Evaluating skin of color (SOC) demands that clinicians avoid solely relying on visual inspection, which can lead to diagnostic error. For example, redness, one of the most obvious signs of cutaneous inflammation, is subtle, appears violaceous, hyperpigmented, or is not apparent in SOC. Erythema in all skin tones is evidence of inflammation, but in darker skin, so is hyperpigmentation. Several inflammatory conditions hallmarked by erythema, such as atopic dermatitis (AD), psoriasis, or pressure injuries (PI), may be misdiagnosed in darker skin because the clinical presentation is inconsistent with textbook descriptions. Moreover, some cancers may go undiagnosed.

Standards of care for skin disease need broadening to be inclusive of SOC. Clinicians must adapt their patient evaluation to account for differences in clinical presentation unique to darker skin. But, if the assessment misinforms the diagnosis or determination of disease severity, it is impossible to provide adequate patient care. Therefore, understanding and identifying the nuances between evaluating and diagnosing disease in darker and lighter skin is essential.

Aside from subtle erythema, the distinguishing features of AD in SOC include papular-perifollicular, extensor distribution, xerosis, Dennie-Morgan lines, palmar hyperlinearity, periorbital hyperpigmentation, prurigo nodularis, and lichenification. Most notably, the rash distributes across flexor instead of extensor surfaces in SOC, which could easily perplex a novice clinician establishing their diagnosis from classic presentations. Also, darker skin children are determined to be six times more likely to develop severe AD than lighter skin children when scoring tools that use skin redness to measure disease severity are excluded because these methods are ineffective in SOC. Psoriasis poses similar diagnostic challenges because hyperpigmentation may reflect active disease versus post-inflammatoratory skin changes in SOC, and plaque types vary across all skin tones: thick, scaly plaques are prevalent among Blacks/African Americans, while pustular plaques are common to Asians and Hispanic/Latinos. Lastly, life-threatening diseases in SOC pediatric populations are not excluded from the diagnostic pitfalls imposed by an underwhelming presentation in darker skin. An erythematous maculopapular rash characterizes Kawasaki disease (KD) and congenital measles (CM). However, the rash appearance is hyperpigmented in SOC, so other symptoms of KD may be more clinically apparent (e.g., conjunctivitis, strawberry tongue, Palmer erythema, cervical lymphadenopathy, and fissured lips). CM rash texture and maternal vaccination status may facilitate diagnosis.

Another diagnostic challenge in patients with SOC during the early stages are PIs. Complications associated with PIs can be fatal; therefore, prevention and early detection are essential strategies to minimize occurrence and progression. Prevention and early detection of PI are essential strategies to minimize occurrence and progression. Still, the initial evidence of PI is undetected in intensely pigmented skin, even among high-risk patients receiving anticipatory skin assessments. For example, relying on visibly red gradients to detect or grade an ulcer causes patients with darker skin to be undiagnosed, leading to more advanced wounds at diagnosis. Skin temperature, edema, induration, and pain are more relevant indicators for SOC. When ocular inspection fails, other medical technology, such as thermal imaging, can detect dermal damage before it is visible, potentially precluding disease and severity. Employing sensitive assessment methods enhances standards of care for PI in SOC.

Basal cell carcinoma (BCC) also presents differently in SOC compared to non-Hispanic White patients. Pearly papules with rolled borders and telangiectasias characteristic of basal cell carcinoma (BCC) are inconspicuous in SOC and twice as likely to be pigmented. Shao et al. report that cutaneous neoplasms, such as squamous cell carcinoma (SCC) and melanoma diagnosed in minorities, cause a higher number of deaths and are more advanced at diagnosis compared to cases among non-Hispanic Whites. SCC in SOC patients can develop in non-sun-exposed areas (lower legs, perianal, and genital), which may partially explain this disparity. The lesions are scaly brown or black hyperpigmented plaques, papules, or nodules.
and the perilesional appearance is hypopigmented, hyperpigmented, or mottled.15,16 SOC melanomas correlate with high-risk regional and distal metastatic sites (genital, perianal, ocular, oral, and acral)—hyperpigmented macule with a history of rapid or radial and vertical changes in growth phases.15-16 Risks for developing BCC and SCC in SOC include radiation, albinism, scarring, and immunosuppression.18 SCC that emerge secondary to an inflammatory scar metastasizes in 20% to 40% of ethnic minorities.16

United States demographics are changing fast, and the growth of minority patients as a collective is outpacing the current majority; the former will be 50% of the population by 2044.17 This population shift will amplify the current disparity: SOC patients endure higher rates of morbidity and mortality caused by dermatological disease because of delayed diagnosis or misdiagnosis.16 Prompt and accurate diagnoses mitigate poor clinical outcomes for SOC patients. Medical schools and training programs must increase the number of images that illustrate cutaneous disease presentations and lecturers specializing in SOC to prepare for the changing demographic so that healthcare professionals are better prepared to assess and diagnose conditions in darker skin.18

REFERENCES: