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Dermatology

SKIN OF COLOUR



MATTERS

**No patient should be disadvantaged because of their skin tone.
Let's improve diversity in dermatology.**

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time to surface

IGA, Investigator global assessment.

References:

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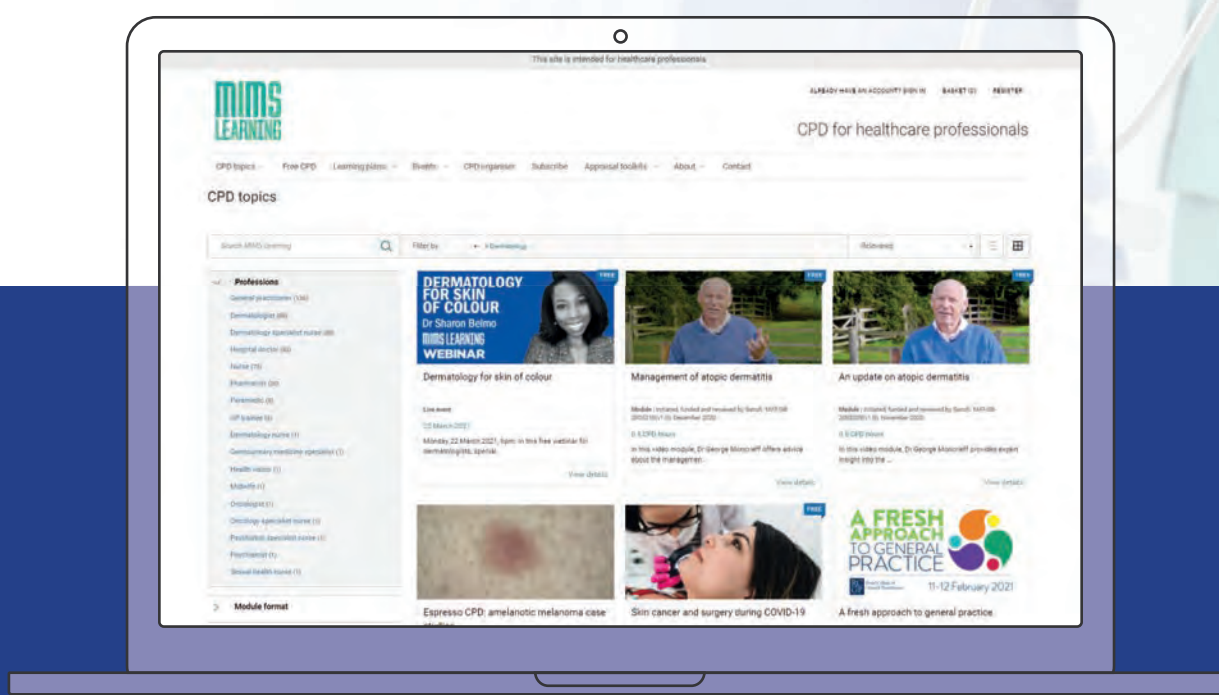
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Let's improve diversity in dermatology



“Clinicians will increasingly encounter skin and hair conditions in people, of colour and need to be trained to manage these effectively

I'm delighted to welcome you to this issue of *MIMS Learning Dermatology* - a special edition dedicated to dermatology for skin of colour.

The UK is diverse and it is important our learning resources reflect this. We know that clinicians are increasingly encountering skin and hair conditions in people of colour. We also know that there is a significant gap in available education, which you may have come across during your training.

We value your trust in *MIMS Learning Dermatology* and believe it is our duty and responsibility to provide knowledge from diverse perspectives. We want to ensure our content serves you and your patients by supporting your ongoing education, so we are proud to dedicate this entire issue to skin and hair conditions in people of colour.

Our guest editorial adviser, Dr Sharon Belmo, a consultant dermatologist and clinical lead for the Centre of Evidence Based Dermatology Skin of Colour Resource is an expert in this area and has worked hard to raise awareness of the issue for many years. She has planned the articles you will read here to give you a thorough grounding in dermatology for skin of colour, including disorders of pigmentation, skin cancer in skin of colour, paediatric dermatology, hair disorders, and more.

Wider conversations around health inequalities experienced by people of colour can serve as a springboard for us all to improve our knowledge.

In the process of creating this issue, I have spoken with colleagues about their experiences as patients and people of colour, struggling with skin problems, or searching online for information to be met with nothing but images of white skin.

To this end, pictures are a crucial part of dermatology education and illustrating our articles with effective and accurate images is a priority. I was surprised to struggle as much as I have done to find suitable pictures of darker skin.

This underrepresentation of skin of colour in medical image libraries and textbooks does a disservice to clinicians and patients alike. Dr Belmo examines this issue in more depth on [page 7](#).

If you enjoy these articles, why not watch Dr Belmo's webinar, available for free on-demand at mimslearning.co.uk. Dr Belmo discusses structural differences in skin of colour, the presentation of dermatological conditions in darker skin and gives an overview of conditions that are more common in skin of colour.

Don't forget that these articles are also available as learning modules online at mimslearning.co.uk/dermatology - free for a limited time - including extra interactive features, content and quizzes.

If you are not already registered at mimslearning.co.uk, signing up is free and straightforward. Select 'dermatology' as your special interest to ensure you don't miss out on our upcoming bulletin full of specialist dermatology learning and tips.

I'm grateful to Dr Belmo for her support and contribution to this issue of *MIMS Learning Dermatology*, and to all of the authors for their informative articles. I hope you enjoy reading them. Skin of colour matters and together we can improve diversity in dermatology.



Rhiannon Ashman
Editor
MIMS Learning Dermatology



Guest editorial adviser
Dr Sharon Belmo
Consultant dermatologist,
Croydon University
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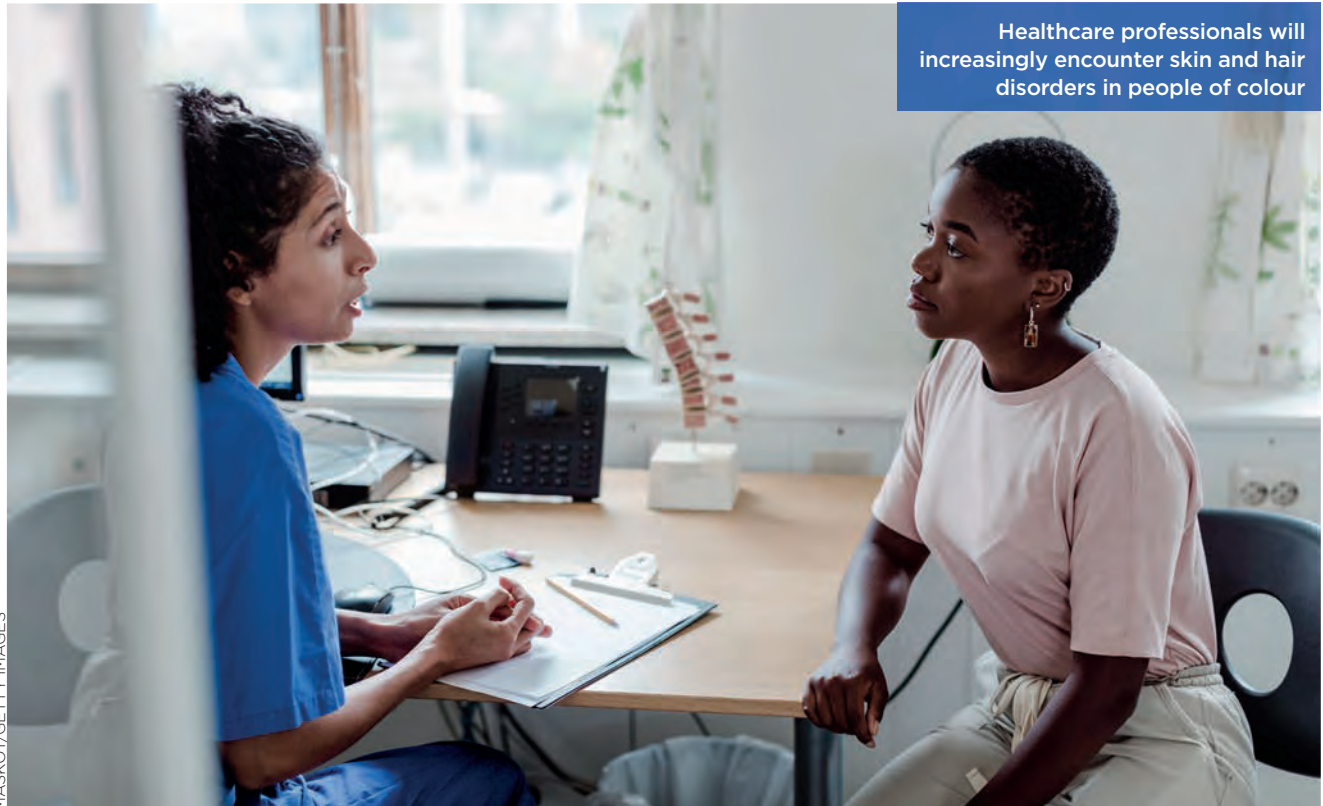
Reference: 1. Gallagher J. *et al.* Poster presented at EADV Congress 2009.

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TOPICAL INNOVATION

Dermatology for skin of colour



No patient should be disadvantaged because of their skin tone so we must take the opportunity to learn more about dermatology for skin of colour, writes **Dr Sharon Belmo**



Dermatology for skin of colour focuses on skin and hair disorders in individuals with darker skin tones than those of white European ancestry. This includes people of African, Asian, Middle Eastern and Hispanic/Latinx descent, also taking inter-racial mixing into account.

WHY IS THIS IMPORTANT?

Dermatology for skin of colour is important because some conditions are unique, more prevalent or clinically variable in darker skin. There are also cultural beliefs and habits among certain ethnic groups that may impact their skin and hair, such as hair grooming practices, skin lightening, hair coverings, traditional Chinese medicine, homeopathy and ayurvedic medicine. Historically, dermatology taught and practised in the Western world has focused on lighter skin types.

There is a notable underrepresentation of skin of colour in our learning resources, and stereotypical images are not uncommon. For example, a review of images in two commonly-used dermatology textbooks found that while 22-32% of images depicted skin of colour, darker skin was more likely to be used to illustrate sexually transmitted disease, accounting for 47-58% of these images.¹ Such representation might condition clinicians to associate skin of colour with infectious disease rather than common dermatological conditions that are more relevant to most non-white people in the UK. This might lead to

unnecessary or inappropriate investigations or questioning that can be detrimental to the patient-clinician relationship.

Inappropriate referrals or investigations might also occur as a result of unfamiliarity with nuances in skin of colour. For example, it is not uncommon for benign racial variants such as longitudinal melanonychia and dermal melanocytosis to be referred as melanoma or non-accidental injury, respectively.

The incidence of COVID-19 is disproportionately high among black, Asian and other minority ethnic groups in the UK and USA. Despite this, a 2020 review of 130 publications relating to cutaneous manifestations of COVID-19 found not a single image showing darker skin.² All of the images used were of lighter skin types.

Furthermore, textbook descriptions of dermatological conditions are generally given in the context of white skin. For example, 'erythema' is widely used to denote inflammation in conditions such as eczema and psoriasis. This description is most relevant to lightly-pigmented skin because redness may be masked in darker skin types, as a result of background pigmentation. Depending on the skin tone, inflammatory changes in skin of colour may be more subtle, or manifest as varying shades of brown, purple or grey, rather than red. Relying on erythema as a sign of inflammation in skin of colour may result in delayed or misdiagnoses. See [page 11](#) for more on this.

“The fundamentals of dermatology must be taught with all skin types in mind”

It is important that the fundamentals of dermatology are taught with all skin types in mind. This may require some re-learning. There has been a significant lack of diversity in dermatology teaching and learning resources. It is time to change the narrative.

IS THIS RELEVANT TO THE UK?

Demographics in the UK are rapidly diversifying. Between 2001 and 2011 there has been a 61% increase in the non-white population.³ By 2051, the non-white population in the UK is projected to increase to 21%, with the white population falling to 79%. The mixed-race population is predicted to rise by 148–249%, while the Asian population will also rise by 95–153%.⁴

Moreover, this diversity will spread to many more parts of the country, beyond the major cities where ethnic minorities are currently concentrated.⁴

Healthcare professionals nationwide will increasingly encounter a variety of skin and hair disorders in people of colour, and need to be trained to manage these effectively.

At present, there is no formal requirement in the dermatology curriculum for UK dermatology trainees to have any experience in skin of colour. In a 2013 survey of dermatology trainees, only 22% reported having ever had formal ethnic dermatology teaching and less than half felt that they would be competent in treating the UK's ethnic minority population at the end of their training.⁵

To best serve our diverse population, it is important that our future dermatologists and other healthcare professionals are confident and competent in recognising and treating dermatological conditions in all skin types.

The ability to recognise subtle skin changes in darker skin is even more crucial now, with the increased use of teledermatology as a result of the COVID-19 pandemic.

It goes without saying that patients want clinicians who can treat disorders in their skin type. A survey of black patients attending a dermatology clinic found that they valued dermatologists who demonstrated experience and knowledge of ethnic dermatology, citing frustration with dermatologists who lacked this knowledge.⁶ Cultural competency has been cited as a major determinant of patient satisfaction.

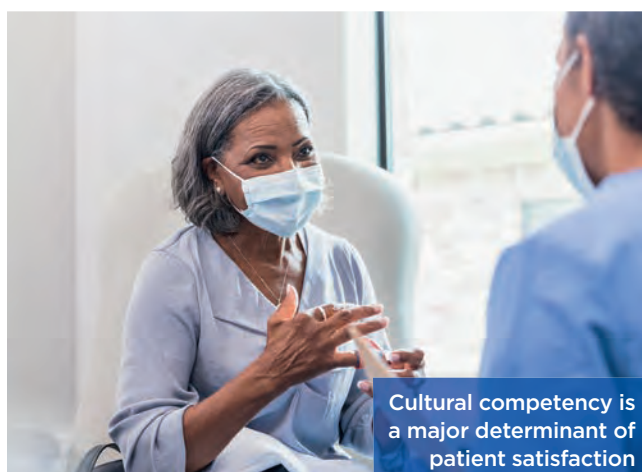
No patient should be disadvantaged because of their skin tone.

WHERE DO WE GO FROM HERE?

There has been a marked increase in awareness of diversity in medicine recently. Perhaps the Black Lives Matter movement and protests following the killing of George Floyd in 2020 have been a catalyst for a new era of diversity that UK dermatology needed. It is long overdue, but this is only the beginning.

I am honoured to have written the first dermatology for skin of colour syllabus, which will become an integral part of the UK dermatology specialist registrar training curriculum, as set by the British Association of Dermatologists (BAD). It is my hope that this will pave the way for future dermatology training and shape skin of colour education for other healthcare professionals.

To date there have only been a handful of training events fully dedicated to dermatology for skin of colour in the UK. It is



imperative that more events follow and become integral to dermatology teaching and training.

My MIMS Learning webinar discussing dermatology for skin of colour in more depth is available to watch for free, on-demand, at mimslearning.co.uk. I will also continue to run the Centre of Evidence Based Dermatology Skin of Colour Resource, a free learning tool dedicated to all things skin of colour (see [page 41](#)).

The BAD is working on integrating skin of colour throughout their workstreams, learning resources and social media platforms, and have recently formed a Skin Diversity Sub-Committee.

I am pleased that dermatology for skin of colour has been given a platform, and it is important to maintain the momentum. We need to see more skin of colour clinical images, promote UK-based skin of colour clinical research and learning opportunities, encourage diversity within the workforce and mentor our trainees. **The demographics of the UK are changing, it's time that dermatology caught up!**

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Dr Sharon Belmo is a consultant dermatologist at Croydon University Hospital and clinical lead for the Centre of Evidence Based Dermatology Skin of Colour Resource

I would like to thank MIMS Learning Dermatology for dedicating this issue to skin of colour and their ongoing contribution to diversity in medicine.



RUSLANDASHINSKY/GETTY IMAGES

Evidence linking UV and melanoma in skin of colour is weak

UV not an important melanoma risk factor in skin of colour

It is well-accepted that UV exposure is a risk factor for cutaneous melanoma in fair-skinned people, but a recent systematic review has found that this may not be the case in skin of colour.

Researchers found that evidence linking UV exposure to melanoma risk in people of colour was weak. As such, current recommendations for people of colour to use UV protection to prevent melanoma are not supported by the evidence.

STUDY DESIGN

Researchers searched databases including PubMed, Cochrane, and Web of Science for studies that analysed UV exposure as a risk factor for cutaneous melanoma in skin of colour. A total of 13 studies reporting on over 7700 melanomas were included. For the purposes of this review, skin of colour was defined as:

- any race/ethnicity other than non-Hispanic white
- Fitzpatrick skin types IV, V and VI, or
- tanning ability of 'rarely' or 'never' burns.

UV exposure measures included in the studies were UV index, irradiance, latitude, history of phototherapy, and history of sunburn.

KEY FINDINGS

Some 11 of the 13 studies reported no link between UV exposure and melanoma in skin of colour. Of the remaining two studies, one found a weak positive association in black men and the other found a weak positive association in Hispanic men.

Notably, the evidence that researchers found was of moderate to low quality and further research is required.

EXPERT VIEW: DR SHARON BELMO

The pathogenesis of melanoma in darker-skinned individuals is poorly understood. People of colour are underrepresented in clinical trials, and UK data on ethnicity and skin cancer are lacking. This has resulted in blanket statements about sunscreen in the prevention of melanoma being applied to all ethnic groups. We need to be able to give our patients of colour evidence-based advice, which means conducting more research that is inclusive.

REFERENCE

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Dermoscopic clues revealed in darker skin

Dermoscopy is a key tool for clinicians and studies have demonstrated its utility in diagnosing non-neoplastic skin diseases, including inflammatory, infiltrative and infectious conditions. However, there is limited research into dermoscopic features of these conditions in darker skin types, with most studies having been done on white or Asian skin.

Researchers have now identified a number of characteristic dermoscopic clues present in darker skin types that might aid the differential diagnosis of non-neoplastic conditions, and determined that dermoscopy is a useful diagnostic tool for non-neoplastic skin diseases in people with darker skin.

STUDY DESIGN

In this retrospective observational study, members of the International Dermoscopy Society submitted cases and dermoscopic images of confirmed non-neoplastic dermatoses in patients with Fitzpatrick skin types V and VI.

A total of 653 patients were included in the analysis. Investigators who were blinded to the diagnosis assessed the images for predefined dermoscopic criteria.

KEY FINDINGS

The investigators identified clinical categories of dermatoses and determined a number of significant dermoscopic features in each category. The basic parameters evaluated were:

- Vessels (morphology and distribution)
- Scales (colour and distribution)
- Appendages findings
- 'Other structures' (features other than vessels, scales and appendages findings, including colour and morphology)
- 'Specific clues'

Dermoscopic clues included, for example, interstitial brown circles and semi-circles in exogenous ochronosis, brown structureless areas with ostial sparing in melasma, and white globules with peripheral brown/grey pigmentation in macular amyloidosis.

EXPERT VIEW: DR SHARON BELMO

Dermoscopy is underused in non-neoplastic dermatoses. It can be a helpful tool in the assessment of pigmentary disorders in skin of colour, such as melasma and benign longitudinal melanonychia. It can also aid diagnosis of basal cell carcinoma, which is often pigmented in skin of colour and can be confused with seborrhoeic keratoses and melanoma.

REFERENCE

Errichetti E, Ankad BS, Sonthalia S, et al. *Eur J Dermatol* 2020. DOI: 10.1684/ejd.2020.3928.



SOUTH CHINA MORNING POST/GETTY IMAGES

Dermoscopy is a useful diagnostic tool in darker skin

EFFICACY OF SILICONE TREATMENT IN SCARRING

Scarring is a major issue which potentially affects tens of thousands of people in the UK every year. People with scarring face physical, aesthetic, psychological and social consequences – as well as emotional and financial costs.¹

THE BURDEN OF SCARS

Annually, 100 million patients develop scars in the developed world alone as a result of 55 million elective operations and 25 million operations after trauma.¹

Scars occur when the dermis is damaged and this can affect anyone. Each year in the UK there are 4.7m surgical procedures,² 174,700 C-sections,³ 31,000 cosmetic surgery procedures⁴ and 130,000 burns admissions to A&E,⁵ all of which can lead to abnormal hypertrophic and keloid scarring.

QUALITY OF LIFE

Scars are often seen as trivial, but their presence significantly affects a patient's quality of life.⁶ Scars can influence a patient's psychological wellbeing, which can lead to a diminished quality of life,⁷ including anxiety, social isolation and depression.^{8,9} Additionally, they can be physically painful, tender and itchy, while physical deformity as a result of skin scar contractures can be disabling.¹ Therefore consideration should be made towards preventative measures of scarring.⁶

TYPES OF SCAR

Scars vary in presentation and can include:

Immature scars: red, sometimes itchy or painful and can be slightly elevated.

Keloid scars: focally raised, itchy scars extending over normal tissue, which may develop up to 1 year after the injury without regression.¹⁰

Linear hypertrophic scars: red, raised and sometimes itchy scars, confined to the border of the original wound.¹⁰ They appear within 4 to 8 weeks following wounding¹¹ and may increase rapidly in size for 3-6 months.¹⁰

Widespread hypertrophic scars: red, widespread, raised, sometimes itchy scars, which remain in the borders of the injury.¹⁰ This scar is common after burns, mechanical trauma or necrotising infections.¹²



RISK FACTORS

Although anyone can develop a hypertrophic or keloid scar, the risk factors are greater for:

Darker skin types (especially for keloid scars)^{13,14}

Younger people between the ages of 10 and 30¹³

Scars that cross joints or skin creases at right angles¹⁵ or areas of high tension after surgery and wound closure, e.g. the back of the shoulder, sternum and lower abdomen.¹⁶

Those with a family history of scarring,^{17,18} previous scarring or who have had a greater number of surgical interventions.¹⁹

People going through hormonal changes, e.g. during pregnancy.^{17,18}

Injuries affecting deeper layers of the skin.¹⁹

EFFICACY OF SILICONE IN THE TREATMENT OF SCARRING

In 2011 a panel of 24 experts developed an extensive practical, **evidence-based set of guidelines**¹² which could be useful for surgeons, dermatologists, GPs and other physicians for scar prevention and management.

They advocated silicone therapy for:¹²

Non-invasive first line prophylactic and treatment option for hypertrophic scars and keloids.

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- Minimal irritation
- Available with UV protection
- Available as a spray

Silicone gels and sheets are the **'gold standard'** non-invasive therapies for prevention and treatment of hypertrophic scars and keloids.¹²



*Clinical data supports efficacy for scars up to 4 years but KELO-COTE® is most effective on new scars.

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Common conditions in skin of colour



Dr Mary Sommerlad presents an essential guide to common dermatoses and their presentations in skin of colour



Eczema on the foot: lichenification is common in skin of colour

Skin of colour characteristically reacts to common dermatoses in different ways to lighter skin tones. An appreciation of these subtle, yet crucial, differences is essential for clinicians to make the correct diagnosis.

In Western Europe, dermatology evolved as a distinctive medical specialty from the late 18th Century onwards. The overwhelmingly predominant skin colours at the time – and until well into the 20th Century – were lightly-pigmented European tones.

For this reason, textbooks classifying skin disease described common skin conditions in terms of how they present in white skin, with little or no reference to presentation in skin of colour.

Population demographics have changed significantly since then – with an influx of people with more pigmented skin arriving in the UK after World War II – but medical textbooks, undergraduate and postgraduate medical education and online resources have largely not deviated from white skin being the reference skin tone.

This makes it difficult for clinicians to confidently diagnose common skin conditions in people of colour.

ERYTHEMA

In white skin, the hallmark of inflammatory skin disease is erythema, which presents as shades of pink and red of varying intensity.

However, erythema is often imperceptible in skin of colour because the widespread dispersion of melanin throughout the stratum corneum masks the redness of inflammation.

Instead, inflammation may present as shades of violet and purple, or as an increase in pigmentation leading to a darkening of the skin.

ECZEMA

Eczema occurs in all ethnic groups, but higher incidences have been reported in black and Asian populations, in comparison with their white counterparts. Additionally, children of African heritage are twice as likely to experience severe disease than children of European heritage.

Eczema presents as itchy areas of skin, which can form papules and blisters in the acute phase and scaling and lichenification in the chronic phase (see image, above). Dry skin, fissures, and staphylococcal and herpetic infection are common in eczematous skin.



DR SHARON BELIMO

Acne: management of hyperpigmentation is important in skin of colour

In skin of colour, hyperpigmentation is present in the acute and chronic phases. Once the inflammation has resolved, post-inflammatory hyper- and hypopigmentation may persist for several months.

Eczema can distribute on the skin in different patterns. Classically, the flexural presentation is most often described. However, in skin of colour, extensor, follicular and discoid presentations are common. These distribution patterns are extremely rare in children of European heritage. Lichenification is also more common in skin of colour than it is in white skin.

ACNE

Acne is prevalent in all skin types and is most commonly seen in teenagers and pre-menopausal women.

The hallmark of acne is the comedone, which evolves into papules and then pustules and cysts. Seborrhoea is a common feature.

People of colour present with hyperpigmentation of papules and pustules (see image, above). Cystic acne is less common in skin of colour than in white skin.

Pomade acne – presenting as widespread comedones, particularly on the forehead – is common in those with Afro-textured hair, where hair products that can trigger acne are used to style or treat the hair. Advice on avoiding these, and any occlusive skin care products, is necessary.

Post-inflammatory hyperpigmentation and keloid scarring are long-term complications that are more likely to occur in skin of colour and can be associated with severe morbidity.

Acne treatment is the same for all skin types. However, in skin of colour, the management of peri- and post-inflammatory hyperpigmentation should be part of standard acne treatment.

This should involve the use of broad-spectrum sunscreens, antioxidants with dark mark lightening qualities such as azelaic acid 20%, and advice against using any treatments that may traumatise skin and potentially lead to exacerbation of the pigmentation.



DR NIGEL STOLLERY

Lichen planus: hyperpigmented lesions on a patient's lower leg

PSORIASIS

Prevalence rates of psoriasis vary in different ethnic groups. The highest rates are found in Scandinavia and the lowest rates are among Indigenous people of the Americas.

Textbooks usually describe psoriatic plaques as being salmon pink with an overlying silver scale. In skin of colour, erythema is very difficult to discern and, instead, psoriasis may present as violaceous, hypochromic, or darker-than-usual skin colour plaques with a grey overlying scale (see image, opposite).

In skin of colour, as in lighter skin, the plaques are sharply demarcated and have a predilection for extensor surfaces, the umbilicus, the hairline and intertriginous areas. Nails and joints can also be affected.

Post-inflammatory hypo- and hyperpigmentation are prevalent and bothersome in skin of colour, but treatment of psoriasis is the same regardless of skin tone and follows a stepwise approach.

LICHEN PLANUS

Lichen planus is an autoimmune condition where auto-reactive T-cells attack the basal keratinocytes. The cause is often idiopathic but it may be associated with hepatitis C and certain medications, including antihypertensive drugs, antimalarials and NSAIDs.

Lichen planus is typically described as the 6Ps: pruritic, polygonal, planar, purple papules and plaques, found particularly on the flexor surfaces of the wrists, the dorsal surfaces of the hands and the anterior surface of the lower legs. In skin of colour, the 'purple' in this list is often substituted for an increased pigment (darker-than-usual skin colour), a violaceous colour (see image, above) or hypopigmentation.

Post-inflammatory hyperpigmentation is common and often more troublesome in darker skin types. Oral involvement is common and its appearance is the same in all skin types – white Wickham's striae visible on the buccal mucosa.

There are numerous variants of lichen planus. Lichen planus actinicus is more common in people from the Middle East,

Comparison of common skin conditions		
Condition	Presentation in white skin	Presentation in skin of colour
Erythema	Shades of pink and red of varying intensity.	Often imperceptible in skin of colour, instead may present as shades of purple or increase in pigmentation.
Eczema	Flexural presentation is most common.	- Extensor, follicular and discoid presentations are common. - Hyperpigmentation in the acute and chronic phases. - Lichenification is more common.
Acne	Post-inflammatory erythema is common.	- Hyperpigmentation of papules and pustules, which may persist. - Cystic acne is less common than in white skin. - Keloid scarring is more common.
Psoriasis	Salmon pink plaques with an overlying silver scale.	- Violaceous, hypochromic, or darker-than-usual skin colour plaques with a grey overlying scale. - Post-inflammatory hypo- and hyperpigmentation are prevalent.
Lichen planus	- Red/purple lesions. - Post-inflammatory erythema or pigmentation may occur.	- Rather than purple lesions, presentation is with increased pigment, a violaceous colour or hypopigmentation. - Post-inflammatory hyperpigmentation is common. - Some variants, such as lichen planus pigmentosus, are more common.

North Africa and India, and presents as a lichenoid eruption in a photo-distributed pattern, classically on the lateral forehead. This condition recurs in the sunny summer months and remits in winter months. Nail and mucosal changes are often absent in this group.

Hypertrophic lichen planus is also thought to be more common in skin of colour, although there are no published epidemiological data.

Lichen planus pigmentosus is a pigmented variant of lichen planus that presents as dark brown or black patches on sun-exposed sites or flexures. This variant is also seen more commonly in skin of colour. The treatment of lichen planus is the same for all skin types.

The table above outlines the dermatoses discussed here.

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MIMS LEARNING
Skin of
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Psoriasis on the lower back: sharply demarcated plaques with a grey overlying scale

Post-inflammatory hyperpigmentation



Dr Emily Akufo-Tetteh outlines the prognosis and management of post-inflammatory hyperpigmentation, and explains why it is problematic in skin of colour

Post-inflammatory hyperpigmentation (PIH) is an acquired increase in pigmentation. This increase in melanin production, or irregular distribution of pigment, occurs in response to inflammation or injury caused by either an endogenous or exogenous stimulus (see table, below).

PIH can be superficial (epidermal) or deep (dermal) depending on the level of inflammation. Melanocytes - which produce melanin - are found in the basal layer of the epidermis. Inflammation causes increased activity as well as hyperplasia and hypertrophy in these melanocytes.

In superficial PIH this activity leads to production of more melanin, which is then taken up by keratinocytes. Deeper injury causes damage to keratinocytes, leading to the release of melanin. The melanin is then phagocytosed by macrophages (to become melanophages) causing deep PIH.

Skin of colour has more melanin than lighter skin so there is an increased chance of PIH and it is more problematic. PIH presents as a macule or darker patch of skin. This can be tan, light/dark brown in superficial PIH or blue/grey in deep PIH.

PROGNOSIS

Epidermal PIH will often resolve spontaneously within a few months to years. Dermal PIH will take longer to resolve and may cause permanent discolouration.

A mix of epidermal and dermal PIH may be seen in the same injury and it is important to note that repeated, intense inflammation can cause long-lasting PIH.

MANAGEMENT

The most important consideration is to treat the underlying cause. This will help to prevent further discolouration. Remember that some treatments may themselves cause inflammation, which can exacerbate or cause further PIH.

Photoprotection

Photoprotection is key and the role of UV radiation in darkening of pigmentation should be emphasised to patients, particularly individuals with darker skin types who would not typically wear sunscreen.

Topical treatments

In addition to photoprotection, topical treatments can be used for epidermal PIH.

Hydroquinone is typically used first line but combination with other topical treatments, such as tretinoin and corticosteroids, is often needed. Prolonged use of hydroquinone should be avoided because of the risk of exogenous ochronosis.

Other topical treatments include adapalene and azelaic acid, which can be prescribed on the NHS, and cysteamine, which requires a private prescription. Adjunctive over-the-counter treatments include vitamin C (ascorbic acid) and kojic acid.

Chemical peels

Superficial chemical peels generally have a good clinical outcome in treating epidermal PIH in people with dark skin.



Post-inflammatory hyperpigmentation caused by irritant dermatitis

It is important to avoid a product that would cause inflammation as this may lead to new areas of PIH as well as scarring. These risk factors should be discussed with patients, along with the importance of sunscreen post-procedure.

Laser treatment and intense pulsed light therapies

Although there is not a lot of evidence to support the use of laser therapy in all skin types, this may be trialled after treatment failure or in combination with topical therapies.

Remember that melanin has a wide absorption spectrum and so may easily absorb wavelengths intended for deeper targets. This can lead to PIH as well as blistering and scarring.

Cosmetic camouflage

Camouflage can be used to conceal, contour or colour-correct skin conditions that cannot be treated medically or surgically. This can be considered for PIH that is not amenable to other treatment options. Typically, cosmetic camouflage would be used on more noticeable areas such as the face, neck and hands.

Common causes of PIH

Endogenous	Exogenous
▶ Eczema ▶ Acne ▶ Lichen planus ▶ Cutaneous infections, such as impetigo, viral exanthem ▶ Psoriasis ▶ Drug eruption, such as SJS/TEN, fixed drug eruption ▶ Connective tissue disease, such as lupus, vasculitis ▶ Vesiculobullous disease, such as pemphigoid, pemphigus	▶ Burns ▶ Laser therapy ▶ Chemical peels ▶ Cryotherapy ▶ Trauma ▶ Friction

Dr Emily Akufo-Tetteh is a consultant dermatologist at University Hospitals Birmingham

MANAGING ECZEMA-PRONE SKIN

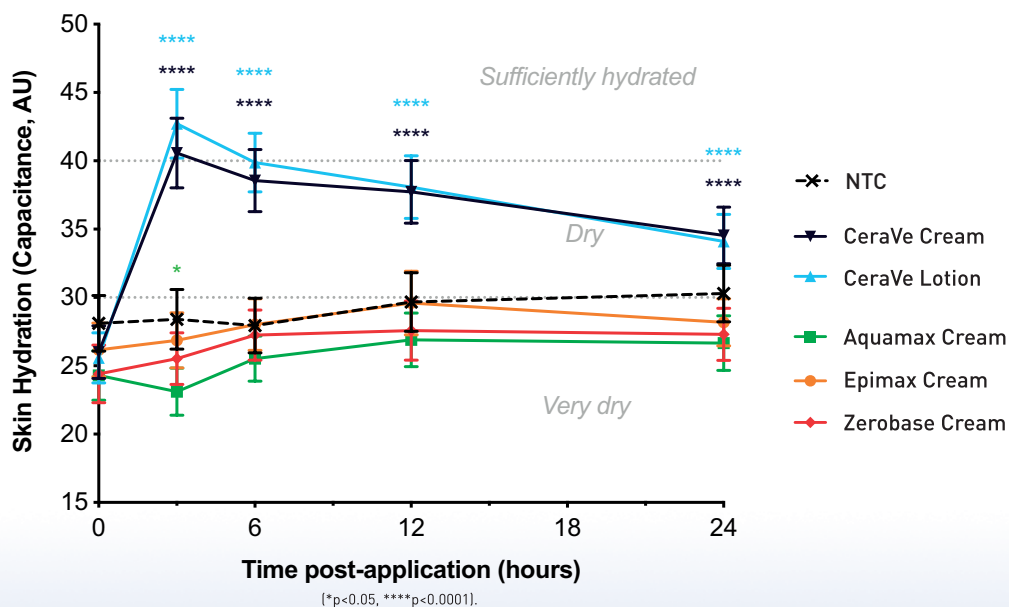
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¹"An investigation of the skin barrier restoring effects of a cream and lotion containing ceramides in a multi-vesicular emulsion in people with dry, eczema-prone, skin: THE RESTORE STUDY PHASE 1" Danby SG, Andrew P, Kay L, Pincock A, Chittock J, and Cork MJ

Hypopigmentation

Dr Nomathamsanqa Mathe discusses the psychological burden of hypopigmentation, as well as causes, clinical findings and management



Pityriasis versicolor: hypopigmented macules

Skin pigmentation depends on a person's racial origin and sun exposure. Melanocytes in the basal layer of the epidermis produce the pigment melanin (melanogenesis), which is carried to the surface of the skin by keratinocytes. Individuals all have a similar number of melanocytes, but they produce variable amounts of melanin, leading to a variety of different skin tones.

Disorders of hypopigmentation occur when melanogenesis is disrupted by cutaneous inflammation and inflammatory disorders, infection, or external trauma.

Hypopigmentation is often chronic and difficult to treat. The contrast of hypopigmented or depigmented patches on darker skin can be devastating for affected individuals. There may also be associated stigma, resulting in poor self-esteem and a significant psychological burden.

PITYRIASIS VERSICOLOR

Pityriasis versicolor is caused by infection with the yeast *Malassezia*, which proliferates in the corneal layer of the skin producing azelaic acid. This inhibits melanogenesis and causes discoloured hypopigmented patches. It tends to occur in seborrhoeic areas of the scalp, face and chest.

It can be asymptomatic or mildly pruritic. It is most common in young adults and occurs more frequently in men than women.

Hot humid climates, hyperhidrosis, immunosuppression and use of oily skin care products are all risk factors for development of this condition.

On examination you may see widespread, scaly hypopigmented macules of varying size with fine scale on the face, chest and trunk. Patches on the scalp may be thick and scaly. Wood's light fluoresces yellow.

Management

- Topical azole creams or shampoos (such as ketoconazole, miconazole)
- Oral itraconazole or fluconazole for widespread or resistant disease.

Hypopigmented patches can persist after treatment. If fine scale reappears over these patches, treatment should be repeated.

SEBORRHOEIC DERMATITIS

Seborrhoeic dermatitis is a chronic and recurrent dermatitis affecting sebaceous-rich areas of the scalp, face and trunk. *Malassezia furfur* yeast is implicated in this condition. It mainly affects young adults, more commonly men than women. It is mildly pruritic and can flare during winter.

Risk factors are immunosuppression (including HIV infection), personal or family history of psoriasis and neurological conditions such as Parkinson's disease and epilepsy. Oily skin, applying oils and pomades to the scalp and infrequent hair washing can also contribute.

On the face and scalp, seborrhoeic dermatitis may present with erythematous, hypopigmented or hyperpigmented skin (erythema can be difficult to appreciate in darker skin). Scaly greasy patches can affect the nasolabial folds, eyebrows, central forehead and behind the ears. Hypopigmented petaloid lesions around the hairline or face may occur in skin of colour. Blepharitis can also occur.

Poorly-defined erythematous or hypopigmented patches with fine scale may present on the upper chest, back and skin flexures. There may also be follicular papules and pustules on the chest and back, as seen in pityrosporum folliculitis.

Children of colour often do not experience the classic 'cradle cap' appearance of seborrhoeic dermatitis, but have erythema, scaling, and hypopigmentation of the affected areas.

Management

- Topical azoles
- Topical corticosteroids
- Topical calcineurin inhibitors
- 2% ketoconazole shampoo
- Selenium sulfide, Capasal or zinc pyrithione-containing shampoos.

Afro-textured hair is fragile and washing daily, particularly with anti-dandruff or anti-yeast shampoos, will result in excessive dryness and breakage. Once-weekly washing is optimal, and this should be considered when prescribing treatment for seborrhoeic dermatitis of the scalp in women.

Frequent application of oils and pomades to the scalp should be advised against. Vehicles of treatments should also be carefully considered in patients with afro-textured hair because preferences may vary as a result of hair styling practices.

POST-INFLAMMATORY HYPOPIGMENTATION

Post-inflammatory hypopigmentation is an acquired partial or total loss of skin pigmentation following inflammation, trauma or dermatological treatments.

Affected skin may return to its usual skin tone over weeks or months, but this is unlikely if there has been complete destruction of melanocytes. There is often a history of preceding symptomatic inflammation.

Common causes include:

- Atopic dermatitis
- Psoriasis
- Seborrhoeic dermatitis
- Lichen sclerosus
- Cutaneous lupus
- Burns/freezing
- Laser resurfacing
- Other inflammatory skin conditions.

Complete post-inflammatory depigmentation can occur in severe atopic dermatitis and in discoid lupus erythematosus.

Management

- If the diagnosis is uncertain, consider skin biopsy.
- Treat the underlying cause.
- Consider topical tacrolimus, steroids or phototherapy.

VITILIGO

Vitiligo is an acquired pigmentary disorder of the skin. It presents with hypopigmented or depigmented macules or patches surrounded by normal skin.

Vitiligo has no racial predilection but is more striking in darker skin types and this can have a huge psychological impact.

Vitiligo can be segmental (childhood onset, rapidly progressive, early hair involvement, not often associated with autoimmune disease) and non-segmental (late onset, slowly progressive, late hair involvement, family history, associated with autoimmune disease).

Management

- Screen for autoimmune diseases
- Potent topical steroid or topical calcineurin inhibitors
- Cosmetic camouflage
- Phototherapy
- Depigmentation
- Surgery – skin grafting.

MYCOSIS FUNGOIDES

Mycosis fungoides (MF) is the most common form of cutaneous T-cell lymphoma (CTCL). Hypopigmented MF occurs in younger, darker skin types and presents as poorly-defined, irregular, widespread hypopigmented patches.

MF can be divided into patch, plaque, and tumour stages. Early-stage MF can be treated with potent topical corticosteroids or phototherapy. Radiotherapy may be



Depigmentation in discoid lupus erythematosus

considered for thicker plaques or tumours. Hypopigmented MF has a better prognosis than other subtypes of CTCL.

SARCOIDOSIS

Sarcoidosis is a granulomatous disease mainly affecting the lungs. However, 25% of patients have skin involvement. The disease is more severe in black patients of African and Caribbean descent.

It can present as hypopigmented macules or patches, or hypopigmented papules. Lesions are often asymptomatic and can be yellow/brown nodules or plaques.

Cutaneous lesions are often biopsied to confirm the diagnosis and treated with potent topical corticosteroids. Thick, nodular cutaneous lesions can be treated with intralesional corticosteroid injections. Systemic involvement is often investigated in secondary care.

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Melasma

Melasma is most prevalent among women of colour and can be particularly challenging to treat effectively, writes **Dr Mariam Balogun**



Melasma (also referred to as chloasma, or mask of pregnancy) is a commonly occurring chronic acquired disorder of hyperpigmentation.

The highest prevalence of this condition is among women of reproductive age with skin of colour. It has been cited as the most common pigmentary disorder in Indians and third most common in those of African descent.

Melasma is most notable on the face and so can cause significant psychological distress, which is exacerbated by its recalcitrance to treatment, tendency for frequent relapses and incomplete clearance.

PATHOPHYSIOLOGY

Although the exact mechanism of induction of melasma has not been delineated, it has been hypothesised that the cause is overactive melanocytes, present in increased amounts, under the induction of UV radiation and/or hormones. These melanocytes contain increased numbers of melanosomes which, in turn, produce greater amounts of melanin in all layers of the epidermis.

The role of UV radiation is highlighted because melasma affects the sun-exposed face, with relative sparing of sun-protected areas such as the philtrum, and the observation that lesions fade over winter. Additionally, visible light has been shown to worsen melasma, particularly in skin of colour.

Oestrogen is also thought to contribute to melasma because the condition occurs most frequently in women, and is associated with pregnancy or use of oestrogen-containing contraception.

However, 10% of those affected are post-menopausal, suggesting melasma may be a photo-ageing disorder. Furthermore, histology of lesional skin shows increased solar elastosis, increased numbers of dermal mast cells and increased vascularisation. This latter feature may account for the improvement in appearance of melasma with tranexamic acid and pulsed dye laser treatment.

Medications, such as phenytoin and phototoxic drugs, and autoimmune thyroid disease, have also been associated with exacerbations of melasma.

CLINICAL PRESENTATION

Patients present with brown to brown-grey macules or patches, mainly on the face, which are symmetrically distributed with an irregular outline.

Melasma follows three classic patterns:

- Centrifacial (most common), involving the forehead, cheeks, nose, upper lips and chin with relative sparing of the philtrum (see image, above right).
- Malar distribution, involving the cheeks and nose (see image, opposite).
- Mandibular distribution, occurring along the jawline.

In practice, there is usually an overlap of these distributions. Melasma does not usually breach either the superior or inferior orbital rim, nor does it extend below the mandible. Rarely, the extensor aspects of the forearms and the mid-upper chest may be involved.

The lesions develop or are accentuated following UV exposure or during pregnancy. In the latter case, lesions tend to fade after childbirth in individuals with lighter skin types, but can persist in patients with more deeply pigmented skin.



Centrifacial pattern melasma

Melasma may be classified as epidermal, dermal, mixed or indeterminate, depending on the primary location of pigment deposition on examination with Wood's lamp. Although this assessment is helpful in determining the likely success of topical depigmenting agents (epidermal pigmentation is associated with positive response), confocal microscopy has shown all melasma to be admixed. In addition, a limitation of Wood's lamp is the difficulty assessing dermal deposition of melanin in patients with darker skin.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis for facial hyperpigmentation is wide. A thorough history and examination can help to distinguish melasma from other conditions. Go to mimslearning.co.uk to see an in-depth comparison of differential diagnoses. In cases of diagnostic uncertainty, a skin biopsy may be required.

MANAGEMENT

The management of melasma can be challenging – there is often partial response and frequent relapse – with greater pigment deposition in the dermis having poorer treatment outcomes.

Often no single treatment is effective, so a multi-faceted approach is required to optimise the chance of success. Treatment is particularly challenging in patients with Fitzpatrick skin types IV–VI because of the risk of rebound post-inflammatory hyperpigmentation as a sequela of the management modalities.

There is no standardised therapeutic approach. However, a stepwise approach to managing melasma can be used based on individual patient characteristics, such as severity and duration of condition, patient skin type, contributing risk factors and affordability of the proposed management plan.

Most of the treatments outlined here are not funded on the NHS, with the exception of off-label use of tranexamic acid, limited ranges of sunscreen and azelaic acid. It is important to



Malar pattern melasma

bear this in mind when counselling the patient about their management plan. Risk factors that may further exacerbate or induce melasma, such as oral contraceptive pills and phenytoin, should be modified if appropriate.

First-line treatment

Photoprotection

Photoprotection is central to melasma management because of the role that UV radiation plays in its development. Avoidance of sun exposure from 10am to 3pm, and wearing protective clothing, such as long-sleeved shirts and a wide-brimmed hat, should be encouraged.

Broad-spectrum sunscreen with an SPF of 30+ and containing a physical blocker such as titanium dioxide is recommended. Furthermore, as visible light plays a prominent role in the disorder, sunscreens containing iron oxide in greater than 3% concentration are recommended.

Clinicians should be aware that darker-skinned patients may be reluctant to use sunscreens containing physical blockers because they tend to leave a cosmetically undesirable white cast. As such, patients may benefit from tinted preparations or brands suited to darker skin.

As a result of the need for strict photoprotection, darker-skinned patients who are already likely to have vitamin D deficiency may require vitamin D supplementation, and baseline levels should be checked at onset of management.

Hydroquinone 4% cream

Hydroquinone 4% cream is considered to be the gold-standard treatment for mild or epidermal melasma.

It acts by competitively inhibiting the conversion of tyrosine to melanin by the tyrosinase enzyme, downregulating the synthesis of melanocytes and degrading melanosomes.

Common short-term side effects (which are dose-dependent) include stinging, irritation, redness and contact allergic dermatitis. Side effects associated with medium- to long-term use include milia, paradoxical post-inflammatory hyperpigmentation (see [page 14](#)) and exogenous ochronosis.

Exogenous ochronosis is a permanent blue-black pigmentation that occurs after prolonged use of hydroquinone, especially when it is formulated with resorcinol. Fortunately, it is extremely rare.

There is robust evidence for the efficacy of hydroquinone, but there are concerns about its carcinogenicity as a result of potential formation of benzene derivatives after hepatic metabolism. However, it is renally excreted and, to date, there have been no reports of hydroquinone-induced malignancies.

Triple therapy combination

Triple therapy combination (TTC) containing 4% hydroquinone, 0.05% tretinoin and 0.01% fluocinolone acetonide is the gold standard in managing moderate to severe melasma. Pigmanorm[®], available in the UK, is similarly formulated and contains hydroquinone 5%, tretinoin 0.1% and hydrocortisone 1%.

Common side effects include burning, redness, irritation and dry, flaky skin. As such, this should be introduced slowly and used infrequently in patients with darker skin to reduce the risk of post-inflammatory hyperpigmentation.

Once tolerance is established, it may be used more frequently. It can be used for up to 6 months, with no reports of steroid-induced atrophy. The retinoid component of the TTC cream confers its effect by increasing keratinocyte turnover, downregulating synthesis of melanin and melanosomes.

Azelaic acid 20%

Azelaic acid 20%, a weak competitive tyrosinase inhibitor, is a category B drug and can be considered for use first line in pregnant women. Some studies have shown it to have similar efficacy to hydroquinone 4%, particularly in skin types IV-VI. Associated side effects include redness, itching and scaling.

Second-line treatment

Tranexemic acid

Tranexemic acid, a haemostatic drug with anti-plasmin activity, has been shown to be effective in the management of melasma, using a twice-daily 250mg dosage. It works by reducing inflammation, tyrosinase activity in melanocytes and also downregulates the vascularisation process involved in melasma.

Despite being lower than the 3500mg dose used for haemorrhagic conditions, patients require thorough screening for thrombotic risk factors prior to its use.

Chemical peels

Chemical peels such as salicylic acid (20–30%) and glycolic acid (20%–70%) can serve as adjunctive treatment in melasma. Lower concentrations should be used in skin types IV-VI to avoid post-inflammatory hyperpigmentation.

Chemical peels are particularly effective when hydroquinone has been commenced at least 2 weeks earlier. Peels such as trichloroacetic acid should be avoided in patients with skin types IV-VI because of the significant risk of scarring and post-inflammatory hyperpigmentation.

Third-line treatment

Lasers and light therapy

Lasers and light therapy may be considered if other treatments have failed. These should be used with extreme caution in patients with skin types IV-VI because of the high risk of post-inflammatory hyperpigmentation.

Most data published for lasers and melasma have been for skin types III-IV, with varying results. Low fluence and low treatment density non-ablative fractional lasers and Q-switched 1064nm lasers may be considered, but more research into laser use in skin types IV-VI is required.

Other topical agents

Other topical agents that may be of benefit in melasma include kojic acid, arbutin, niacinamide, cysteamine, liquorice extract and ascorbic acid. Camouflage may also be considered.

Suggestions for further reading on melasma are available in the online version of this article, at mimslearning.co.uk.

Dr Mariam Balogun is a dermatology registrar at Sandwell and West Birmingham NHS Trust

Central centrifugal cicatricial alopecia



The cause of this condition is not well understood, but it is increasingly apparent that it is not solely related to hair styling practices, writes [Dr Sharon Belmo](#)



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CCCA presents with hair loss at the crown with peripheral spread, usually sparing the lateral and posterior scalp

Central centrifugal cicatricial alopecia (CCCA) – previously known as hot comb alopecia – is a lymphocytic primary cicatricial alopecia and is the most common cause of scarring alopecia in black women. It has also been reported in men and children.

The prevalence is estimated to be between 2.7% and 5.7%. However, the real prevalence is not known as no population studies have been published to date. The average age at presentation is 36 years.

CLINICAL FEATURES

CCCA presents with scarring hair loss at the crown with peripheral spread, usually sparing the lateral and posterior scalp (see image, above). Patchy cases have also been reported.

It is often associated with burning, itching, soreness, altered sensation, scaling, papules or pustules but may be asymptomatic or present with broken hairs on the crown. It has been noted that the marked fibrosis and scarring that occurs in CCCA is often disproportionate to the clinical and histological signs of inflammation observed.

Differential diagnoses include female pattern hair loss, lichen planopilaris, alopecia areata, telogen effluvium and trichotillomania.

Dermoscopically, a peripilar grey-white halo can be seen, which is highly specific and sensitive for CCCA. Other features that may be seen include reduced hair density, hair shaft variability, absence of follicular openings and irregularly distributed pin-point white dots.

Histologically, a goggle sign is often present – that is, a fused outer root sheath of hair follicles emerging from the same ostium. Other features include premature desquamation of the inner root sheath, lymphocytic infiltration and perifollicular fibrosis.

AETIOLOGY

In the late 1960s, when CCCA was first reported, it was thought that the cause was use of the hot comb among black women. Later, CCCA was thought to be a result of chemical straightening and/or traction-related hair styles.

More recently, a genetic component has been proposed following reports of CCCA across two generations of families in

South Africa, including children with untreated natural hair. Additionally, *PADI3* gene mutations in CCCA-affected patients have now been identified. This is a significant finding because *PADI3* encodes for a protein that is essential to proper hair shaft formation.

Furthermore, critical genes implicated in fibroproliferative disorders, such as fibroids and keloids, have been shown to be upregulated in CCCA-affected scalp tissue.

The quest for the cause of CCCA is ongoing but it is becoming increasingly apparent that this disorder is likely to be multifactorial and not solely related to hair styling practices.

MANAGEMENT

Treatment of CCCA is challenging. Early diagnosis is crucial and general hair care advice should be given.

Treatment is aimed at suppressing inflammation and stopping the scarring process, preventing further hair loss. This is best approached using a combination of potent topical and intralesional steroids.

Key learning points

- ▶ CCCA is the most common cause of scarring alopecia in black women.
- ▶ CCCA presents with scarring hair loss at the crown with peripheral spread.
- ▶ The average age at presentation is 36 years.
- ▶ A peripilar grey-white halo on dermoscopy is highly specific and sensitive for CCCA.
- ▶ Initial treatment is with a combination of potent topical and intralesional steroids.
- ▶ In extensive, end-stage disease, wigs or scalp micropigmentation can help to camouflage hair loss.

Anti-dandruff shampoos and topical or oral minoxidil might also be used.

In refractory disease, systemic treatments such as doxycycline, hydroxychloroquine or ciclosporin may be required to halt the inflammation. Success with 10% topical metformin has recently been reported in patients for whom these other standard therapies have not worked.

In cases of extensive end-stage scarring, wigs or scalp micropigmentation can be used to camouflage the areas of hair loss.

Hair transplantation can be considered but is reserved for CCCA patients who have had stable disease for 9–12 months with an absence of inflammation histologically.

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Traction alopecia

Dr Olayemi Bello gives an overview of traction alopecia, which is common in women of African ancestry, including advice on differential diagnoses and patient education



Traction alopecia (TA) is hair loss caused by pulling forces on the hair that induce sufficient strain on the follicle to uproot the hair prematurely.

It typically occurs as a result of hair styling practices, or head coverings that are applied tightly on a repeated basis. Significant hair loss has been known to occur after a one-off event, although this is less common.

As the causative factors are primarily extrinsic, TA can affect people of all ages, genders and ethnicities. However, TA has most notably been reported in women and girls of African ancestry, with documented prevalence rates as high as 37% in this group.¹

CLINICAL FEATURES

The clinical features depend on the stage of the condition and can be observed at sites of maximal tension or exposure. This is commonly, but not restricted to, the perimeters of the scalp, particularly the fronto-temporal hairline (see image, right).

Soon after a tight hair style has been installed, pain, papules, pustules, erythema, tenting and dimpling of the scalp may be noted.

If the tension is repeatedly applied over a prolonged period, miniaturisation of the hair shafts and, eventually, patches of hair loss ensue.²

TA is typically reversible in the early stages but as it progresses, scarring takes place, resulting in permanent hair loss. At this stage, affected areas appear smooth and shiny with loss of follicular openings.

While the diagnosis of TA is largely clinical, there may be some diagnostic uncertainty. In this case, a biopsy may be required. Histology may show a reduced number of hair follicles, an increased ratio of miniaturised/vellus follicles to terminal follicles and an increased proportion of hairs in the catagen and telogen phases.^{2,3}

Occasionally, perifollicular inflammation and trichomalacia (distorted hair shafts) may also be identified.^{2,4} In longstanding or end-stage TA, perifollicular fibrosis is seen.³

AETIOLOGY AND RISK FACTORS

Hair styles that are commonly implicated in TA are tight ponytails and buns, braids, cornrows, weaves, wigs and locs. Head coverings may also contribute. These practices and hair styles may be of social, cultural or religious importance.

Key considerations include how tightly the styles have been applied and how frequently they are performed.

While the initial amount of tension applied by the stylist is important, manipulation of the hair following completion of the hairstyle can also play a role. The addition of extensions can increase the risk, particularly when added to chemically straightened hair.⁵ Having hair styles such as braids for a long duration may also contribute to the risk of TA.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses of TA include trichotillomania, androgenetic alopecia and frontal fibrosing alopecia (FFA).

Trichotillomania

Trichotillomania is a psychiatric condition characterised by repetitive pulling of an individual's own hair. It may result in



DR SHARON BELMO

Traction alopecia at the fronto-temporal hairline

significant functional impairment and often coexists with other mental health conditions, such as anxiety.⁶

As with TA, reduced hair density and short vellus hairs may be seen. However, in trichotillomania, there are usually broken hairs of unequal length and split ends.⁷

Female pattern hair loss

In female pattern hair loss (FPHL), miniaturisation of hair follicles is observed (similarly to TA), and differentiating between the two conditions can sometimes prove challenging. However, FPHL tends to present with diffuse hair loss most noticeable within the natural parting and the vertex.

Frontal fibrosing alopecia

FFA is a progressive scarring alopecia most commonly observed in white women over the age of 50 years. The literature

suggests a younger age of presentation (over 40 years) in women of African ancestry.⁸

Both TA and FFA commonly affect the frontal hairline, so it is important that a potential diagnosis of FFA is considered. Differentiation between the two conditions relies on a detailed history and careful examination. It is also important to be aware that both conditions may coexist in some circumstances.

A comparison of both conditions is outlined in the table below.

MANAGEMENT

The management of TA may be conservative, medical or surgical, but patient education is the cornerstone of effective treatment.

Patient education

Patients should be encouraged to modify or stop causative hair care practices (taking any social, cultural or religious factors into consideration).

For this to be effective, it is essential that patients have an understanding of the causes and potential long-term consequences of TA to provide motivation and context for the advised changes.

Furthermore, education of the wider community and hair care professionals is important for prevention.

Comparison of TA and FFA	
Traction alopecia	Frontal fibrosing alopecia
▶ Papules/pustules (traction folliculitis) ▶ Perifollicular erythema ▶ Pain/discomfort ▶ Fringe sign ▶ Hair loss only along frontal hairline or areas of maximal tension ▶ Tenting/dimpling of scalp ▶ History of traction-related hair styles/practices	▶ Papules ▶ Perifollicular erythema/pigmentation/scaling ▶ Pain/pruritus ▶ Lonely hair sign ▶ Hair loss along frontal hairline and other sites, particularly eyebrows ▶ Prominent frontal veins ▶ Glabellar red dots ▶ Benign facial papules ▶ Hypopigmentation of frontal hairline in skin of colour ▶ May be associated lichen planus pigmentosus in skin of colour

Medical treatment

There is a lack of high-quality evidence for the management of TA. Treatment options that have been documented include corticosteroids (topical and intralesional), antibiotics (topical and oral) and topical minoxidil.

Both corticosteroids and antibiotics have been used for their anti-inflammatory effects.⁷⁹ Antibiotics have also been used for associated folliculitis. Oral minoxidil has been reported to be effective but further data are required.¹⁰

Other treatments that are being used include platelet-rich plasma and microneedling but, again, further data are required.

Camouflage techniques

Camouflage techniques, such as scalp micropigmentation, hair fibres, hair paints and powders, can be helpful ways to create the illusion of increased hair density.

Wigs and hairpieces may also be used. However, caution must be exercised because inappropriate use of attachment methods such as tight combs and adhesives can further exacerbate TA.

Surgical treatment

In cases of inadequate response to medical treatment, or in end-stage TA, surgical management may be appropriate. This may include scalp reduction surgery or hair transplantation.

It is important that patient factors such as curl type and history of scarring are taken into consideration to select the correct surgical technique.

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Key learning points

- ▶ TA is a preventable condition most commonly affecting women and girls of African ancestry.
- ▶ TA typically occurs as a result of hair styling practices, or tight head coverings.
- ▶ Pain, papules, pustules, erythema, tenting and dimpling of the scalp are the first signs of TA.
- ▶ In longstanding or end-stage disease, perifollicular fibrosis is seen.
- ▶ Differential diagnoses include trichotillomania, androgenetic alopecia and FFA.
- ▶ Patient education and advice on modification or cessation of causative hair care practices is crucial.
- ▶ Higher-quality studies are required to explore the benefits of, and optimal treatment regimens for, agents such as corticosteroids, antibiotics and minoxidil.

Pseudofolliculitis barbae

Dr Monica Anyasodor takes a look at the clinical presentation and management of this disorder, which is most common in darker-skinned men

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Pseudofolliculitis barbae (PFB) is a common inflammatory skin disorder of the beard area, colloquially known as 'razor bumps'.

In PFB, shaved hairs become ingrown, initiating a foreign body reaction that leads to follicular and perifollicular inflammation.

The condition is most common in men with darker skin, particularly those of African-Caribbean ethnicity. The curly hair type that is predominant in this group is more prone to becoming ingrown. Evidence has also shown that there is a genetic predisposition to the condition.

CLINICAL PRESENTATION

Clinical features in PFB vary from a mild rash with superficial ingrown hairs to prominent inflammatory papules (see image, right). There may be significant post-inflammatory hyperpigmentation and more severe cases can lead to keloid scarring.

Masks, helmet straps and other protective gear worn for occupational or leisure reasons can exacerbate the condition as a result of occlusion and friction. PFB can have a considerable effect on an individual's appearance and psychosocial wellbeing.

Pseudofolliculitis can also occur at other sites of hair removal, such as in women who wax their bikini line.

PFB – and pseudofolliculitis in general – are distinct from folliculitis barbae, which presents with painful pustules caused by infected hair follicles. However, PFB can predispose to true folliculitis and the two conditions can coexist.

MANAGEMENT

If shaving continues, PFB follows a chronic course so, traditionally, patients have been advised to stop shaving completely. This halts the inflammatory process, eventually leading to a resolution in symptoms and a gradual improvement in post-inflammatory hyperpigmentation.

However, stopping shaving is often not desirable or practical in the long term.

Modifications to shaving techniques

Modifications such as reducing the frequency of shaving, using electric trimmers and not stretching the skin while shaving have been suggested to improve PFB, although evidence for these techniques is limited.

In general, good skin care practices should be adhered to and skin should be adequately prepared for shaving by washing with a gentle cleanser. Men who have PFB should avoid dry shaving, and use shaving gels and foams to reduce friction. It is recommended that they shave their beards in the direction of hair growth to minimise irritation.

People with pseudofolliculitis often learn to manage the condition by purchasing over-the-counter topical preparations. These often contain salicylic acid, a keratolytic which lifts surface skin cells and debris. This can reduce the number of ingrown hairs and allow them to be released from the skin more easily. Harsh and abrasive treatments should be avoided as these may worsen the condition and lead to scarring.

Medical treatments

Many of the treatments used for acne can be used to manage PFB. Benzoyl peroxide and topical retinoids may be beneficial and can be used in combination. Topical anti-inflammatory



Shaved hairs become ingrown, causing inflammation

antibiotics such as macrolides and tetracyclines may also improve symptoms and, in some cases, oral antibiotics may be required.

Topical corticosteroids can also be used short term to improve inflammation. Intralesional steroid injections may be required to treat large nodules and keloid scars.

Long-term hair removal

Longer-lasting hair removal methods can be a good solution for pseudofolliculitis at sites where complete hair removal is desired. A course of laser hair removal can lead to long-term hair reduction and a complete resolution of symptoms.

This must be performed by qualified practitioners with laser types suited to darker skin types, to minimise the risk of burning, scarring and permanent changes in pigmentation.

Electrolysis can offer permanent hair removal. However, this is expensive and is more time-consuming than laser hair removal, so may be best reserved for treating smaller areas.

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Acne keloidalis nuchae

Dr Oluwadamilola Jagun discusses this inflammatory condition – seen almost exclusively in men of African descent – including advice on prevention



Lesions may become itchy, painful or bleed

DR KEN GREER/VISUALS UNLIMITED, INC./SCIENCE PHOTO LIBRARY

Multiple individual and coalescent papules on the occipital scalp

Acne keloidalis nuchae (AKN) – also known as folliculitis keloidalis – was first described in 1869 by Kaposi.¹ It is an inflammatory condition marked by the presence of keloid-like acne lesions on the posterior neck and occipital scalp. While these lesions do resemble acne and keloids, the name is actually a misnomer. The pathology of AKN points to a type of chronic folliculitis or perifolliculitis, as evidenced by skin biopsies showing inflammation of different parts of the hair follicles and surrounding skin.

AKN is seen almost exclusively in men of African descent and is least common in white populations. Women can be affected, but this is rare (the ratio of men to women affected is 20:1). AKN is virtually non-existent in pre-pubertal patients and after the age of 50 years. The most common age of onset is the late 20s to early 30s.²

RISK FACTORS

The cause of AKN is not fully understood and it is likely to be multifactorial. However, the condition is thought to be preceded by acute inflammation of the hair follicles and surrounding skin, which progresses to a chronic folliculitis, before developing into true AKN.¹

AKN is most common in people of African origin who have curly hair, so it is thought that the growing tip of recently shaved hair curls back and penetrates the scalp, triggering an inflammatory response.

The mechanical trauma from a close shave with a razor or clippers, or from hats or shirt collars, is also thought to play a role in driving the inflammation.

There have been case reports citing anti-epileptic drugs (such as carbamazepine and diphenylhydantoin) and ciclosporin as causative factors. Research has also pointed to elevated androgens, chronic low-grade folliculitis, an autoimmune process and secondary infection having a role in AKN.^{1,3}

CLINICAL FEATURES

AKN is characterised by discrete reddish to skin-coloured or hyperpigmented 2–4mm papules or pustules on the posterior neck and/or occipital scalp.

There are no comedones, which differentiates it from acne, and the number, shape and distribution of the lesions differentiates it from a keloid or hypertrophic scar (see image, above).

The lesions are usually asymptomatic but, as they enlarge, they may become itchy, painful or bleed. As the disease progresses, new papules appear and the pre-existing papules may enlarge and coalesce to form a horizontal keloid plaque, often with tufts of hair (from a single follicular opening) on the periphery.

These keloidal plaques can grow as large as 10cm in diameter and can be cosmetically disfiguring. As such, early treatment is crucial.

MANAGEMENT

The first step in AKN treatment is prevention. Advise patients to avoid hard shirt collars or head gear that could cause irritation. They should also avoid extremely low or bald haircuts and avoid using clippers to line the posterior hairline (often desired aesthetically). Washing with an antimicrobial cleanser may be helpful.

Medical treatment includes the use of high-potency topical steroids and topical retinoids. For pustules or any other sign of infection, swabs should be taken, followed by topical or systemic antibiotics. For keloidal papules and plaques, intralesional triamcinolone (10–40mg/ml) is often effective.²

Surgical management varies depending on the size of the lesions. For small papules, a punch excision using a hair transplant punch that extends below the level of the hair follicles is recommended, to reduce the risk of recurrence. Such

wounds may be closed with sutures or left to heal by secondary intention.^{1,2} For lesions <1.5cm thick, an excision with primary closure is recommended, while larger plaques >1.5cm thick require a horizontal ellipse excision including the posterior hairline, extending to the fascia or deep subcutaneous tissue. Such wounds are better left to heal by secondary intention. Steroid injections should be delayed until the wound has fully healed as they can prevent further wound contraction.¹

Other treatments that have been effective include lasers (CO₂, Nd:YAG 1064nm, long-pulse diode) and cryotherapy.^{2,4} Freezing for longer than 25 seconds should be avoided because of the risk of hypopigmentation (find out more on [page 16](#)).¹

Lesions treated early often subside. Therefore, it is imperative that patients are managed promptly in primary care and referred to secondary care if no improvement is seen.

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Key learning points

- ▶ AKN is a type of chronic folliculitis or perifolliculitis; patients have inflammation of the hair follicles and surrounding skin.
- ▶ AKN is seen almost exclusively in men of African descent, most commonly in their late 20s and early 30s.
- ▶ Mechanical trauma from a close shave, or from hats or shirt collars, is thought to contribute to inflammation so advise patients to avoid these.
- ▶ In AKN there are no comedones, which differentiates it from acne.
- ▶ Lesions are usually asymptomatic but may become itchy, painful or bleed.
- ▶ Medical treatment includes the use of high-potency topical steroids and topical retinoids.
- ▶ Surgical management and lasers are also options.

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Keloids

Dr Nomathamsanqa Mathe and Dr Sharon Belmo explain what causes keloid scars and why they occur much more often in darker skin



The risk of keloid development significantly rises with increasing skin pigmentation

Keloids are benign overgrowths of scar tissue that occur more commonly in skin of colour, particularly black people.

They often result from direct injury, such as burns or scratches, or occur secondary to cutaneous inflammation, such as acne. The original trauma may be minor and is often unnoticed. Consequently, keloids may appear to arise spontaneously. They can appear weeks to months after the original insult.

In contrast to hypertrophic scars, keloid scar tissue overgrows and extends beyond the site of the original injury or inflammation.

They can be itchy, painful and cause significant disfigurement, resulting in marked psychological burden for patients.

EPIDEMIOLOGY

Keloids can affect people of any age, but are most commonly seen in adolescents and young adults. They are rarely seen in the elderly and are almost never seen in prepubescent children.

Keloids are thought to affect men and women equally, but some authors have reported higher incidences in women.

The risk of keloid development significantly rises with increasing skin pigmentation. Reported population incidences are variable, and more recent figures are needed, but rates of up to 16% have been noted in people of African, Hispanic and Asian ancestry, with much lower incidences in white populations. Studies have also reported that keloids occur 5-16 times more frequently in black people than in white people.

Keloids have been reported to run in families, raising the possibility of a genetic predisposition.

SCARRING

Distinguishing hypertrophic and keloid scars

Hypertrophic scar	Keloid scar
▶ Always results from injury ▶ Immediate onset ▶ Confined to wound margin ▶ Usually resolve spontaneously ▶ Good response to treatment ▶ Usually asymptomatic	▶ Preceding injury not always obvious ▶ Delayed onset – develops over time ▶ Extends beyond wound margin ▶ Very rarely resolve spontaneously ▶ Poor response to treatment ▶ Painful, itchy

PATHOGENESIS

The mechanisms behind keloid scarring remain unclear. Several pathological factors have been described.

Fibroblasts are important in both normal and abnormal wound healing, and are responsible for collagen and extracellular matrix deposition. Their action is mainly driven by growth factors, such as transforming growth factor-beta, platelet-derived growth factor, fibroblast growth factor-beta and insulin-like growth factor 1.

Keloid tissue has been shown to be more sensitive to these growth factors than normal skin. Moreover, keloid fibroblast density and proliferation rates are increased, while apoptotic rates are decreased.

Keratinocytes found in keloid tissue have been shown to induce keloid-like behaviour in normal fibroblasts. They are also thought to contribute to the decreased apoptotic rate of fibroblasts in keloids.

Myofibroblasts are also involved in wound healing and contraction. The presence of cell markers characteristic to activated myofibroblasts and alpha-smooth muscle actin-positive myofibroblasts has been reported in keloid tissue, indicating a potential role in keloid pathogenesis.

It has been suggested that melanocytes have an effect on keloid formation, evidenced by the increased incidence of keloids in darker skin compared with lighter skin.

Additionally, keloids are more likely to occur in areas of the body where there is a high concentration of melanocytes and are almost absent where melanocytes are less common, such as on the palms of the hands and soles of the feet.

CLINICAL PRESENTATION

Keloid scars initially appear as raised, erythematous-to-hyperpigmented smooth nodules or plaques arising in areas of tension or local injury.

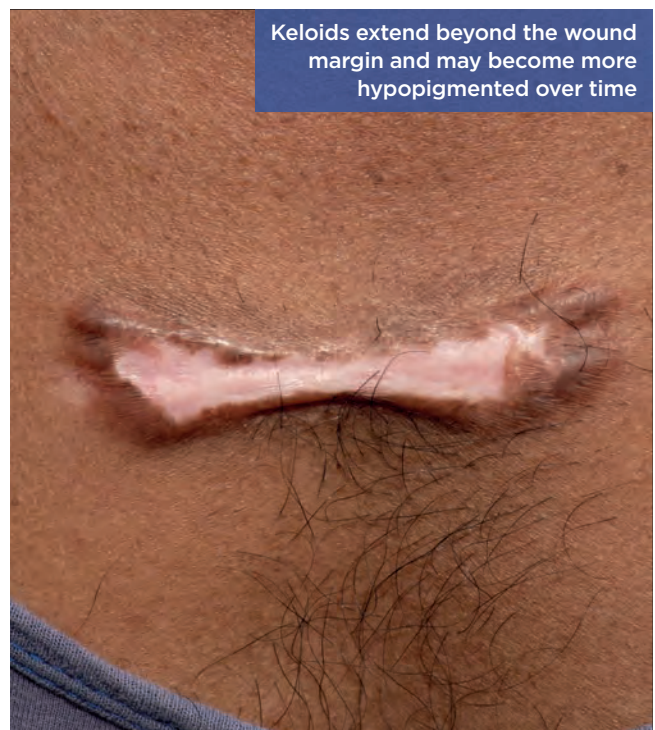
They extend beyond the wound margin into normal skin and may become more indurated and hypopigmented over time (see image, above).

Keloids can be found anywhere on the body but commonly occur on the head and neck, ears, upper chest and arms (see images, above and opposite). They are often pruritic and/or tender.

RISK FACTORS

The following factors increase the risk of keloid development:

- Darker skin
- Family history



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- Acne
- Trauma or local injury (such as surgery, burns, scratches, ear piercings)
- Skin tension
- Infection.

DIFFERENTIAL DIAGNOSES

The following conditions and lesions can present in skin of colour with similar scar-like features and are important to consider in patients with suspected keloid scars:

- Hypertrophic scars
- Dermatofibrosarcoma protuberans
- Dermatofibroma
- Sarcoidosis.

In particular, it can be hard to distinguish hypertrophic scars from keloid scars. They are also raised, erythematous-to-hyperpigmented scars that form after injury or cutaneous inflammation. But, in contrast to keloid scars, hypertrophic scars are usually confined to the wound margin and gradually improve over months to years.

The table above, left, outlines the differences between hypertrophic and keloid scars.

MANAGEMENT

Keloids are exceedingly difficult to treat. They are often recalcitrant to treatment and can increase in size or recur following therapy.

There are multiple treatment modalities for keloids including silicone gel or dressings, corticosteroids (topical and intralesional), cryotherapy and laser therapy. No single treatment is best and they are often combined.

Surgical management is an option but keloids recur in 80% of cases, so this is not recommended as monotherapy.

Radiotherapy or imiquimod may be used post-operatively.

Prevention and patient education is key. This includes advising patients who are known to form keloids to avoid trauma to the skin wherever possible, such as tattoos, piercings and elective cosmetic surgery. Other preventative measures relate to minimising risk of keloid formation if skin trauma, such as surgery, is necessary (see box, opposite).



Keloid scars can be found anywhere on the body but commonly occur on the head and neck

Key learning points

- ▶ Keloids result from direct injury or occur secondary to cutaneous inflammation.
- ▶ They can appear weeks to months after the original insult.
- ▶ Risk of keloid development significantly rises with increasing skin pigmentation.
- ▶ Keloids occur 5-16 times more frequently in black people than in white people.
- ▶ Scars can be itchy, painful and cause significant disfigurement.
- ▶ Adolescents and young adults are most commonly affected.
- ▶ Keloids extend beyond the wound margin; this distinguishes them from hypertrophic scars.
- ▶ They are often recalcitrant to treatment.
- ▶ To prevent keloids, patients should avoid skin injury where possible.
- ▶ Management options include silicone gel or dressings, topical or intralesional corticosteroids and cryotherapy.
- ▶ Keloids recur after surgical excision in 80% of cases.

Treatment and prevention of keloids

Treatment options

- ▶ Silicone gel or dressings
- ▶ Corticosteroid tape
- ▶ Topical corticosteroid under occlusive dressings
- ▶ Intralesional corticosteroid injections repeated every few weeks
- ▶ Cryotherapy
- ▶ Intralesional interferon-alpha2b
- ▶ Surgical excision – recurrence occurs in 80% of cases. Not recommended as monotherapy. Debulking the keloid pre-operatively may reduce the chance of recurrence.
- ▶ Radiotherapy (post-operative)
- ▶ Laser therapy
- ▶ Imiquimod (post-operative)
- ▶ Intralesional 5-fluorouracil

Preventative measures

- ▶ Advise patients to avoid tattoos, ear piercing or elective cosmetic surgery if they are known to form keloids
- ▶ Close surgical wounds with minimal tension
- ▶ Follow skin creases when making incisions
- ▶ Avoid mid-chest incisions
- ▶ Limit skin stretching during healing
- ▶ Use fixable garments, bandages, tape, silicone gel sheets after surgery

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“Keloids are 5-16 times more common in black people

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– risk of severe burns. Fabric that has been in contact with these products burns more easily and is a serious fire hazard. Avoid contact with the eyes. Talk to your doctor or healthcare professional before use if the skin is broken, badly cracked, infected or bleeding. **Contraindications:** Patients with known hypersensitivity to any ingredient, including: Lanolin, Cetostearyl Alcohol, and Hydroxybenzoates, Phenoxyethanol, Isopropyl Palmitate and Benzyl Alcohol, Triethanolamine; **Side effects:** Rarely mild reactions such as skin rash can occur. Allergic reactions. **Product classification:** Class I Medical Devices. **Cost:** Epimax Moisturising Cream: £2.99 (500g), £1.99 (100g) [NHS Price List]; **License holder:** Aspire Pharma Ltd, Unit 4, Rotherbrook Court, Bedford Road, Petersfield, Hampshire, GU32 3QG, UK. **Date reviewed:** February 2021. **Version number:** 1010451581 v 6.0.

References: * Our 500g creams and gel formulations are packaged in bottles and our ointments in 500g tubs 1. <http://mims.co.uk> (accessed March 2021)

10104511137 v 1.0 March 2021

ASPIRE
PHARMA

Dermatosis papulosa nigra

Dr Monica Anyasodor offers a quick overview of the clinical features and management options for this condition, commonly seen in darker skin



DR NIGEL STOLLERY

Tiny brown papules on the right side of the neck

Dermatosis papulosa nigra is a benign skin condition that is commonly seen in people with darker skin (skin types IV–VI). It is considered to be a variant of seborrheic keratoses.

CLINICAL FEATURES

Dermatosis papulosa nigra presents as tiny brown or black papules or plaques that measure up to 5mm in diameter (see images, above).

The lesions are asymptomatic and have a predilection for the cheeks, forehead and temples.

The condition can start in adolescence and tends to be progressive, with individuals gradually developing more lesions as they get older. However, the extent to which patients are affected varies a great deal. Some people only have a few lesions while others have multiple papules.

Key learning points

- ▶ Dermatitis papulosa nigra is a benign skin condition.
- ▶ It presents as small smooth brown or black papules or plaques, most commonly on the cheeks, forehead and temples.
- ▶ The condition is progressive with age.
- ▶ Women are more commonly affected than men.
- ▶ Some patients may want the lesions removed for cosmetic reasons.
- ▶ Patients considering destructive treatment should be advised of the risks of scarring and pigmentary changes.



DR SHARON BELMO

Lesions commonly occur on the cheeks and temples

Women are more commonly affected than men and patients often have a family history of the condition.

Dermatosis papulosa nigra is a clinical diagnosis, but skin biopsy can be taken if there is any diagnostic uncertainty.

MANAGEMENT

Dermatosis papulosa nigra is asymptomatic and, because it is a benign condition, does not necessarily require treatment.

Some individuals may seek to have the lesions removed according to cosmetic preference.

Options for removal include:

- electrodesiccation
- cryotherapy
- curettage and cautery
- laser therapy.

Those seeking treatment for dermatosis papulosa nigra should be counselled adequately. The cosmetic benefits of removing the lesions must be balanced with the risk of scarring, including keloid scarring (see page 29 for more information on keloids). Permanent pigmentary changes are also more common in darker skin types (see page 14 for more on post-inflammatory hyperpigmentation) and this should be considered before embarking on any treatment.

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Paediatric dermatology

Dr Aarthi Uthayakumar and Dr Bisola Laguda highlight common skin conditions in children of colour, including clinical features to look out for and advice on management



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Eczema: erythema is often subtle in dark skin

ECZEMA

Atopic dermatitis is the most common inflammatory disease affecting children worldwide. In a study of African-Caribbean children in South East England, atopic eczema and tinea capitis were the most common dermatoses, comprising 63% of diagnoses recorded (see further reading for more information).

The National Health and Nutrition Examination Survey in the USA found that 19.3% of African-American children have atopic dermatitis, compared with 16.1% of white and 7.8% of Asian children.

Genetic and environmental factors play a role in the complex pathogenesis of eczema. Filaggrin is an important protein in the stratum corneum, responsible for maintaining skin moisture. In African-American children there is a reduction in filaggrin metabolites and uncommon filaggrin variants, which contribute to atopic dermatitis. In addition, these children have reduced ceramide levels and increased transepidermal water loss, contributing to xerosis.

Clinical features

- Atopic dermatitis typically presents during infancy and early childhood; 85% of cases occur before the age of 1 year and 95% before the age of 5 years.
- Common sites include cheeks, neck, scalp and extensor surfaces (rather than typical flexor surfaces).
- Erythema is usually subtle in very dark skin (see image, above). Rather, active eczema may present with darker brown, purple or ashen colours. Gentle pressure to blanch areas of involvement may help to reveal subtle erythema on removal of pressure.
- Papular eczema is more common in darker skin types, often on the limbs and periumbilical area.
- Follicular accentuation is frequently observed in darker skin types.
- A lichen planus-type appearance has been reported in darker skin types, as a result of lichenification and difficulties in perceiving erythema in darker skin.
- Prurigo-type lesions are more common in skin of colour, often on the lower limbs.
- Discoid eczema is more common in skin of colour.
- Post-inflammatory hyperpigmentation and hypopigmentation are particularly evident in children of



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Pityriasis alba: onset is typically 3-16 years of age

colour, and this discolouration can last for months to years, even after the eczema is treated.

- As for all children with eczema, its effect on sleep and school attendance should be explored.

Management

- Avoid very hot or cold showers and fragrances.
- Use regular emollients – ointments and creams are preferable to water-based lotions.
- Topical steroids can be used for active flares.
- Steroid-sparing agents, such as topical tacrolimus and pimecrolimus, can be used for flares and/or maintenance treatment.
- Occlusion therapy with wet wraps can be used at night on top of emollients and topical steroids.
- For recurrent secondary skin infection, consider topical/oral antibiotics and bleach baths.
- Phototherapy is an option.
- Oral immunosuppression may be helpful in recalcitrant cases.
- Dupilumab (IL-4 and IL-13 inhibitor) is approved for moderate-severe atopic dermatitis in children aged 6 years and older.
- Oral antihistamines (particularly sedating) can be helpful for persistent pruritus, especially when sleep is affected.

PITYRIASIS ALBA

Pityriasis alba is a benign skin condition for which the exact cause is unknown, but it may represent a mild eczematous dermatitis.

There is no clear sex predilection, and triggers include heat, detergents and micro-organisms, such as *Pityrosporum*, *Staphylococcus* and *Streptococcus*. It can be a manifestation of post-inflammatory hypopigmentation in atopic dermatitis.

Clinical features

- Onset is typically 3-16 years of age.
- Poorly-defined, hypopigmented macules and patches with minimal scale (see image, above).
- Lesions can vary in size from 0.5-5cm and are associated with xerosis. They are usually symmetrically distributed.



Dermal melanocytosis on the thigh of an Asian baby, showing an evenly-pigmented, blue-grey patch

- Early lesions may present with mild inflammation and erythema, which is often subtle in skin of colour.
- Lesions are typically symmetrical and located on the face, though the chest, back and upper arms can also be affected.
- There may be concurrent atopy – pityriasis alba may precede other clinical findings of atopic dermatitis.
- It usually worsens in summer with sun exposure.

Management

- The majority of cases undergo spontaneous remission by adulthood.
- Sun protection is important.
- Low-potency topical steroids and calcineurin inhibitors can be effective.

TINEA CAPITIS

Tinea capitis is a dermatophyte infection of the scalp and hair, most commonly caused by *Trichophyton tonsurans* in the UK but, worldwide, the most common cause is *Microsporum canis*.

This can be acquired through contact with infected animals, especially cats and dogs. It can also spread through human-human contact, including via objects such as combs or towels.

Clinical features

- Most often occurs in children 3-7 years of age.
- Some forms present with discrete plaques of dry, flaky, erythematous skin on the scalp.
- It is often itchy and there may be associated alopecia.
- The hairs are often broken, fragile, and easily pulled out (visible as black dots on the scalp).

- There may be a boggy, inflamed swelling on the scalp, known as a kerion.
- More severe forms may present with inflammatory papules, pustules and systemic symptoms, including fever and malaise.
- Cervical and occipital lymph nodes may be palpable.

Management

- Oral antifungals are required for tinea capitis because the organism invades the hair follicle, which topical preparations cannot reach. Griseofulvin is the only licensed treatment for tinea capitis in children. However, terbinafine is often more effective against *T. Tonsurans*. See table, overleaf, for dosages.
- Shampoo containing selenium sulphide, in addition to oral treatment, helps to loosen scale and reduce shedding of spores.
- Test of cure fungal scraping should be performed.

CONGENITAL DERMAL MELANOCYTOSIS

Congenital dermal melanocytosis – also known as Mongolian blue spot – is found in approximately 90% of children of colour.

The lesions should be photographed and documented as they can occasionally be confused with bruising, leading to suspicions of abuse.

Clinical features

- Ill-defined, evenly-pigmented blue-grey patches (see image, above), typically on the lower back/sacral region.
- Lesions range in size from 1-10cm.

CHILDHOOD SKIN CONDITIONS

- Lesions are present at birth and reach peak diameter by 2 years of age.
- Pigmentation is usually most obvious at 1 year.

Management

- Dermal melanocytosis tends to fade spontaneously over time. Larger patches may persist longer than smaller ones.

TRANSIENT NEONATAL PUSTULAR MELANOSIS

Transient neonatal pustular melanosis is a benign idiopathic disorder of neonates and occurs most commonly in African-Caribbean neonates.

Clinical features

- Presents at birth with a combination of pustules, hyperpigmented macules, and collarettes of scale.
- There are no constitutional symptoms.
- Pustules have no surrounding erythema and rupture easily with mild friction.
- Any site (including palms and soles) can be affected, but common sites include the chin, neck, upper chest, buttocks and lower back.
- Lesions last 24-48 hours and spontaneously resolve within 2 weeks.
- Hyperpigmented macules may persist for 3 weeks to 3 months.
- The pustules and lesions are present in utero and new lesions do not form after delivery.

Management

- Lesions resolve spontaneously and no active management is required.

KAWASAKI DISEASE

Kawasaki disease is a multisystem vasculitis, usually affecting children younger than 6 years (peak incidence is under 2 years of age).

The aetiology is likely to be viral, which provokes an abnormal immunologic response (see image, below). Most cases occur in children of Asian ancestry.

Clinical features

- Fever for at least 5 days in addition to the criteria listed in the box, right.
- Desquamation occurs during the second week of the illness.
- Elevated inflammatory markers, such as ESR and CRP.



Swelling of the hand of a child with Kawasaki disease

Oral antifungals for tinea capitis

Drug	Dosage
Griseofulvin	▶ 20-25mg/kg for minimum 8 weeks
Terbinafine (off-label use)	▶ 4-8 weeks, weight-based dose: • 10-20kg 62.5mg once daily • 20-40kg 125mg once daily • >40kg 250mg once daily

Criteria for diagnosis of Kawasaki disease

- ▶ Fever for at least 5 days in addition to four of the following criteria:
 - Bilateral conjunctival injection (without exudate)
 - Oral mucosal changes, including 'strawberry tongue', cracked lips, diffuse mucosal erythema
 - Palmar erythema, induration, periungual peeling
 - Polymorphous exanthem, typically a maculopapular or morbilliform eruption affecting the trunk, lower abdomen and groin
 - Cervical lymphadenopathy (usually unilateral)

- Cardiac complications occur in 15-25% of untreated patients, including coronary artery aneurysms, myocarditis and valvular disease (usually mitral or aortic).
- Central nervous system involvement can lead to aseptic meningitis and cranial nerve palsies.
- Gastrointestinal involvement can cause abdominal pain and diarrhoea.

Management

- Treatment is aimed at reducing the risk of cardiac complications. Echocardiogram should be performed at diagnosis and after 4-6 weeks.
- Give IV immunoglobulin (2g/kg) single dose (live vaccines should be deferred for 12 months).
- Aspirin can be used until fever resolves (if there are cardiac complications, aspirin should be continued).

TRACTION ALOPECIA

Traction alopecia is a form of hair loss caused by chronic tension on the hair follicles.

Although traction alopecia is predominantly seen in women of African descent, it is not uncommon in children as a result of hair styling practices. For more on traction alopecia, see [page 22](#).

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Reference: 1. Djokic-Gallagher J., Rosher P., Hart V. & Walker J. Steroid-sparing effects and acceptability of a new skin gel containing the anti-inflammatory medicinal substance-nicotinamide. Clinical, Cosmetic and Investigational Dermatology 2019;12:545-552.

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days of treatment, however, if they are troublesome or persist, stop using and seek medical advice. Rarely skin irritation (mild rashes) or allergic skin reactions can occur on extremely sensitive skin, these tend to occur during or soon after the first few uses and if this occurs stop treatment. Vitamin B derivative requirements are increased during pregnancy and infancy, however, with prolonged use over significant areas, it may be possible to exceed the minimum recommended levels of nicotinamide in pregnancy. Safety trials have not been conducted in pregnancy and breast feeding therefore, as with other treatments, caution should be exercised, particularly in the first three months of pregnancy.

Instruct patients not to smoke or go near naked flames. Fabric (clothing, bedding, dressings etc) that has been in contact with this product burns more easily and is a potential fire hazard. Washing clothing and bedding may reduce product build-up but not totally remove it.

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Skin cancer in skin of colour

Skin cancer occurs less frequently in skin of colour, but patients experience disproportionate mortality and morbidity. **Dr Arif Aslam** explains why



DR NIGEL STOLLERY

Pigmented BCC with a pearly, rolled border

Skin cancer is the most common malignancy in the UK and includes malignant melanoma (MM) and non-melanoma skin cancer (NMSC). In the USA, the incidence of MM and NMSC is approximately 5% in Hispanic people, 4% in Asian people, and 2% in African-American people, compared with 40% in white people.^{1,2} Unfortunately, there are no such figures for the UK.

Although skin cancer occurs less frequently in skin of colour than in lighter skin, the diagnosis is often associated with a disproportionately higher mortality and morbidity.³ This poor prognosis is multifactorial but can partly be attributed to clinical presentations that vary from those observed in white skin. This often leads to delayed diagnoses and more advanced disease at presentation, ultimately resulting in poorer outcomes for people of colour.

The proportion of non-white individuals is set to rise in the UK. Therefore, it is imperative that clinicians become more familiar with the prevalence and variation in clinical presentation of skin cancer among the UK's non-white population.

SQUAMOUS CELL CARCINOMA

Squamous cell carcinoma (SCC) is the most common skin cancer in African-American people and the second most common skin cancer in white people, Asian people and Hispanic people.³

Risk factors include immunosuppression and chronic UV radiation (UVR) exposure, with chronic scarring and inflammatory disease accounting for 20-40% of cases.^{1,3}

In skin of colour, the metastatic rate of an SCC from a chronic scarring process is 20-40%, compared with 1-4% when SCCs develop from UVR in white skin.¹

The distribution and clinical features of SCCs are also different in skin of colour. For example, SCCs predominantly present in sun-protected sites in skin of colour, in contrast to sun-exposed sites in most white people. Common sites of SCC in people of colour include the lower legs and anogenital region (in almost one quarter of cases) and these sites demonstrate increased potential for metastasis.

In white people, SCC has a spectrum of morphologies varying from poorly-defined rough pink patches to well-circumscribed hyperkeratotic papules or plaques.⁴ They may present as an ulcerated plaque arising from an elevated indurated base. However, in skin of colour, SCC can appear as keratotic brown or black hyperpigmented plaques with thinner hyperpigmented papules often accompanying the primary lesion. The surrounding skin can have a mottled appearance and erythema is not a prominent feature.

Another notable feature of SCC in skin of colour is that it can present in the context of other cutaneous pathology, such as discoid lupus erythematosus.

BASAL CELL CARCINOMA

Basal cell carcinoma (BCC) is the most common skin cancer in white people, Hispanic people and Asian people, but the second most common skin cancer in African-American people.^{1,5}

It has been predominantly studied in white skin and is primarily related to prolonged UVR exposure.

On white skin, BCC commonly presents on sun-exposed sites as asymptomatic, translucent solitary nodules with telangiectasia, central ulceration and rolled, pearly borders. In skin of colour, BCC may also present on non-exposed sites.



Acral lentiginous melanoma is most commonly reported on the plantar foot

The histological subtypes of BCC in skin of colour are similar to those seen in lighter skin. They can also present similarly, but may have several distinguishing features in skin of colour, including the propensity towards multiple tumours and pigmentation – pigmentation is present in more than 50% of BCCs in skin of colour (see image, left), compared with only 5–6% of BCCs in white skin.⁶

Telangiectasias and a pearly, rolled border may be difficult to discern in dark skin or in a pigmented tumour. As a result, BCCs in skin of colour are often misdiagnosed as seborrheic keratosis or MM.

Morpheaform BCCs represent only 3% of all BCC cases but their aggressive clinical behaviour warrants prompt recognition. Morpheaform BCCs present similarly in skin of colour and in white skin; as indurated, shiny atrophic plaques on the head and neck, with indistinct borders. Pigmentation may also be a feature.

MALIGNANT MELANOMA

MM accounts for only 1% of all skin cancer cases, but it is the cause of the majority of skin cancer deaths (75%).^{3,6}

While MM primarily affects white people and is less common in people of colour, the diagnosis is often associated with a disproportionately increased mortality and morbidity in the latter population.

When diagnosed early, melanoma is associated with favourable survival rates in comparison with the poor prognosis of advanced-stage melanoma. A population-based clinical outcome study reported that people of colour are 1.5 times more likely to die from melanoma than white people. These poor outcomes are attributed, at least in part, to the different clinical presentation in skin of colour, with regard to both demographics and tumour characteristics.⁶

Studies show white people present at a younger age compared with people of colour.^{3,6} Melanoma in skin of colour

presents more commonly on the lower extremities, whereas it is more common on the trunk in white people, and is more frequently associated with ulceration, an important predictor of survival. Melanoma is also more commonly located on the lips and mucosal surfaces in people of colour.⁶

The acral lentiginous melanoma subtype is significantly more common in skin of colour and presents as an irregular dark brown pigmented patch on the palmar, plantar, subungual, and mucosal surfaces, with the plantar foot being the most common reported location (see image, left).

While current evidence supports UVR as an important risk factor for melanoma in white populations, the evidence for this association in skin of colour is less clear, as are current recommendations promoting UV protection for melanoma prevention in skin of colour (see page 9 for more information).⁷

Historically, skin cancer studies have focused on white skin, but, with the increasing diversity of the UK population and known disproportionate mortality and morbidity of skin cancer among people of colour, it is important that our knowledge of skin cancer prevalence and clinical presentation in this group is enriched through teaching, training and research.

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Key learning points

- ▶ People of colour experience disproportionately high mortality and morbidity associated with skin cancer.
- ▶ SCCs predominantly present in sun-protected sites in skin of colour.
- ▶ Pigmentation is present in more than 50% of BCCs in skin of colour.
- ▶ BCCs in skin of colour are often misdiagnosed as seborrheic keratosis or MM.
- ▶ Melanoma in skin of colour presents more commonly on the lower extremities (compared with the trunk in white people).
- ▶ UVR exposure is an important melanoma risk factor in white populations, but evidence for this association in skin of colour is less clear.

Prescribing Information – Please refer to the Summary of Product Characteristics (SmPC) before prescribing.

Metoject PEN (methotrexate) 7.5 mg, 10mg, 12.5mg, 15mg, 17.5mg, 20mg, 22.5mg, 25mg, 27.5mg, 30mg solution for injection in pre-filled pen

Qualitative and quantitative composition: 1 pre-filled pen with 0.15 ml (0.20 ml, 0.25 ml, 0.30 ml, 0.35 ml, 0.40 ml, 0.45 ml, 0.50 ml, 0.55 ml, 0.60 ml) contains 7.5 mg (10 mg, 12.5 mg, 15 mg, 17.5 mg, 20 mg, 22.5 mg, 25 mg, 27.5 mg, 30 mg) methotrexate. Excipients: NaCl, NaOH, HCl, water for injections.

Therapeutic indications: Active rheumatoid arthritis in adult patients; polyarthritic forms of severe, active juvenile idiopathic arthritis, when the response to nonsteroidal anti-inflammatory drugs (NSAIDs) has been inadequate, moderate to severe psoriasis in adult patients who are candidates for systemic therapy, and severe psoriatic arthritis in adults; mild to moderate Crohn's disease either alone or in combination with corticosteroids in adult patients refractory or intolerant to thiopurines.

Posology and method of administration: Should only be prescribed by physicians who are familiar with the various characteristics of the medicinal product and its mode of action. Patients must be educated to use the proper injection technique. The first injection of Metoject PEN should be performed under direct medical supervision. **Adults, rheumatoid arthritis:** The recommended initial dose is 7.5 mg of Metoject once weekly, administered subcutaneously. Depending on the individual activity of the disease and tolerability, the dose may be increased gradually by 2.5 mg per week. A weekly dose of 25 mg should in general not be exceeded. **Polyarthritic forms of juvenile idiopathic arthritis:** The recommended dose is 10–15 mg/m² body surface area (BSA)/once weekly, administered subcutaneously. In therapy-refractory cases the weekly dosage may be increased up to 20 mg/m² BSA/once weekly. Use in children < 3 years of age is not recommended as insufficient data on efficacy and safety is available for this population. **Psoriasis vulgaris, psoriatic arthritis:** Test dose of 5 – 10 mg should be administered parenterally, one week prior to therapy to detect idiosyncratic adverse reactions. The recommended initial dose is 7.5 mg of methotrexate once weekly, administered subcutaneously. The dose is to be increased gradually but should not, in general, exceed a weekly dose of 25 mg of methotrexate. **Crohn's disease:** Induction treatment: 25 mg/week administered subcutaneously. Response to treatment can be expected after approximately 8–12 weeks. Maintenance treatment: 15 mg/week. **Elderly:** Dose reduction should be considered due to reduced liver and kidney function as well as lower folate reserves. If changing the oral to parenteral administration, a reduction of dose may be required due to the variable bioavailability.

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Special warnings and precautions for use: In the treatment of rheumatoid arthritis, juvenile idiopathic arthritis, psoriasis and psoriatic arthritis, and Crohn's disease, Metoject PEN (methotrexate) must only be used once a week. Dosage errors in the use of Metoject PEN (methotrexate) can result in serious adverse reactions, including death.

Undesirable effects: Most serious adverse reactions of methotrexate include bone marrow suppression, pulmonary toxicity, hepatotoxicity, renal toxicity, neurotoxicity, thromboembolic events, anaphylactic shock and Stevens-Johnson syndrome. Most frequently (very common) observed adverse reactions of methotrexate include gastrointestinal disorders (e.g. stomatitis, dyspepsia, abdominal pain, nausea, loss of appetite and abnormal liver function tests (e.g. increased ALAT, ASAT, bilirubin, alkaline phosphatase). Other frequently (common) occurring adverse reactions are leukopenia, anaemia, thrombopenia, headache, tiredness, drowsiness, pneumonia, interstitial alveolitis/pneumonitis (often associated with eosinophilia, oral ulcers, diarrhoea, exanthema, erythema and pruritus). **Effects:** Pharyngitis, infection (incl. reactivation of inactive chronic infection), sepsis, conjunctivitis, lymphoma. Leukopenia, anaemia, thrombopenia, pancytopenia, agranulocytosis, severe courses of bone marrow depression, lymphoproliferative disorders, eosinophilia. Allergic reactions, anaphylactic shock, hypogammaglobulinaemia. Precipitation of diabetes mellitus. Depression, confusion, mood alterations. Headache, tiredness, drowsiness, dizziness, pain, muscular asthenia or paraesthesia, hypoaesthesia, changes in sense of taste (metallic taste), convulsions, meningism, acute aseptic meningitis, paralysis, encephalopathy/leukoencephalopathy. Visual disturbances, impaired vision, retinopathy. Pericarditis, pericardial effusion, pericardial tamponade. Hypotension, thromboembolic events. Pneumonia, interstitial alveolitis/pneumonitis often associated with eosinophilia. Symptoms indicating potentially severe lung injury (interstitial pneumonitis) are dry, not productive cough, short of breath and fever, pulmonary fibrosis. Pneumocystis jirovecii pneumonia, shortness of breath and bronchial asthma, pleural effusion, epistaxis, pulmonary alveolar haemorrhage. Stomatitis, dyspepsia, nausea, loss of appetite, abdominal pain, oral ulcers, diarrhoea, gastrointestinal ulcers and bleeding, enteritis, vomiting, pancreatitis, gingivitis, haematemesis, haematemesis, toxic megacolon. Abnormal liver function tests (increased ALAT, ASAT, alkaline phosphatase and bilirubin), cirrhosis, fibrosis and fatty degeneration of the liver, decrease in serum albumin, acute hepatitis, hepatic failure. Exanthema, erythema, pruritus, photosensitisation, loss of hair, increase in rheumatic nodules, skin ulcer, herpes zoster, vasculitis, herpetiform eruptions of the skin, urticarial, increased pigmentation, acne, petechiae, ecchymosis, allergic vasculitis, Stevens-Johnson syndrome, toxic epidermal necrolysis (Lyell's syndrome), increased pigmentary changes of the nails, acute paronychia, furunculosis, telangiectasia, skin exfoliation/dermatitis exfoliative. Arthralgia, myalgia, osteoporosis, stress fracture, osteonecrosis of jaw (secondary to lymphoproliferative disorders). Inflammation and ulceration of the urinary bladder, renal impairment, disturbed micturition, renal failure, oliguria, anuria, electrolyte disturbances, proteinuria. Inflammation and ulceration of the vagina, loss of libido, impotence, gynaecomastia, oligospermia, impaired menstruation, vaginal discharge. Fever, wound-healing impairment, asthenia, injection site necrosis, oedema. Subcutaneous application of methotrexate is locally well tolerated. Only mild local skin reactions (such as burning sensations, erythema, swelling, discoloration, pruritus, severe itching, pain) were observed, decreasing during therapy. See SmPC for details of other adverse events. **Overdose:** Calcium folinate is the specific antidote for neutralising the toxic undesirable effects of methotrexate.

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Dermatology for skin of colour: learning resources

Dr Sharon Belmo outlines her recommendations for further resources to support your learning about dermatology for skin of colour

WEBSITES

Centre of Evidence Based Dermatology Skin of Colour Resource

nottingham.ac.uk/research/groups/cebd/resources/skin-of-colour

With the support of Professor Hywel Williams and information specialist Douglas Grindlay, I launched the Skin of Colour Resource on the CEBD website in 2016.

This is my own contribution to UK dermatology; an attempt to advance dermatology for skin of colour.

In this free resource, we present a range of dermatological conditions pertinent to skin of colour, supported by clinical images, systematic reviews, review articles and patient information leaflets, where available. The purpose is to provide healthcare professionals with regularly updated, evidence-based information.

You will also find a comprehensive list of dermatology for skin of colour textbooks and websites that I have recommended.

DermNet NZ

dermnetnz.org/topics/skin-conditions-in-skin-of-colour

DermNet NZ is a comprehensive free resource covering all aspects of dermatology. It was founded by Dr Amanda Oakley in 1995 and has become well-known worldwide.

In 2015, DermNet NZ launched a section on 'ethnic dermatology', where a number of conditions that are more common in skin of colour are presented and supported by great clinical images.

IMAGE LIBRARIES

Skin Deep

dftbskinddeep.com

Skin Deep is a subsection of paediatric resource 'Don't forget the Bubbles', which was set up by British and Australian paediatricians in 2013.

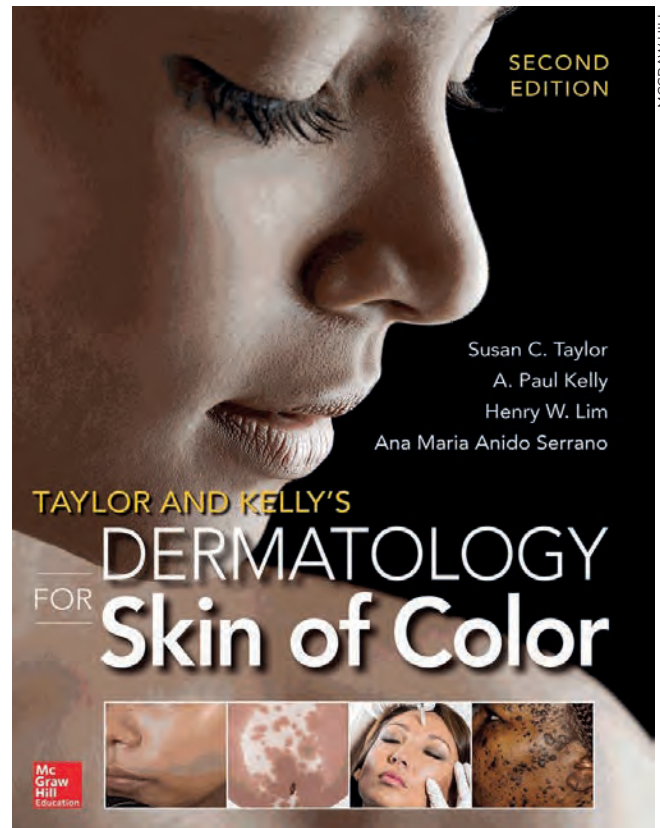
SkinDeep is an excellent showcase of high-quality paediatric skin of colour images from patients seen mainly in paediatric emergency care. This free resource is a great initiative that promotes diversity in medicine.

VisualDx

visualdx.com/diversity

VisualDx is a US-based diagnostic toolkit that can be accessed through a paid subscription. The platform is extensive, covering a wide variety of medical fields, including dermatology.

As part of the subscription, VisualDx offers an impressive skin of colour image atlas showcasing dermatological disorders in darker skin types.



TEXTBOOKS

Dermatology textbooks have traditionally focused on lightly-pigmented skin, with little emphasis on skin of colour. Here I present a few of the dermatology for skin of colour textbooks available.

These have been published in the last decade by some of the pioneering skin of colour dermatologists.

As a UK-trained dermatologist, I have personally found such textbooks to be essential to my own skin of colour education. A comprehensive list of textbooks can be found on the CEBD Skin of Colour Resource.

Recommended reading

- Kelly AP, Taylor SC, Lim HC, Serrano AMA. Taylor and Kelly's Dermatology for Skin of Color, 2nd edition. New York: McGraw-Hill Medical; 2016.
- Dadzie OE, Petit A, Alexis AF. Ethnic Dermatology: Principles and Practice. New Jersey: Wiley-Blackwell; 2013.
- Alexis A, Barbosa VH. Skin of Color: A Practical Guide to Dermatologic Diagnosis and Treatment. New York: Springer; 2014.
- Jackson-Richards D, Pandya AG. Dermatology Atlas for Skin of Color. New York: Springer; 2014.
- Taylor SC, Badreshia-Bansal S, Callender VD, et al. Treatments for Skin of Color. Philadelphia: Saunders Elsevier; 2011.
- Silverberg N, Durán-McKinster C, Kwang Tay Y. Pediatric Skin of Color. New York: Springer; 2015.

FURTHER RESOURCES

- Love PB, Kundu RV. Clinical Cases in Skin of Color: Adnexal, Inflammation, Infections, and Pigmentary Disorders (Clinical Cases in Dermatology). New York: Springer; 2015.
- Tay YK, Chan YC. Textbook Of Laser And Light Dermatology In The Asian Skin. Singapore: World Scientific Publishing; 2011.

SOCIETIES

Skin of Color Society USA skinofcolorsociety.org

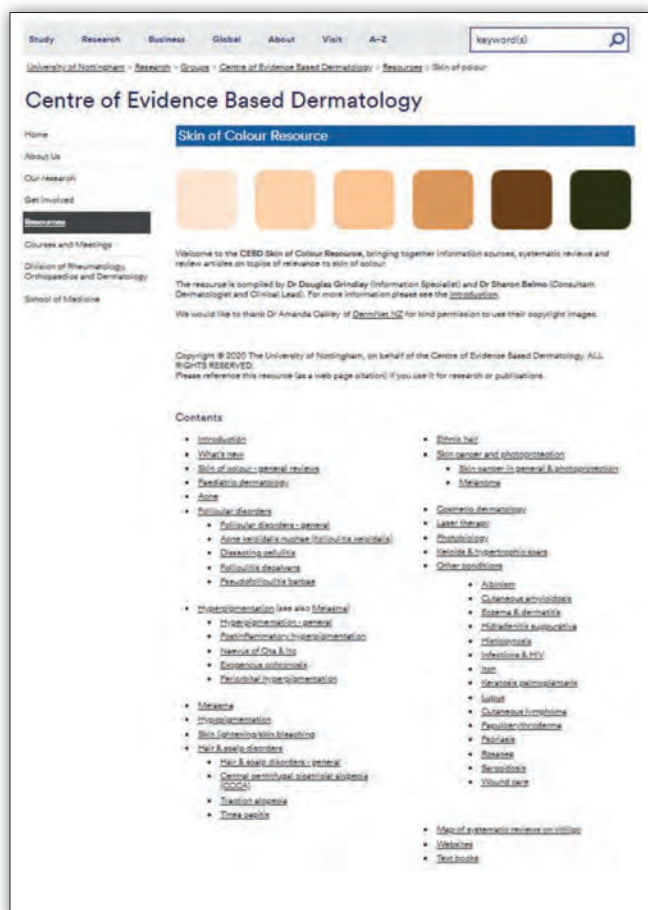
The Skin of Color Society was founded in 2004 by Dr Susan Taylor, a pioneer of dermatology for skin of colour.

Through educational events, research, mentorship, textbooks, symposiums and partnerships with various organisations, the society has raised awareness of dermatology for skin of colour and its importance.

It remains committed to the education of healthcare providers and the general public on dermatological health issues related to skin of colour. Its website provides a variety of educational videos and patient information leaflets, which can be accessed for free.

Dr Sharon Belmo is a consultant dermatologist at Croydon University Hospital and clinical lead for the Centre of Evidence Based Dermatology Skin of Colour Resource

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1 SKIN CANCER IN SKIN OF COLOUR (page 38)

Which of the following features is significantly more common in BCCs in skin of colour, compared with white skin?

- ☐ A Pearly, rolled edges
- ☐ B Ulceration
- ☐ C Telangiectasia
- ☐ D Pigmentation

2 HYPOPIGMENTATION (page 16)

Which of these conditions would not commonly be treated with phototherapy?

- ☐ A Sarcoidosis
- ☐ B Mycosis fungoides
- ☐ C Vitiligo
- ☐ D Post-inflammatory hypopigmentation

3 CENTRAL CENTRIFUGAL CICATRICAL ALOPECIA (page 20)

In which of the following groups does central centrifugal cicatricial alopecia occur most frequently?

- ☐ A Black women in their 30s
- ☐ B Black men in their 60s
- ☐ C Children under the age of 10 years

4 POST-INFLAMMATORY HYPERPIGMENTATION (page 14)

True or false? Post-inflammatory hyperpigmentation (PIH) can be caused by trauma to the skin, and epidermal PIH usually takes longer to fade than dermal PIH.

- ☐ A True
- ☐ B False

5 PAEDIATRIC DERMATOLOGY (page 34)

What percentage of children of colour have congenital dermal melanocytosis?

- ☐ A 1%
- ☐ B 15%
- ☐ C 40%
- ☐ D 90%

6 ACNE KELOIDALIS NUCHAE (page 26)

Which of the following statements about the presentation of acne keloidalis nuchae is correct?

- ☐ A There are discrete hypopigmented lesions
- ☐ B Papules or pustules present on the posterior neck and/or occipital scalp
- ☐ C Comedones are usually present
- ☐ D Lesions are usually painful and itchy on initial presentation

7 PSEUDOFOLLICULITIS BARBAE (page 24)

True or false? There is a genetic predisposition for pseudofolliculitis barbae.

- ☐ A True
- ☐ B False

8 DERMATOSIS PAPULOSA NIGRA (page 33)

Which of the following statements about dermatosis papulosa nigra is correct?

- ☐ A It is most commonly seen in white skin
- ☐ B The papules or plaques commonly measure over 1cm each
- ☐ C The lesions have a predilection for the cheeks, forehead and temples
- ☐ D The lesions are usually itchy and sore

9 MELASMA (page 18)

Which of the following does not pose a risk of inducing or exacerbating melasma?

- ☐ A Tranexamic acid
- ☐ B Autoimmune thyroid disease
- ☐ C UV radiation
- ☐ D Oral contraceptive pills
- ☐ E Phenytoin

10 TRACTION ALOPECIA (page 22)

Which area of the scalp is most commonly affected by traction alopecia?

- ☐ A Fronto-temporal hairline
- ☐ B Vertex
- ☐ C Occipital scalp

11 COMMON CONDITIONS IN SKIN OF COLOUR (page 11)

Which of the following statements about psoriasis in skin of colour is correct?

- ☐ A It presents with salmon pink plaques with an overlying silver scale
- ☐ B The plaques are sharply demarcated
- ☐ C Erythema is a common feature

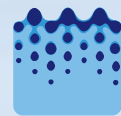
12 KELOIDS (page 29)

What percentage of keloid scars may recur after surgical excision?

- ☐ A 2%
- ☐ B 10%
- ☐ C 25%
- ☐ D 50%
- ☐ E 80%

ANSWERS 1) D 2) A 3) A 4) B 5) D 6) B 7) A 8) C 9) A 10) A 11) B 12) E

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