Skin of colour: essentials for the non-dermatologist

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ABSTRACT

Doctors-in-training often receive an inadequate dermatology education. Furthermore, studies have highlighted the under-representation of skin of colour (SOC) in dermatological teaching, learning resources and research. Our image-based questionnaire, distributed to all internal medicine trainees in southwest England, highlighted knowledge gaps regarding SOC among training physicians. It is intrinsically more challenging for clinicians to confidently formulate dermatological diagnoses in SOC. In this review, we provide guidance for physicians to help make the diagnostic process more straightforward. First, we outline how skin colour is determined and classified. We discuss how inflammation presents in SOC, with the typical 'erythema' that physicians often associate with inflammation being a less prominent feature in darker skin tones. We then summarise nine important conditions that we believe physicians working in all specialties should be able to identify in patients with SOC, covering both conditions encountered on the medical take and conditions disproportionately affecting individuals with SOC. The population of the UK is rapidly diversifying; thus, as physicians, we have a professional duty to educate ourselves on dermatological conditions in SOC to provide the best quality of care for all our patients, regardless of their skin type.

KEYWORDS: dermatology, skin of colour, diversity, education

DOI: 10.7861/clinmed.2022-0335

Introduction

Doctors-in-training often receive an inadequate dermatology education, with one study reporting that 83% of internal medicine trainees feel uncomfortable managing dermatology presentations on the ward.[1] Furthermore, research has highlighted the under-representation of skin of colour (SOC) in dermatological teaching, resulting in clinicians lacking confidence to safely diagnose and manage dermatological disease in patients with SOC.[2]

We investigated the effect that this might have on patient safety by distributing an image-based questionnaire on 10 dermatological conditions in white skin and SOC to all internal medicine trainees in southwest England. Common conditions, such as eczema, psoriasis and melanoma, were misdiagnosed by 100%, 80% and 60% of participants, respectively. Furthermore, participants were less likely to refer patients with SOC to Dermatology for further management, with only 25% of participants referring a drug rash in patients with SOC compared with 40% for patients with white skin.

Differences in the structure and function of pigmented skin mean that common dermatological conditions can appear significantly different in disparate skin tones. Importantly, the redness traditionally associated with inflammation in white skin is often masked by background pigmentation in SOC.[3] Instead, inflammatory lesions in SOC can appear brown, brown-red, grey, purple, blue or black.[3] There can also be changes to the texture and contours of the skin, in addition to other cardinal signs of inflammation, such as tenderness, oedema and warmth.[3] This makes it intrinsically more challenging for clinicians to confidently formulate dermatological diagnoses in SOC.

In this review, we provide guidance for physicians to help make the diagnostic process more straightforward. We begin by outlining how skin colour is determined and classified. We then summarise nine important conditions that we believe physicians working in all specialties should be able to identify in patients with SOC, covering both conditions encountered on the medical take and conditions disproportionately affecting individuals with SOC.

What determines skin colour and how is it classified?

Melanin is the key determinant of skin colour.[4] It is synthesised by melanocytes in the basal layer of the epidermis and is then packaged into melanosomes and transported into surrounding keratinocytes.[4] Skin pigmentation is influenced by the size and distribution of melanosomes. For example, individuals of African descent have larger melanosomes containing more melanin, which are dispersed throughout the epidermis and take longer to be degraded.[4] Additionally, melanocytes synthesise two types of melanin: eumelanin (the black-brown form) and pheomelanin (the red-yellow form). Individuals with darker skin have a greater proportion of eumelanin, whereas individuals with fairer skin have a greater proportion of pheomelanin.[4]

There are several systems that can be used to classify skin types, with the Fitzpatrick Skin Phototype Classification System remaining the most frequently used in dermatological practice. It was originally designed in 1975 to assess the propensity of white skin to burn during phototherapy and was later developed to categorise skin into six types based on the amount of melanin and its response to UV radiation.[5] In this review, the term 'skin of colour' correlates with Fitzpatrick skin types IV–VI. However, the

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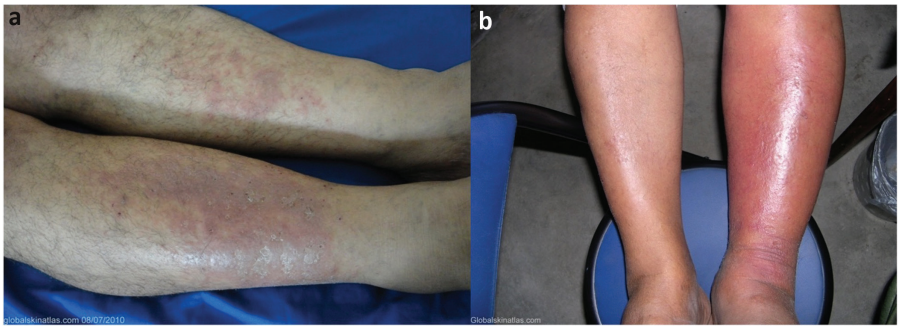

Fig 1. Venous eczema and cellulitis. (a) Venous eczema in skin of colour (SOC). (b) Cellulitis in SOC. Reproduced with permission from Global Skin Atlas (globalskinatlas.com).

Fitzpatrick system is undoubtedly flawed and has been criticised for many reasons, notably for its subjectivity and failure to account for the variation observed between individuals with moderately and darkly pigmented skin.[5] Alternative systems have been proposed, such as the recently developed Eumelanin Human Skin Colour Scale, which classifies individuals into one of five categories based on their 'melanin index' value.[6]

Important conditions to recognise in skin of colour

Eczema

Venous eczema

Venous eczema is seen in 20% of those aged over 70 and, therefore, is a frequent condition encountered on the medical take.[7] It is caused by venous insufficiency leading to fluid collection in tissues and subsequent inflammation.[8] Risk factors include: increasing age; a previous deep vein thrombosis; varicose veins; and chronic lower limb swelling.[8]

Venous eczema typically presents as pruritic, scaly plaques over the lower legs, which can appear red-pink in white skin in contrast to a more subtle brown, purple or grey discolouration in SOC (Fig 1a). Other features include: macular hyperpigmentation secondary to haemosiderin deposition; atrophie blanche (white scars); and an 'inverted champagne bottle' appearance secondary to lipodermatosclerosis.[8] Venous ulcers and secondary infection can also develop. Venous eczema is often misdiagnosed as cellulitis, which, by contrast, is usually unilateral, tender, well demarcated, of a more acute onset and with associated systemic symptoms (Fig 1b).

Venous eczema tends to be chronic. Management includes lower limb elevation, compression stockings, regular emollients, topical steroids for itching and antibiotics for secondary infection.[8]

Atopic dermatitis

Atopic dermatitis (AD) is another common variant of eczema, affecting one in five children but often persisting into adulthood, with 11.7% of over 75s affected in the UK.[9] The incidence of AD is higher in Black and Asian-Indian patients compared with White patients[10–12]; thus, it is vital that physicians feel confident identifying AD in darker skin tones.

AD classically presents as pruritic, ill-defined red-pink patches with a fine overlying scale affecting the flexures. By contrast, in patients of African descent, AD tends to affect the extensor surfaces, and Asian-Indian individuals tend to have better-demarcated lesions with greater scaling and lichenification (thickened, leathery areas of skin caused by chronic scratching).[13] Other patterns more commonly seen in SOC include follicular eczema, which affects the hair follicles and resembles goosebumps,[14] and discoid eczema, which presents as extremely itchy, well-defined, coin-shaped lesions that can be misdiagnosed as ringworm (Fig 2a).[15] Furthermore, in darker skin, AD often appears violaceous and might be missed completely (Fig 2b).[16] Therefore, it is crucial to look for other signs of AD, such as oedema, warmth, scratch marks, oozing and crusting.[15] In addition, pigmentation changes are more prominent in SOC, with hyperpigmentation often being present during active disease and post-inflammatory hyper- or hypopigmentation persisting once the active disease has subsided.[17]

There is limited evidence for AD treatment efficacy in patients with SOC because of their under-representation in clinical trials.[18] However, current guidelines are similar across all skin types, with regular emollients and topical steroids being the mainstay of treatment for most patients with mild to moderate disease.[19] It is important to consider that topical steroids carry a greater risk

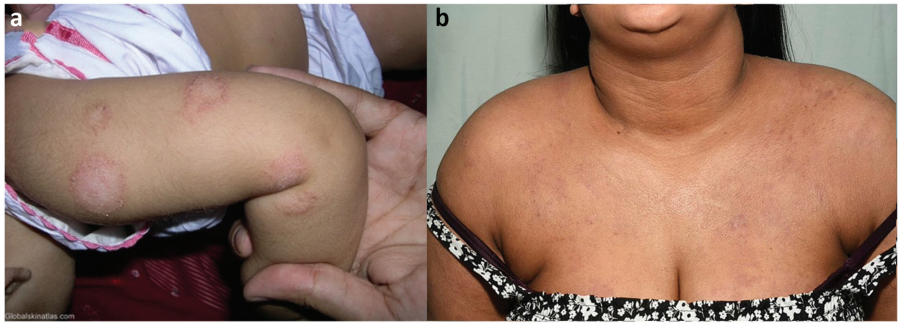

Fig 2. Eczema. (a) Discoid eczema in skin of colour (SOC). Reproduced with permission from Global Skin Atlas (globalskinatlas.com). (b) Atopic dermatitis in SOC. Reproduced with permission from Gloucestershire Hospitals NHS Foundation Trust.

of hypopigmentation in darker skin.[19] Furthermore, regarding systemic treatments for severe disease, studies have found that Black patients have a higher risk of alopecia secondary to methotrexate and a lower bioavailability of ciclosporin, thus requiring higher doses.[20,21]

Eczema herpeticum

Eczema herpeticum (EH) is a severe complication affecting 3% of patients with AD.[22] Despite predominantly affecting children, EH can occur in adults and is frequently missed by clinicians. It is a dermatological emergency and occurs because of disseminated cutaneous herpes simplex virus infection, often affecting patients with underlying AD because of impaired epidermal barrier function.[22] EH typically presents as clusters of pruritic, painful, monomorphic blisters, often on the face and neck (Fig 3).[23] Blisters usually contain fluid, which can be blood-stained, giving them a red, purple or black appearance. Within days, the blisters rupture to leave 'punched-out' erosions with haemorrhagic crusting.[23] Patients can also have associated systemic symptoms, such as fever, malaise and lymphadenopathy.[23] Lesions usually heal without scarring within 6 weeks.[23]

Diagnosis of EH is clinical and confirmed by viral swabs from the blisters, along with bacterial swabs to rule out secondary bacterial infection with *Staphylococcus aureus* and *Streptococcus pyogenes*.[24] Serious complications include herpetic encephalitis and keratoconjunctivitis.[22] An urgent ophthalmology opinion should be sought when ocular involvement is suspected, for example if the rash extends to skin around the eye or if the patient has ocular signs and symptoms (i.e. conjunctival injection, eye pain, photophobia, foreign body sensation or reduced visual acuity). Prompt administration of oral aciclovir or valaciclovir is advised, switching to the intravenous route for patients who are more unwell.[22]

Psoriasis

With a prevalence of 2–3% and a peak incidence in young and middle-aged adults, psoriasis is another frequently encountered condition in hospital inpatients.[25] It is less common in SOC but statistics in this group might be underestimated because of a lack of research.[25] Black patients reportedly have a larger surface area of skin involved and Asian-Indian and Hispanic patients are more likely to have severe psoriasis compared with White patients.[26,27]

Psoriasis typically presents as well-defined plaques affecting the extensor surfaces, umbilicus and hairline in all skin types. There might be associated nail and joint involvement. In contrast to the red-pink plaques seen in white skin, plaques in SOC can appear violaceous or hyperpigmented with an overlying silvery scale (Fig 4a).[25] Therefore, in patients with darker skin, active psoriatic lesions are harder to diagnose and can be mistaken for post-inflammatory hyperpigmentation (Fig 4b).[28] Furthermore, plaques in Black patients tend to be thicker with more scaling compared with those in White patients.[25] As in AD, patients with SOC are more prone to long-term pigmentation changes, which can be psychologically distressing and persist for years after lesion resolution.[29]

Treatment for psoriasis is generally similar across patients of different ethnic backgrounds and is guided by disease severity, comorbidities and patient preference.[25] In terms of biological therapy, minor clinical differences in efficacy and safety between races have been noted.[30] Ixekizumab is thought to provide the highest skin clearance for Asian-Indian and Latino patients, whereas brodalumab is most efficacious among black patients.[30] It is also important to consider that patients with SOC might have different hair textures and practices; thus, topical scalp treatments need to be individualised.[28] For example, Afro-textured hair can be fragile and dry; thus, it should not be washed every day and oil-based emollient foams and lotions are preferred.[28]

Skin cancer

Skin cancer has a lower incidence in patients with SOC compared with White patients because of the increased melanin in the epidermis providing greater protection against UV light.[31] Despite this, skin cancer has a higher mortality rate in patients with SOC because of a delay in detection and diagnosis.[32] This can be attributed to several factors: misconceptions regarding immunity to skin cancer among people of colour; unfamiliarity among both patients and clinicians regarding the appearance of skin cancer in SOC; and a lack of diversity in public health skin cancer campaigns.[31,33,34]

Skin cancer can be broadly classified into melanoma and non-melanoma skin cancer. Melanoma is the deadliest form of skin cancer in all racial groups. It develops from melanocytes in the basal layer of the epidermis and presents as a dark, rapidly spreading macule or patch.[31] In White patients, it predominantly affects sun-exposed sites. By contrast, in Black and Asian-Indian patients, up to 75% of melanomas affect non-sun-exposed sites, such as the palms, soles and nails.[31] This subtype is known as acral lentiginous melanoma (Fig 5). Lesions in these areas are more likely to go unnoticed by patients and clinicians or can be misdiagnosed as a fungal infection or viral wart, leading to a delayed diagnosis.[31]

The two predominant types of non-melanoma skin cancer are squamous cell carcinoma (SCC) and basal cell carcinoma (BCC). Whereas BCC is the most common cutaneous malignancy in White individuals, SCC is the most common in African-Americans and Asian-Indians.[31] SCC represents 30–65% of skin cancers in dark-skinned people.[31] It typically presents as a rapidly growing, firm plaque or nodule with a crusted or scaly surface. In contrast to its distribution in fair skin, SCC in darker skin often affects non-sun-exposed sites, such as the legs, feet and anogenital region.[35] SCC is also more likely to affect areas of chronic inflammation and scarring in SOC compared with White skin.[35] BCC represents

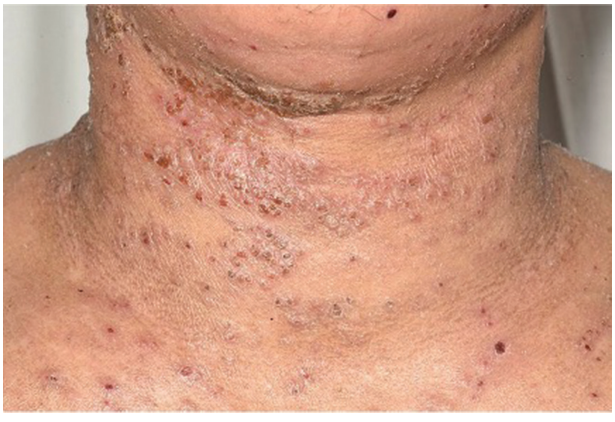

Fig 3. Eczema herpeticum affecting the neck in skin of colour (SOC). Reproduced with permission from Gloucestershire Hospitals NHS Foundation Trust.

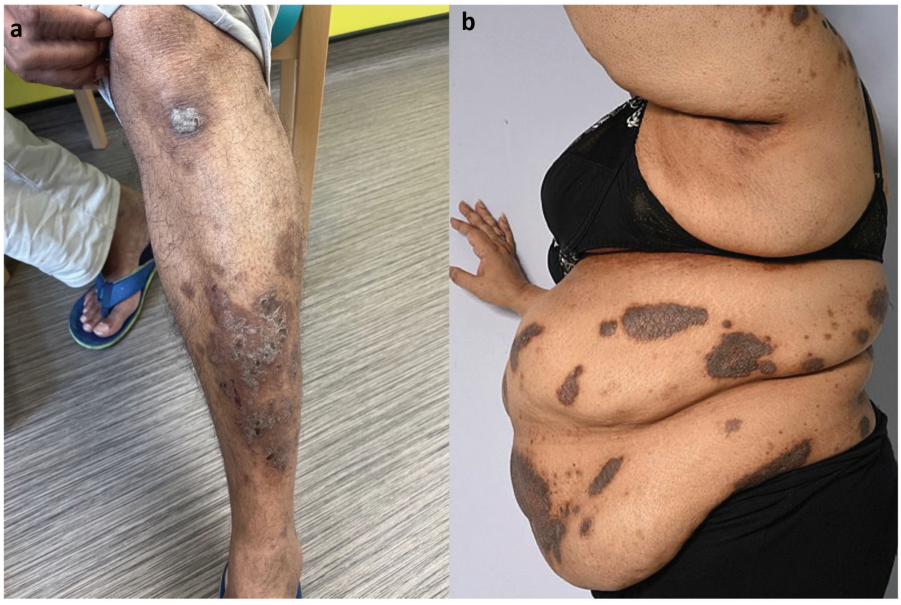

Fig 4. Psoriasis. (a) Plaque psoriasis affecting the leg in skin of colour (SOC). (b) Plaque psoriasis affecting the trunk in SOC. Reproduced with permission from Gloucestershire Hospitals NHS Foundation Trust.

20–30% of skin cancers in dark-skinned people.[31] It is typically associated with UV light exposure and affects sun-exposed sites in all races, with almost 90% of cases occurring on the head and neck in patients with SOC.[31] Over 50% of BCCs are pigmented in SOC; thus, diagnosis can be challenging because they could easily be misdiagnosed as seborrhoeic keratoses.[31]

Skin cancer treatment varies depending on its size and stage, with surgical removal being the mainstay of treatment, followed by radiotherapy, chemotherapy, immunotherapy or targeted therapy for metastatic disease.[31] There is evidence that individuals with SOC might be less likely to receive standard-of-care excisions for melanoma, and Black patients might have increased time to therapy.[35]

Drug eruptions

Cutaneous drug eruptions are common, affecting ~2–3% of hospitalised patients.[36] Common culprits include antibiotics, anticonvulsants and allopurinol.[36] Reactions can range from mild and self-limiting to severe and potentially life-threatening, involving multiple organ systems.

Morbilliform drug eruptions

Morbilliform drug eruptions account for 94% of cases.[36] 'Morbilliform' refers to the measles-like, maculopapular appearance of the rash. It appears 4–14 days after drug initiation and 1–3 days following re-exposure.[37] Patients present with symmetrically distributed red-pink macules and papules, starting on the trunk and pressure areas and spreading to the extremities.[37] In patients with SOC, colour changes might be less obvious and, thus, patients should be examined closely for subtle changes in skin tone and papules (Fig 6a, b). Although mucosal involvement is absent, there might be associated mild fever and pruritus.[37] The rash clears within 7–14 days of stopping the causative drug.[37] Emollients and topical steroids can also be helpful.[37]

Stevens–Johnson syndrome and toxic epidermal necrolysis

In contrast to morbilliform drug eruptions, Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN) are rare, life-threatening reactions usually triggered by medication. They are variants of the same condition and are distinguished by the extent of skin detachment.[38] Asian-Indian and Black patients

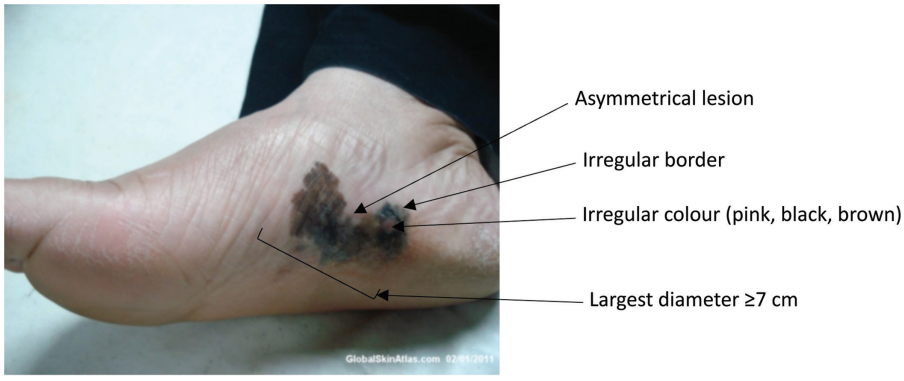

Fig 5. Acral lentiginous melanoma in skin of colour (SOC), with concerning features labelled. Reproduced with permission from Global Skin Atlas (globalskinatlas.com).

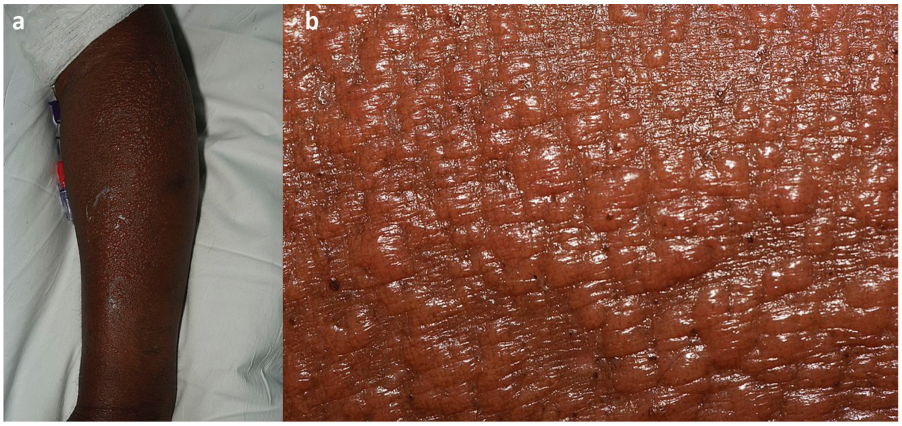

Fig 6. Morbilliform drug eruption in skin of colour (SOC): (a) distant and (b) close-up views. Reproduced with permission from Gloucestershire Hospitals NHS Foundation Trust.

are more susceptible to TEN compared with White patients.[39] Additionally, an association has been found between HLA-B*1502 and carbamazepine-induced SJS and TEN in Han Chinese and Thai populations, and it is now recommended to screen these individuals for the presence of this allele before starting carbamazepine therapy.[40]

Symptoms usually develop within 4 weeks of drug initiation.[38] A flu-like prodrome precedes an abrupt onset of tender, red-pink macules, atypical target lesions and blisters, beginning on the trunk and spreading rapidly to the face and limbs (Fig 7a).[38] Cutaneous pain is an important early feature of SJS/TEN.[38] Blisters merge to form sheets of epidermal detachment, exposing a weeping dermis.[38] Mucosal involvement occurs in most patients (Fig 7b).[38] Red-pink colour changes can be masked by background pigmentation in SOC; thus, patients should be examined thoroughly to identify blisters, epidermal detachment, mucosal lesions and tenderness. Management requires a supportive, multidisciplinary approach, preferably in a burn centre with intensive care involvement for ventilatory and circulatory support if required.[38] The causative drug should be identified and stopped immediately.[38] Treatment of SJS and TEN is controversial and beyond the scope of this review.

Pigmentary disorders

Individuals with SOC are more likely to suffer from pigmentary disorders, with dyschromia affecting 19% of Black patients presenting to dermatology clinics in one study.[29] Post-inflammatory hyperpigmentation (PIH) is an acquired disorder characterised by an increase in skin pigmentation in response to skin damage.[41] It can occur secondary to many of the conditions discussed above in addition to acne vulgaris, for example. Inflammation or trauma to the skin triggers an increase in melanin synthesis, which is transferred to surrounding keratinocytes in the epidermis, and occasionally into the dermis.[41] This leads to hyperpigmented patches at the site of original disease after it has healed, which might become darker on exposure to UV light.[41] More rarely, individuals can develop post-inflammatory

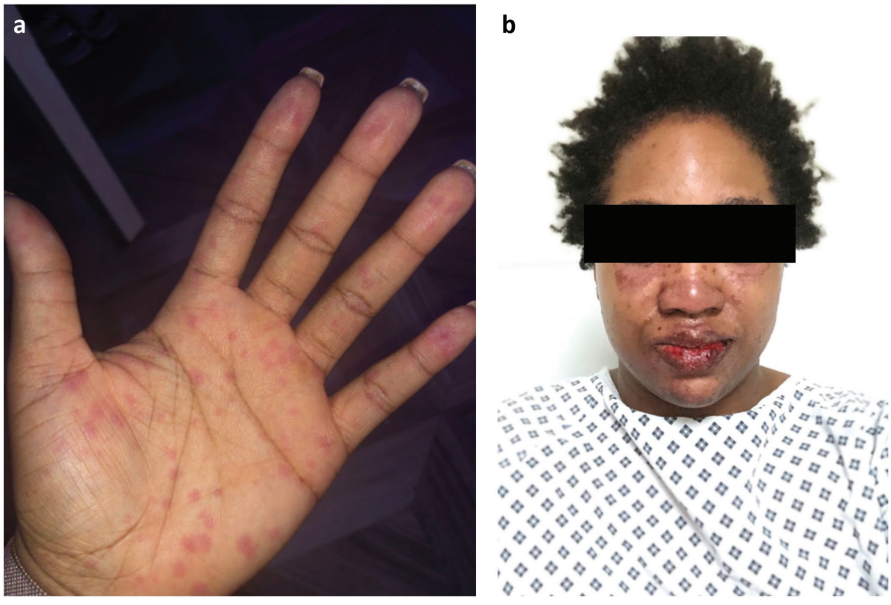

Fig 7. Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN). (a) Stevens–Johnson syndrome (SJS)/toxic epidermal necrolysis (TEN) target lesions on hand in skin of colour (SOC). (b) Mucosal involvement in SJS/TEN in SOC. Reproduced with permission from Gloucestershire Hospitals NHS Foundation Trust.

hypopigmentation. This is poorly understood but thought to be caused by the inhibition of melanogenesis and, in severe cases, the loss of melanocytes.[42] It is more prominent in SOC because of the colour contrast with the normal surrounding skin.[42]

Another acquired pigmentary disorder that is more prevalent in individuals with SOC is melasma.[43] Colloquially known as the 'mask of pregnancy', melasma presents as bilateral brown macules or patches, typically on sun-exposed areas such as the face.[43] It is more prevalent in women of reproductive age with skin types III and IV and is caused by an overproduction of melanin, which is deposited in the epidermis and/or dermis.[43]

Hyperpigmentation is notoriously difficult to manage and can often be more distressing for patients than the primary disease itself.[44] In PIH, the initial priority should be to address the underlying inflammatory dermatosis.[41] Given that UV light exposure can worsen hyperpigmentation, sun avoidance and regular sun cream application are recommended.[41] Often a variety of treatments are required for significant improvement, and can include topical and systemic agents, laser and light-based therapies or chemical peels.[41,43] Hydroquinone, a topical agent that reduces melanin production by inhibiting the enzyme tyrosinase, has traditionally been used first-line for many years.[45] However, prolonged use has been associated with exogenous ochronosis, which is characterised by blue-black or blue-grey hyperpigmentation of the skin; thus, it should not be used in isolation.[45] Tranexamic acid is an alternative emerging treatment for melasma.[46] However, it is important to use these treatments with caution because many of them can inadvertently aggravate the hyperpigmentation by damaging the epidermis further.[41]

Keloidal scarring

Keloidal scarring is another condition that disproportionally affects individuals with SOC, with an estimated 4.5–16% of Black and Hispanic populations in the USA being affected.[47] A keloid is a benign, firm, smooth growth caused by an abnormal fibroproliferative wound-healing response (Fig 8).[47] The scar tissue grows excessively beyond the original wound borders and often progresses over time.[47] Keloids usually occur at sites of trauma and can develop months to years after the original injury, commonly arising on the ear lobes, scalp, sternum, deltoid region and suprapubic area.[47] The higher prevalence of keloidal scarring in SOC might result from inherent structural differences in the skin, because individuals of African descent have been found to have reduced levels of collagenase, the enzyme responsible for degrading collagen.[48] Additionally, there is a hereditary component, with 5–10% of patients with keloids having a family history of the condition.[47]

The most important factor in keloid scar formation is prevention, and clinicians should take particular care when operating on patients prone to developing keloids, including individuals with SOC.[47] A wide variety of treatments are available, with variable success rates, and management can be incredibly challenging.[47] Examples include surgical excision, which carries a high risk of recurrence, in addition to silicone-based materials to aid healing and intralesional steroid injections to reduce inflammation.[47]

Conclusion

In this review, we have outlined how skin colour is determined and classified, why diagnosing conditions in SOC can be challenging, and nine important conditions that we believe physicians should be able to identify in patients with SOC. SOC is under-represented in medical curricula, dermatology learning resources and research.[2,49] Efforts are being made to rectify this, but disparities still exist. It is estimated that, by 2051, the non-White proportion of the population of the UK will increase to 25%.[50] By being aware of our knowledge gaps and educating ourselves and others about SOC, we can help to provide the best quality of care for patients of all skin types in our increasingly diverse population. ■

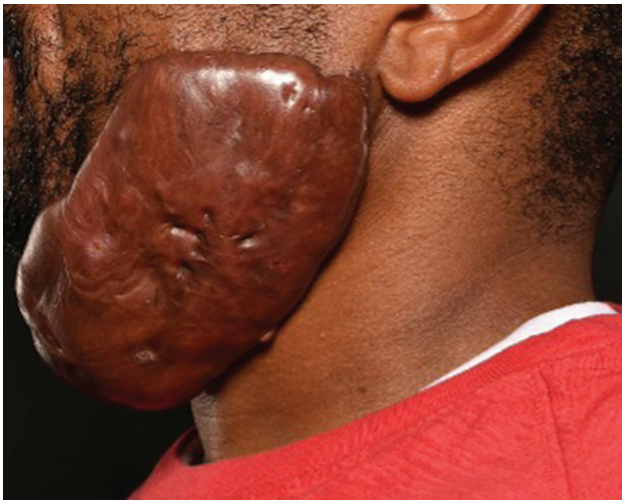

Fig 8. Keloid scar in skin of colour (SOC). Reproduced with permission from Gloucestershire Hospitals NHS Foundation Trust.

Acknowledgements

The authors would like to thank Global Skin Atlas (globalskinatlas.com) and Gloucestershire Hospitals NHS Foundation Trust for kindly providing the images used in this article.

Supplementary material

Additional supplementary material may be found in the online version of this article at www.rcpjournals.org/clinmedicine:
S1 – Examples of key conditions in different skin tones

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