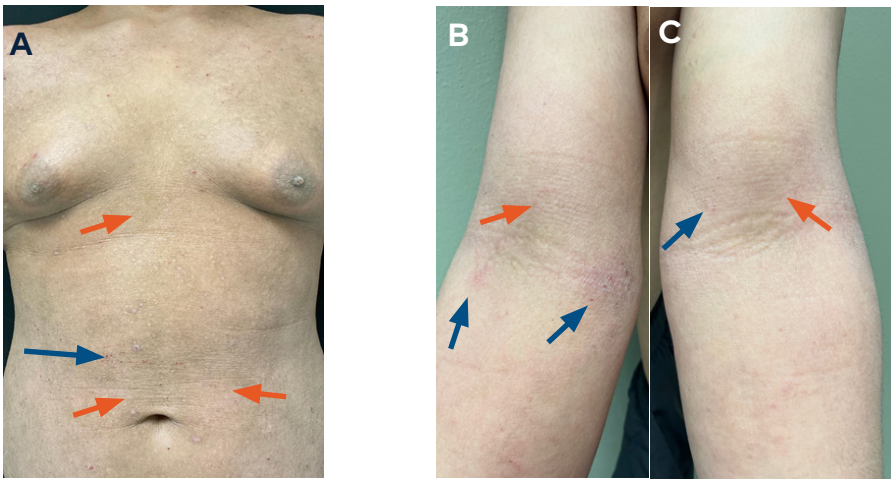

Figure 4. Fitzpatrick IV (rarely burns, mostly tans). (**A**) A 21-year-old man demonstrating chronic atopic dermatitis across the chest and abdomen. (**B** and **C**) A 14-year-old boy with lesions on the antecubital fossae. Follicular-based scaly papules can be appreciated. Chronic features can be seen with diffuse lichenification (orange arrows). There is scattered hypo- and hyperpigmentation throughout. In this darker skin type, minimal pink erythema is observed over hypopigmented areas but is undetectable over hyperpigmented skin. Areas of excoriation can also be seen (blue arrows).

both the upper and lower eyelids. Dennie-Morgan lines, which are commonly associated with AD and included as a minor criterion for its diagnosis, are also appreciated in these images as accentuated lower eyelid creases.

Figure 4A demonstrates a 21-year-old man with chronic AD across his chest and abdomen. **Figure 4B** and **C** demonstrates a 14-year-old boy with chronic AD in his antecubital fossa. Both of these patients have Fitzpatrick IV (rarely burns, mostly tans) skin, which causes the erythema to be less evident than in previous cases. Areas of hyper- and hypopigmentation can be appreciated in these cases, which is a common finding in patients of darker skin tones. Additionally, as in previous cases, chronic features of AD can be seen with lichenified skin in affected areas.

Figure 5 shows a 3-year-old male with Fitzpatrick type V (almost never burns, always tans)

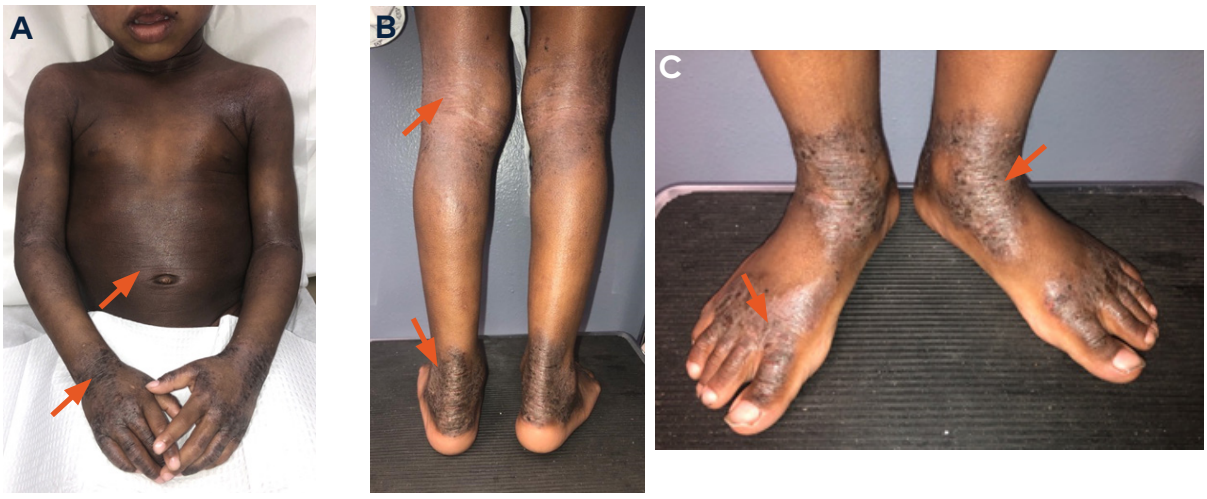

Figure 5. Fitzpatrick V (almost never burns, always tans). A 3-year-old male demonstrating chronic atopic dermatitis on the neck, chest, abdomen, antecubital and popliteal fossae, dorsum of the hands, feet, and flexures of the wrists and ankles. Follicular-based lesions, scaly papules, and plaques are seen on the (**A**) mid-abdomen, (**B**) legs, and (**C**) feet. There is prominent dark brown hyperpigmentation, and erythema is not apparent. Pronounced and widespread hyperpigmented lichenification is seen (orange arrows), demonstrating the chronicity typical in this stage of atopic dermatitis. Erosions consistent with excoriation illustrate the pruritic nature of this condition.

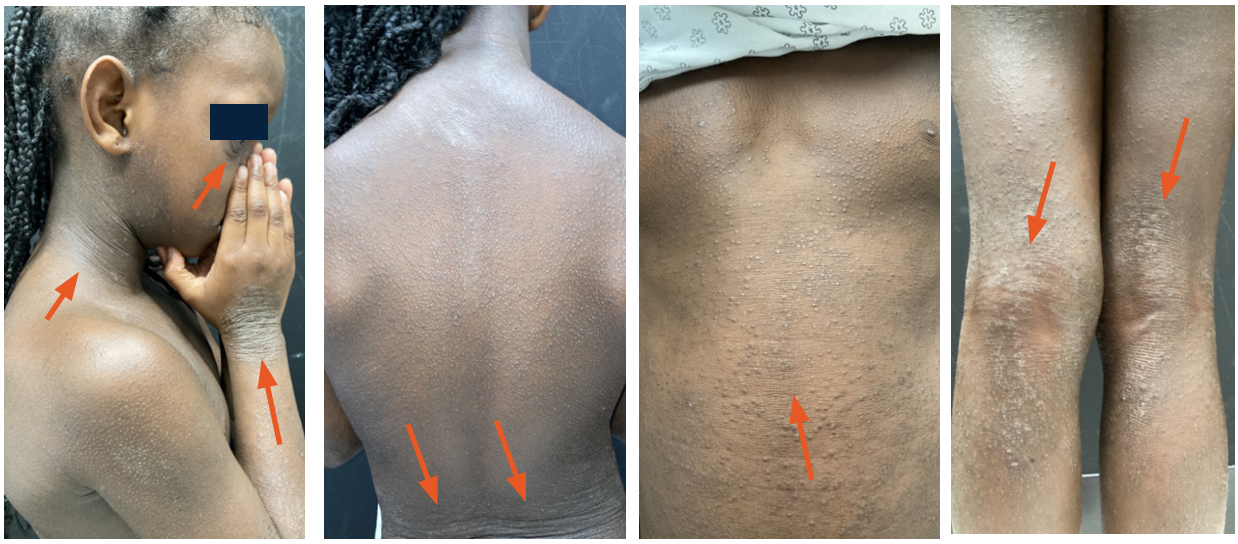

Figure 6. Fitzpatrick VI (Never burns, always tans). A 6-year-old female demonstrating diffuse chronic atopic dermatitis on the face, neck, back, abdomen, and popliteal fossae. Similar to **Figure 5**, follicular-based, scaly papules and plaques are noted throughout with prominent dark brown hyperpigmentation and lichenification (orange arrows). There is prominent dark brown hyperpigmentation, and erythema is not apparent.

skin. The images demonstrate chronic AD with lesions diffusely across his body. This case illustrates the dark hyperpigmentation that becomes very common with AD in darker skin tones. Additionally, erythema is not apparent at all. Widespread lichenification demonstrates the chronicity of his condition, and erosions with excoriations showcase the pruritic features of AD.

Figure 6 shows a 6-year-old female with Fitzpatrick type VI (never burns, always tans) skin and diffuse AD across her body. Similarly to the previous case, the erythema is not present, and the lesions instead present with hyperpigmentation. Additionally, the lesions are more follicular-based, which is commonly seen in darker skin tones. Lichenification can also be appreciated in these images.

Table 1 provides a summary of common differential diagnoses for chronic atopic dermatitis.

Figure 7 demonstrates a young adult man with Fitzpatrick type IV (rarely burns, mostly tans) skin. His case demonstrates seborrheic dermatitis across his face. While the erythematous scaly papules can resemble AD, the scale that accompanies seborrheic dermatitis is often thick, greasy, and yellow compared to the lighter, white, and fine scale that accompanies AD. Additionally, seborrheic dermatitis is

Table 1. A Summary of Common Differential Diagnosis of Chronic Atopic Dermatitis.

Condition	Morphology	Distribution	Other symptoms
Seborrheic dermatitis	Salmon-colored papules and plaques with yellow scale	Scalp, face, skin folds	Pruritus Post-inflammatory pigmentary changes
Ichthyosis vulgaris	Polygonal scale	Generalized	Associated with atopic dermatitis
Psoriasis	Erythematous plaques with silvery scale	Scalp, extensor surfaces	Pruritus Post-inflammatory pigmentary changes Nail pitting
Pityriasis rosea	Isolated plaque with central clearing and peripheral scale	Trunk	Pruritus Self-resolving

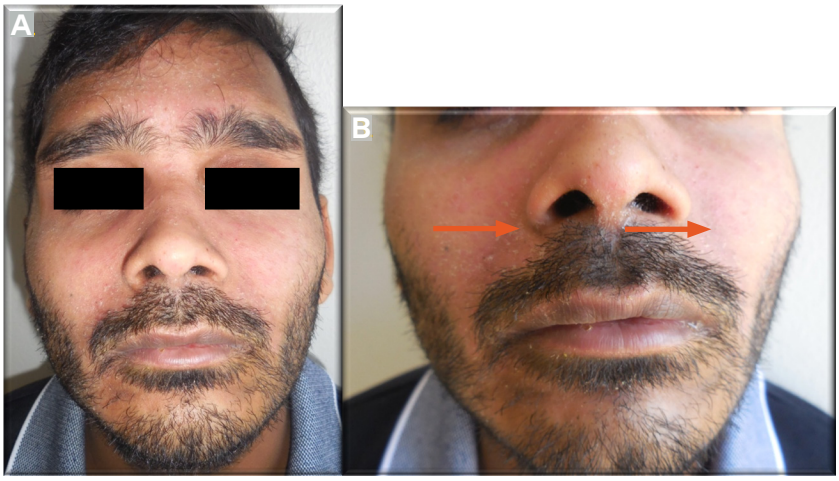

Figure 7. Fitzpatrick IV (rarely burns, mostly tans). (**A**) A young adult man with seborrheic dermatitis on the central face and forehead. Faint erythema and fine scales can be seen. (**B**) Scaling is appreciated on the bilateral nasolabial folds (orange arrows), involving the mustache and beard on the upper lip, and cheeks, and chin.

classically distributed on areas of skin that are rich in sebaceous glands (eg, scalp, eyebrows, and nasolabial folds), rather than in the flexural areas of the upper and lower extremities more characteristic of AD.

Figure 8 illustrates a 7-year-old girl with Fitzpatrick type IV (rarely burns, mostly tans) skin. The images demonstrate ichthyosis vulgaris along her lower extremities. Ichthyosis vulgaris is a minor criterion of and is often associated with AD, but in itself can be an isolated finding that does not necessitate the diagnosis of AD.

Figure 9 demonstrates two different patients with Fitzpatrick type II (mostly burns, rarely tans) skin. The lesions in the image illustrate plaque psoriasis, which shares signs and symptoms with AD. Lesions also appear as erythematous scaly papules and plaques. However, these lesions are very well-demarcated with distinct borders. Additionally, the quality of the scale in psoriasis, classically described as silvery scale, can be easily appreciated in these images and differs from the scale of AD.

Figure 10 shows a 14-month-old boy with Fitzpatrick type III (sometimes burns, often tans) skin. His images demonstrate pityriasis rosea, another common condition that can present similarly to AD. The erythematous scaly papules and plaques are easily appreciated on his

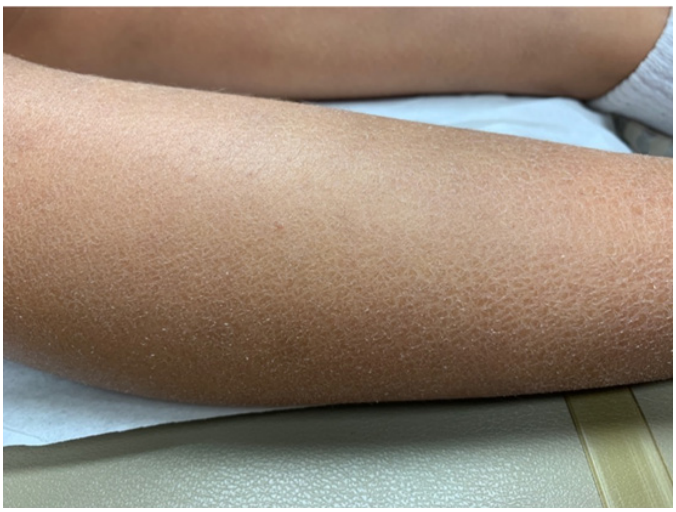

Figure 8. Fitzpatrick IV (rarely burns, mostly tans. A 7-year-old female with quadrilateral scales (often called fish scales) demonstrating ichthyosis vulgaris on the extensor surface of her lower extremities. Ichthyosis vulgaris is a minor criterion for diagnosis of atopic dermatitis.

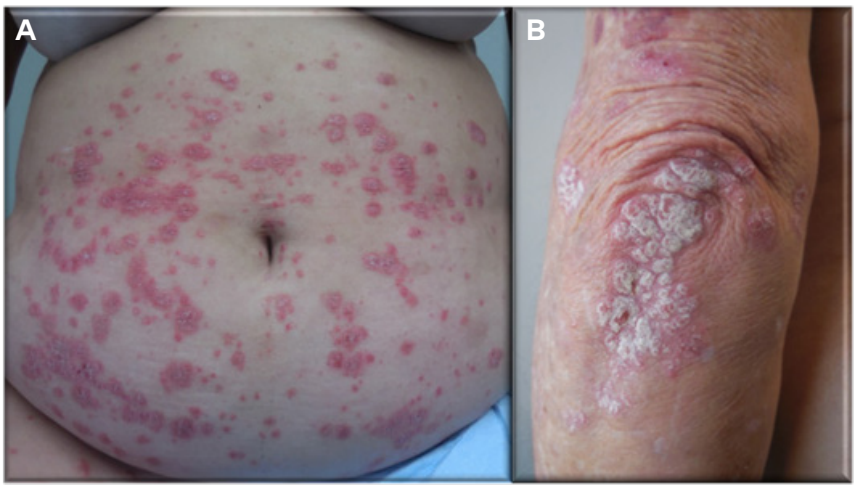

Figure 9. Fitzpatrick II (mostly burns, rarely tans). Plaque psoriasis in 2 separate patients. (A) Multiple erythematous, pink or red-colored papules and plaques are seen on the abdomen. The lesions are well demarcated (distinctly different from the adjacent normal skin where the border is readily identified). (B) Similar lesions, with micaceous (silvery, white, thick) scales are seen on the elbow.

skin. The key differentiating factor for this condition is the distribution, demonstrated here with lesions following skin tension lines and classically described as a "Christmas tree" distribution.

Discussion

In the United States, AD affects approximately 11.3% to 12.7% of children and 6.9% to 7.6% of adults.[3,5] Most cases of AD manifest before the fifth year of life.[3,5] Adult-onset AD can arise due to changes in the environment, such as individuals who move from a humid, tropical climate to a temperate and higher latitude location.[1] Black and Asian patients more frequently have AD compared to White patients.[5] Children with darker skin are approximately 6 times more likely to develop AD in comparison to children with lighter skin.[6] More severe clinical presentations can be seen in patients with darker pigmentation due to the masking of erythema, causing late diagnosis or late referral to dermatology.[2]

Presentations of chronic AD vary due to age and color of skin. Chronic lesions are clinically characterized as hyperpigmented plaques that are dry, cracked, and/or scaly with lichenification.[1,3] Infants typically do not present with chronic AD as they express acute symptoms of

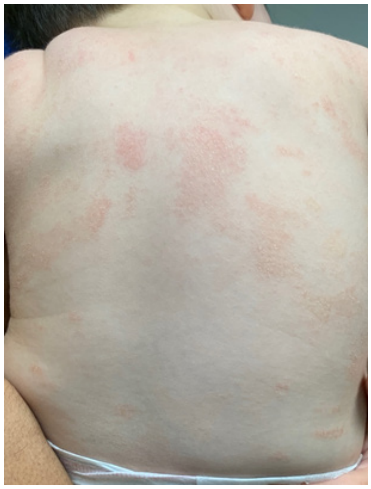

Figure 10. Estimated Fitzpatrick III (sometimes burns, often tans). A 14-month-old male with pityriasis rosea. There are multiple scattered erythematous, pink or red-colored papules and plaques seen on the back. The lesions have very fine, delicate scaling and follow a Christmas tree distribution (along skin tension lines).

erythema, edema, and exudative rashes located on the face, neck, scalp, trunk, and extensor extremities.[1,3,7] Children can present with chronic AD that is more localized, with less exudative and erythematous lesions, located on the neck, eyelids, and flexor surfaces. Repetitive scratching may also cause xerosis and thickening of skin, easily seen as accentuated skin lines, also called lichenification.[1,3] Adults with chronic AD present with lichenification developing on the hands, feet, nipples, eyelids, and flexural surfaces.[1] In darker-skinned individuals, AD can have a violaceous lichen planus-like presentation and involve the extensor areas.[2] Darker-skinned individuals may also have a high risk of developing post-inflammatory dyspigmentation, which can take months to subside and/or lead to permanent dyspigmentation with chronic excoriation.[7] Asian individuals can have more well-demarcated lesions with increased scaling and lichenification in comparison to White patients.[2] Follicular prominence (papules) is clinically observed in patients of African or Asian descent and Fitzpatrick skin types IV-VI.[3,6]

Most cases of AD have been linked to genetic factors and epidermal structures.[8] A family history of AD is the strongest known risk factor as it can increase a child's risk by 1.5-fold.[3] Multiple cases can be linked to a loss of function (LoF) mutation in the filaggrin gene (FLG), which codes for a skin barrier protein in the stratum corneum that prevents transepidermal water loss and skin infections.[1,5,9] FLG LoF causes defects within the skin barrier by breaking down the skin's natural moisturizing factor.[1,2] An impaired skin barrier can contribute to the pathogenesis of AD as it leads to skin inflammation and allergic sensitization.[5] Homozygous mutations in the FLG can manifest as severe and/or early onset AD with longer persistence and skin infections. Interleukins 4, 13, and 17 have been reported to decrease the expression of the FLG as well. In chronic AD, Type 1 T helper cells (Th1), Th22, and Th17 subsets are recruited due to tissue inflammation, which can further exacerbate epidermal thickening and abnormal keratinocyte proliferation.[5] Variants exist among different races, which can help explain the unequal prevalence and persistence in different racial groups.[9] For example, in comparison to Western populations, Japanese and Korean patients have a lower frequency of FLG mutations.[5] According to Alexis et al, lower rates of FLG mutations have been described among Black populations as well.[7] Therefore, the more severe clinical manifestations of AD in the Black and Asian population may not be due to an FLG mutation but a different factor. For example, a strong polarization of Th17 and Th22 is seen in the Asian population, leading to an increase in the prevalence of AD.[2]

AD can have a significant quality of life impairment and disease burden in diagnosed individuals, especially those with darker skin pigmentation.[1,3] It can progress to a systemic disorder called the "atopic march" where allergic conditions develop, including asthma, allergic rhinitis, and food allergies.[1,3] Pruritus, a hallmark symptom of AD, may lead to increased sleep disturbances, fatigue, and mental health symptoms, which ultimately affects growth, school performance, attention, and accident rates in children.[3] AD has been linked to attention-deficient/hyperactivity disorder in children and increased rates of depression and anxiety in teenagers and adults.[1,3] An advanced disease presentation of post-inflammatory dyspigmentation is observed more in Black patients in comparison to White patients due to decreased health care access and different clinical manifestations, notably with inapparent erythema.[2]

Differential diagnoses for chronic AD include seborrheic dermatitis, ichthyoses, plaque psoriasis, and pityriasis rosea. Seborrheic dermatitis (**Figure 7**) involves the scalp, face, and skin folds, which are the sebum-rich areas of the body, and it clinically presents with pruritus, salmon-colored papules and plaques, and greasy scale-crust.[10] Similar to chronic AD, seborrheic dermatitis can result in post-inflammatory dyspigmentation in individuals with darker skin colors, but the abnormal immune response is related to the Malassezia (formerly Pityrosporum) yeast.[10] Ichthyosis vulgaris (**Figure 8**) is an inherited skin condition with a fish scale (polygonal) appearance on extensor surfaces.[11] It is associated with early-onset AD due to loss of skin barrier function from a mutation in the FLG.[11] Plaque psoriasis (**Figure 9**) is a chronic inflammatory disease affecting the scalp and extensor surfaces, such as elbows and knees, with well-demarcated erythematous, silvery, and scaly plaques.[12] Psoriasis can lead to post-inflammatory hyperpigmentation in darker skin colors, like AD, but can also present with nail pitting and psoriatic arthritis, and is less pruritic than AD.[13] Telangiectasia and erythema are less prominent in patients with

darker skin types, and additional symptoms, such as burning and stinging, may be more evident of disease in these populations.[14] Pityriasis rosea (**Figure 10**) is a viral exanthema associated with human herpesvirus 6 and 7 infection. It presents with a primary isolated oval plaque, also known as a "herald patch," that has a central clearing and develops with a peripheral scale.[15] The initial lesion is followed by a secondary eruption of similar appearing lesions that develop along the cleavage lines of the skin on the trunk and extremities.[15,16] Similarly to chronic AD, the lesions of pityriasis rosea often present with pruritis; however, pityriasis rosea is self-resolving.[16] The initial herald patch exhibits scaling within the borders of the plaque and is often confused with tinea corporis, where scaling is located at the periphery of the plaque instead.[15] In addition to tinea corporis, secondary syphilis should be considered in the differential diagnosis.[15] To help differentiate AD from other etiologies, a thorough family history and temporal course of the lesions should be gathered. In cases where a clinical diagnosis is difficult to identify, skin biopsies for histologic examination may aid in diagnosis.

Conclusion

Hallmark features of chronic AD include pruritic lesions that appear as erythematous to hyperpigmented scaly plaques, often accompanied by lichenification and linear excoriations. The ability to accurately recognize and treat chronic AD in patients of all skin types allowing clinicians to provide excellent and equitable care to an increasingly diverse patient population.

Conflicts of Interest

The authors declare they have no conflicts of interest.

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