

SIGNS



IN DARK

SKIN

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Thanks for the photographic contribution:

Acne

Acne keloidalis nuchae

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Acne

Tinea capitis

Tinea corporis

Traction alopecia

Chloasma and melasma

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Cover photo

by Aldo Morrone

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INTRODUCTION

Dermatology, more than any other discipline, relies heavily on visual observation. But what happens when clinical signs manifest on skin neglected by medical texts? This atlas was created to address a concrete lack: the visual representation of cutaneous diseases in dark phototypes.

Throughout the history of dermatology, dark skin has too often remained invisible. Clinical signs, elementary lesions, and manifestations of common and rare diseases – when observed on higher phototypes – may appear different, sometimes muted, sometimes accentuated. Yet in reference texts, iconographic repertoires and even specialist training, representation of this variability has been scarce, partial or absent.

This clinical guide arises from the need – and the responsibility – to fill that gap.

Signs on Dark Skin is a clinical and visual atlas devoted exclusively to higher phototypes. It is not an atlas of “rare” or “exotic” diseases but an essential repertory for improving diagnostic capacity in millions of people who, worldwide and in multicultural contexts, represent the norm – not the exception.

It brings together real images from clinics, hospitals, urban and rural dermatology services and health projects in Africa, Asia, Latin America and Europe, accompanied by essential descriptions and practical notes. Natural clinical images were preferred, avoiding filters, misleading lighting or overly “cleaned up” didactic pictures.

Signs on Dark Skin is structured to facilitate the visual and clinical identification of dermatological manifestations in dark phototypes (Fitzpatrick V–VI), also including intermediate phototypes where features may significantly differ from light skin.

Dual objective:

1. Strengthen clinical competence in diagnosing on dark skin, reducing delays or errors due to incomplete representation;
2. Encourage broader reflection on transcultural medicine, where skin color is not a shadow on diagnosis but a real datum to know and value.

The aim is not only to show but to educate the clinical eye. An atlas is not merely a collection of images but an invitation to reconsider the act of observing, to train the clinical eye, practice equitable diagnosis and care with competence and respect.

It is not only about observing but about seeing better: recognizing, including and respecting the clinical, biological and cultural specificities that skin carries.

Signs on Dark Skin is intended for physicians, dermatologists, nurses, students and health workers who want to refine a more competent, fair and aware gaze.

We hope this volume will become not only a practical tool but also a symbol of a more complete, more global, and above all more just dermatology.

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Author of more than 600 peer-reviewed scientific publications and 70 books, his work bridges clinical and scientific excellence with social justice and humanitarian commitment.

He served as Vice President of the International Society of Dermatology (2013–2017) and is Co-Chair of its Community Dermatology Committee.

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ACNE AND ROSACEA

ACNE

Sebaceous glands are neither more numerous nor larger in subjects with pigmented skin in comparison with the so-called Caucasians. No significant differences in sebaceous gland activity between Caucasian and pigmented skin have been recorded. The amount of free fatty acids and triglycerides is similar in all ethnicities. Wax ester amount is higher in sebum of African-American women.

Epidemiology of acne is similar in both high and low phototypes.

Anatomic distribution and morphology of acne lesions is superimposable in all ethnicities. A lower incidence of nodular acne in higher phototypes has not been confirmed.

Pomade acne is more frequent in females rather than males. It is caused by the chronic application of oily products to smooth the hair. It is characterized by closed comedones, with some papules and rare pustules, located mainly on the forehead and temples. Pomade acne is often associated with traction alopecia.

Post-inflammatory hyperpigmentation is characterized by more or less numerous brown macules, of different morphology and size, located mainly on the face; involvement of neck, shoulders, chest and back is less frequent. Post-inflammatory hyperpigmentation is the final clinical result of: a) previous or concomitant, long lasting, inflammatory lesions of acne; b) chronic irritant/allergic contact dermatitis caused by topical anti-acne products and drugs; c) chronic scratching and d) chronic exposure to ultraviolet rays: the latter is considered as the most important risk factor.

Scars and keloids occur in >50% of acne patients with pigmented skin: both genders are equally involved. Ice picks, boxcars and rolling scars usually occur on the face, in particular the cheeks; hypertrophic scars are more common on the shoulders, chest and back. The development of hypertrophic scars and keloids would be caused by the presence of more numerous, larger and often binucleated or multinucleated fibroblasts and a reduced activity of collagenases.

The therapy of acne is superimposable in all phototypes. However, minocycline must not be used in acne patients with pigmented skin: it is responsible for severe and sometimes fatal cases of *drug reaction with eosinophilia and systemic symptom syndrome* (DRESS syndrome). The latter is a hypersensitivity reaction to the drug, characterized by macular-papular rash, fever, pharyngitis, lymphadenopathy, involvement of heart, lungs, liver, pancreas and kidneys. It appears from 3 to 8 weeks after the beginning of the therapy. Mortality can reach 20% of patients and it is often caused by liver failure. A genetic predisposition that causes abnormalities of immune response has been hypothesized. Triggering factors, such as herpes virus and drugs, have been reported. Abnormalities in minocycline metabolism, such as failure to eliminate the drug or its metabolites, have also been suggested.





ROSACEA

Rosacea would be rare in dark-skinned subjects in comparison with the so-called Caucasians. The reasons for this are unknown.

However, in very dark skin, rosacea is also less visible, in particular the erythemato-telangiectatic variety.

Clinical presentation is superimposable in all ethnicities.



ALLERGIC DISEASES

ALLERGIC CONTACT DERMATITIS

EPIDEMIOLOGY

Allergic Contact Dermatitis is equally prevalent across skin types but is frequently under-recognized in darker phototypes because erythema is subtle or invisible.

CLINICAL FEATURES IN DARK SKIN

Feature	Pigmented / Dark Skin.
Erythema	Violaceous, gray, or dark brown; may appear dull and less inflamed.
Vesiculation	Less evident, replaced by papular or scaly morphology.
Scaling	Ashy-gray scaling prominent.
Lichenification	Marked thickening, accentuated skin lines, hyperpigmentation.
Post-inflammatory change	Severe and prolonged dyschromia, sometimes more noticeable than active dermatitis.

COMMON ALLERGEN SOURCES BY REGION / CULTURAL CONTEXT

Region / Population	Frequent Allergens	Exposure Context
Sub-Saharan Africa	Nickel, chromates, PPD, rubber accelerators, hair relaxers	Jewelry, cement, hair salons
Middle East / South Asia	Fragrances, attar, henna dyes, cosmetics, mercury in skin lighteners	Traditional beauty rituals
Latin America	Paraphenylenediamine, topical antibiotics (neomycin), rubber, balsam of Peru	Hair dyes, topical medicaments
Western populations of African descent	PPD, fragrance mix, preservatives	Hair products, perfumes, nail cosmetics

CLINICAL PEARLS IN DARK SKIN

- The erythematous component is easily missed — inflammation is inferred from texture, swelling, or warmth.
- Papular or follicular Allergic Contact Dermatitis is more frequent in African and Asian populations.
- Chronic lesions show diffuse hyperpigmentation with ill-defined borders, often mistaken for chronic eczema or lupus erythematosus.
- Pruritus is intense and may lead to excoriations, prurigo-like nodules, or secondary bacterial infection.

DIAGNOSTIC CHALLENGES IN DARK SKIN

- Erythema absent → induration and scaling are key diagnostic clues.
- Hyperpigmentation may persist and obscure lesion borders.
- Patch testing is equally reliable but interpretation differs:
 - *Positive reactions* may present as papular or violaceous infiltrates rather than red vesicles.
 - The 1+, 2+, 3+ grading still applies; focus on induration and papulation.
- Differential diagnoses include:
 - *Lichen planus pigmentosus, chronic eczema, discoid lupus, fixed drug eruption, frictional dermatitis.*

PSYCHOSOCIAL ASPECTS

- Pigment alteration on face, neck, or scalp is often more distressing than the dermatitis itself.
- Misinformation and stigma (believing it is infection or contagion) may lead to social withdrawal.
- In women, facial or scalp hyperpigmentation may interfere with professional or social life; psychological support is vital.



ATOPIC DERMATITIS

EPIDEMIOLOGY

- Global prevalence: 10–20% in children, 2–10% in adults; slightly lower in African and Asian populations but often underreported because mild and atypical presentations are misdiagnosed.
- Higher prevalence and severity have been observed in people of African descent living in urban and industrialized settings (e.g., African-Americans, Afro-Caribbeans, Ethiopian highlanders, urban West Africans).

CLINICAL FEATURES IN DARK SKIN

Feature	Pigmented / Dark Skin.
Erythema	Violet, violaceous, gray, or dark brown — sometimes invisible to the naked eye.
Papules and plaques	Papular or follicular pattern common; lesions may appear dry, rough, and “ashy”.
Lichenification	More pronounced, with hyperpigmented thickened plaques and “lichenified sheen”.
Pruritus	Severe, often relentless, leading to excoriations, prurigo nodules, and secondary infections.
Post-inflammatory change	Marked hyperpigmentation or hypopigmentation, lasting months or years.
Distribution	Extensor, face, neck, upper trunk; follicular eczema common.
Acute lesions	Papulovesicular, may appear darker without visible erythema.

TYPICAL PRESENTATION IN DARK SKIN

- Follicular eczema: grouped papules around hair follicles on trunk or limbs.
- Papular prurigo-like form: in African and Caribbean patients, lesions resemble prurigo nodularis.
- Facial eczema: periocular, perioral, and forehead involvement more frequent.
- Neck lichenification (“dirty neck” appearance) often striking in darker phototypes.

DIAGNOSTIC CHALLENGES

- Erythema may be invisible — lesions appear gray, violaceous, or hyperpigmented, leading to underestimation of disease activity.
- Excoriations, prurigo nodules, and dyschromia may mask the classic eczematous pattern.
- Differential diagnoses in dark skin:
 - *Lichen planus, lichen simplex chronicus, pityriasis alba, tinea incognita, scabies, prurigo nodularis, cutaneous lupus erythematosus.*
- Histopathology can help: chronic spongiotic dermatitis with hyperkeratosis and acanthosis; melanophages in dermis reflect post-inflammatory pigmentation.

PSYCHOSOCIAL AND CULTURAL ASPECTS

- Stigma and visible dyschromia (particularly on face and neck) cause significant emotional distress and social isolation.
- In some cultures, Atopic Dermatitis lesions or pigmentation are misattributed to “infection,” “curse,” or “dirtiness”, leading to delayed care or harmful traditional treatments.
- Sleep disturbance due to intense pruritus is more common, affecting quality of life and mental health.
- Counseling and patient education are essential components of care, alongside community sensitization programs.

KEY POINTS FOR CLINICAL PRACTICE

- Recognize that absence of visible redness ≠ absence of inflammation.
- Pruritus, xerosis, and texture are more reliable diagnostic cues than erythema.
- Always consider psychological burden and cultural beliefs when managing Atopic Dermatitis in dark-skinned patients.
- Address dyschromia proactively — both hyper- and hypopigmentation profoundly affect quality of life.
- Patient and family education remains the cornerstone of effective long-term control.



AUTOIMMUNE DISEASES

DISCOID LUPUS ERYTHEMATOSUS

This disease is of common observation in Sub-Saharan populations, in whom lack of photoprotection is widespread.

Both genders are involved in sun exposed areas, the shoulders and back.

The appearance of atrophic-hypopigmented, whitish lesions, of different morphology and size, is early. Involvement of the scalp results in scarring alopecia.

Degeneration into squamous cell carcinoma is not uncommon in long-lasting lesions.

No reliable data are available about the possible evolution of discoid lupus erythematosus into systemic lupus erythematosus.





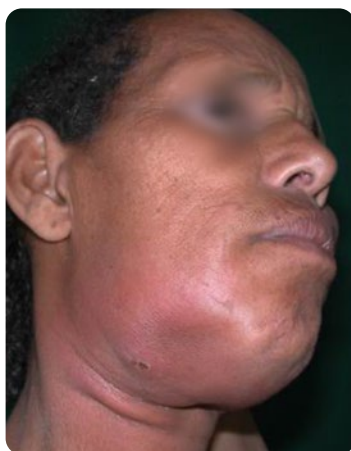
BACTERIAL DISEASES

ERYSIPELAS

Erysipelas is of not rare observation in Sub-Saharan adult patients. Also in these patients, typical locations of this infection are the face and legs, although the forearms, hands and thighs may be involved.

Bacteriological studies carried out in black Africa are scarce; however, it is possible that *Streptococcus pyogenes* is still the most frequently implicated agent.

Clinical picture is characterized by the acute appearance of a severe erythematous oedema, with well defined borders, warm to the touch and parenchymatous—hard in consistency. Pain is often severe. Fever is usually present. This cutaneous picture may be followed by the appearance of vesicular, bullous and/or pustular lesions. The latter are followed by erosions and/or ulcers. Cases of amputation of a leg, because of the shortage of systemic antibiotics, were and are possible.



ERYTHRASMA

Hot-humid climate is an important predisposing factor.

Axillae, submammary and inguinal folds are the typical locations. The clinical picture is characterized by brownish macules, mild erythema, irregular surface because of the presence of tiny scales and well-defined borders. A chronic clinical course is common.

Patients with erythrasma usually complain of mild itching.



IMPETIGO

In Sub-Saharan populations, impetigo is very common, especially in children. Hot-humid climate and poor environmental and personal hygiene play an important etiopathogenetic role.

Impetigo may be a primary infection, usually caused by *Streptococcus pyogenes* or, less frequently, by Gram-negative bacteria, or the final clinical result of previous injuries.

Erythema is mild or invisible. The first bullous-pustular lesions are followed by chronic squamous-crusted-verrucous lesions. Mild symptoms and the shortage of topical and systemic antibiotics explain the very common widespread involvement.



BULLOUS DISEASES

BULLOUS PEMPHIGOID

As for pemphigus, no epidemiological data are available about incidence and prevalence of bullous pemphigoid in African patients.

Bullous pemphigoid is the most common autoimmune blistering disease, characterized by tense bullae on an erythematous or urticarial base and intense pruritus. It primarily affects older adults, but it may sometimes occur in younger individuals, including children.

It may present as urticarial, eczematous or papular lesions, frequently misdiagnosed as eczema or scabies. On dark skin, erythematous background may appear violet, coppery or gray-brown rather than red. Scratching produces hyperpigmented, lichenified plaques before the appearance of blisters. The surrounding erythema is subdued or absent; blisters appear as dark-yellow, grayish or violaceous in colour. Healing results in hyper- or hypopigmented macules. Older erosions show brown or slate-coloured crusts, sometimes confused with impetigo or insect bites. This abnormal postinflammatory pigmentation may persist for months.

The occurrence of skin bacterial superinfections due to chronic scratching is frequent.



PEMPHIGUS

Incidence and prevalence of pemphigus in Afro-Americans are similar to those of the so-called Caucasians. No recent epidemiological data are available for pemphigus in people living in Sub-Saharan Africa.

Skin and mucous lesions of pemphigus are superimposable in high and low phototypes. However, the frequent observation in African patients of widespread, large bullous-erosive lesions is due to delay in diagnosis and therapy. In addition, hot-humid climate and poor environmental and personal hygiene cause skin bacterial superinfections.

It is possible that pemphigus vegetans is slightly more common in African patients: isolated or grouped, mildly erythematous, roundish papules, located on axillae and inguinal folds, are the most typical clinical manifestations.



FUNGAL DISEASES

PITYRIASIS VERSICOLOR

It is possible that pityriasis versicolor is more common in African males than in females.

Facial involvement is not rare both in adolescents and adults. In patients with dark skin, pityriasis versicolor is characterized almost exclusively by widespread, hypopigmented macules, of different morphology and size. Some lesions may be squamous and crusted. Hyperchromic lesions are rarer and, on darker skin, they are difficult to see. Itching is usually mild or even absent.

Differential diagnosis includes pityriasis alba, post-inflammatory hypochromic lesions and leprosy.



TINEA CAPITIS

Tinea capitis is common in children living in poor areas of Sub-Saharan Africa.

In the past, mainly in Eastern Africa, *Trichophyton soudanense* was, according to the epidemiological point of view, an important etiological agent. Also *Trichophyton schoenleinii*, etiological agent of tinea favosa, currently is of less common observation.

In addition to the typical lesions of alopecia, often roundish in shape and of several centimeters in diameter, scales, crusts and verrucous lesions are common, as final clinical result of scratching and chronic bacterial superinfections.

The inflammatory variety of trichophytic tinea (kerion or kerion celsi) occurs also in adults, in particular cattlemen, on the scalp, beard and upper arms. Pustules, abscesses, nodules and fistulae, warm to the touch and soft in consistency, are often followed by regional lymphadenitis. If left untreated, kerion heals after months or years with scarring alopecia.





TINEA CORPORIS

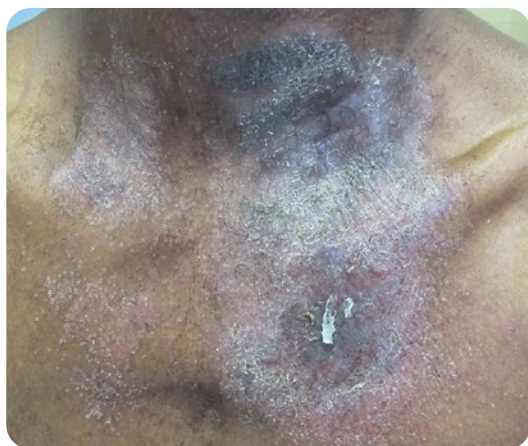
Clinical diagnosis of tinea corporis on dark skin is sometimes difficult because:

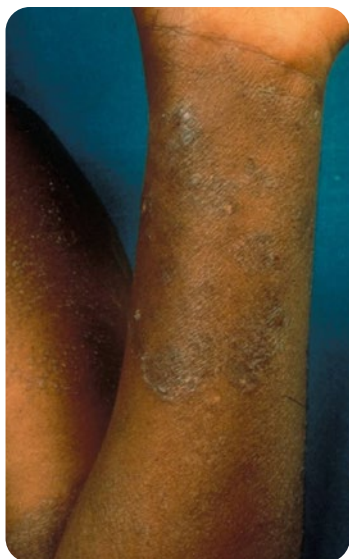
- a. erythema is often mild or even invisible;
- b. borders are not well-defined and often free of vesicles and pustules;
- c. the inner surface of the lesions is characterized by more or less important scaling.

In children with recent lesions the clinical presentation is more typical.

Tinea corporis is often more pruritic in patients living in hot-humid climates. Bacterial superinfections due to chronic scratching are common.

Tinea imbricata is a chronic superficial mycosis caused by *Trichophyton concentricum*. It is endemic in three geographical areas: Southwest Pacific, Southeast Asia and Central and South America. Tinea imbricata is characterized by widespread, annular, concentric, mildly inflammatory, squamous lesions.





TINEA MANUUM

Tinea manuum is common in subjects living in Sub-Saharan Africa, in particular farmers: the soil is the main source of infection.

Although it is obviously possible to observe typical clinical presentations, tinea manuum is very often characterized by a chronic clinical course: this is the reason why mildly inflammatory, squamous-crusted lesions affecting both hands are the most frequently observed features. These lesions initially occur on the back of one hand; afterwards, also palmar surfaces of both hands are infected.

Pruritus is usually mild. In consideration of this clinical picture, diagnosis is often difficult: psoriasis and chronic contact dermatitis must be considered in differential diagnosis.

In chronic tinea manuum, involvement of nails is not rare, as well as the occurrence of tinea corporis because of autoinoculation. Furthermore, cases of co-infection with viral warts are possible.



TINEA PEDIS

On dark skin, the intertriginous variety of tinea pedis is characterized by non-inflammatory, macerated, whitish lesions. The 3rd and 4th interdigital folds, uni- or bilaterally, are usually affected.

The involvement of all interdigital folds and the back of the feet is prevalent: erythema is mild or even absent, while scales and crusts are dominant. The presence of well-defined borders is helpful for the diagnosis.





HAIR DISEASES

ACNE KELOIDALIS NUCHAE

Acne keloidalis nuchae is a chronic cicatricial folliculitis and pseudofolliculitis that mainly affects adult Sub-Saharan, Afro-American and Hispanic males. It is extremely rare in females. An ethnic/family predisposition is possible. Curly hair is also a predisposing factor. Daily and close manual shaving is considered an additional factor.

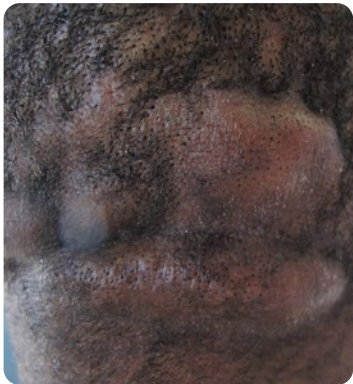
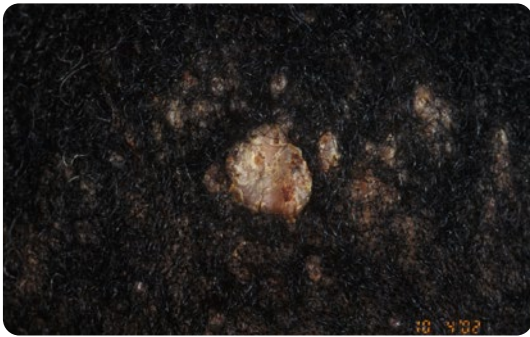
The clinical course is chronic. The first lesions are follicular, mildly inflammatory, hyperkeratotic papules and pustules located on the top of the scalp, occipital area and nape. Sometimes also abscesses and fistulae develop. Subsequently, nodules and plaques, parenchymatous-hard in consistency, appear. Hypertrophic scars, keloids and cicatricial alopecia are the final clinical lesions.

The therapy of acne keloidalis nuchae is based on avoiding daily and close shaving. The use of electric razors, in order to reduce microtrauma, is helpful. In early phases, topical antiseptics, antibiotics and retinoids, as well as oral antibiotics and isotretinoin are often used, although with conflicting clinical results. In late phases, potent topical or intralesional corticosteroids, silicone, electrosurgery, radiotherapy, lasers and surgery are the most frequently used approaches.

We believe that the name of acne keloidalis nuchae should be changed for several reasons:

a) it is not a clinical variety of acne, although some patients are also affected by acne; b) it is not always and only characterized by keloids (hypertrophic scars are more frequent than keloids) and c) it does not always and only involve the nape. Also the names of *acne keloidalis*, *dermatitis papillaris capillitii*, *dermatitis papillaris nuchae*, *folliculitis keloidalis nuchae*, *folliculitis nuchae scleroticans*, *keloid acne* and *nuchal keloid acne*, for the same reasons, are not satisfactory. Based on these last considerations, we suggest a new name for acne keloidalis nuchae: we think that *cicatricial folliculitis and pseudofolliculitis of the scalp in non-Caucasians* could be a better name.





ALOPECIA AREATA

Alopecia areata on dark skin is characterized by:

- a. gray-brown, alopecic patches, sometimes with subtle hypopigmentation or post-inflammatory hyperpigmentation;
- b. minimal visible erythema; diagnosis relies on texture and border palpation;
- c. less conspicuous exclamation mark hairs: they are best detected by dermoscopy or oblique lighting;
- d. regrowing hairs: depigmented vellus hairs are very noticeable on dark skin, producing a “poliosis-like” effect;
- e. post-inflammatory changes: pronounced hyper- or hypopigmentation around healed patches;
- f. scarring: slight atrophy or shine may mimic early scarring alopecia: dermoscopy is essential for differentiation.

Clinical tip: on dark skin, colour contrast between alopecic and normal areas is reduced; dermoscopy is the most reliable diagnostic tool. Regrowing hairs often appear white or hypopigmented, sometimes interpreted by patients as a new disease.

On dark skin, pigmentary sequelae (hyper- hypopigmentation) often persist for a long period of time after regrowth, but hair regrowth itself is structurally normal.

Clinical pearls for pigmented skin:

- a. erythema may be invisible; rely on dermoscopy and palpation rather than colour;
- b. poliosis-like regrowth (white vellus hairs) may cause unnecessary alarm: reassure patients that pigment usually returns;
- c. post-inflammatory hyperpigmentation around patches can obscure borders: use oblique light;
- d. avoid aggressive topical corticosteroids on frontal hairline and temples to prevent hypopigmented halos and atrophy;
- e. psychosocial impact is often severe; early intervention and counseling are very important.



PSEUDOFOLLICULITIS

Pseudofolliculitis is of common observation in adult males with dark skin and curly hair. Typical location is the area of the beard; arms and pubis may also be involved.

The pathogenesis is due to the transfollicular penetration of the hair (the latter does not emerge from the skin surface) and/or the extrafollicular penetration (the hair emerges from the skin surface, but it re-enters the epidermis and dermis).

The clinical picture is characterized by mildly inflammatory, hyperkeratotic papules, parenchymatous-hard in consistency, and pustules. Post-inflammatory hyperpigmented macules are a frequent final clinical result.

The therapy is based on avoiding daily and close shaving. The use of electric razors, in order to reduce microtrauma, is helpful.



TRACTION ALOPECIA

Traction alopecia is caused by chronic pulling forces applied to the hair of the scalp. It is more common in females with dark skin and curly hair. Chronic traction causes a recession of the frontal hairline: differential diagnosis includes alopecia areata and especially frontal fibrosing alopecia.

Traction alopecia is frequently associated with pomade acne: the latter is due to the chronic application of greasy products to smooth the hair. Pomade acne is characterized by closed comedones, with some papules and rare pustules, located mainly on the forehead and temples. Furthermore, the chronic use of oily products can cause irritant/allergic contact dermatitis.

No pharmacological therapies exist for traction alopecia. Cessation of the chronic traction is mandatory.



INFLAMMATORY DISEASES

FIXED DRUG ERUPTION

Fixed drug eruption is not common in Sub-Saharan populations because of the scarcity of systemic drugs (“no drugs, no fixed drug eruption”). The same applies to erythema multiforme and Stevens-Johnson syndrome.

Clinical picture is characterized by single or multiple hyperpigmented macules, roundish or oval in shape, dark brown in colour, with blurred borders, sometimes surrounded by a pale halo. The surface is smooth and not infiltrated.



LICHEN

Lichen is of common observation in populations living in Sub-Saharan countries. Children are not infrequently affected.

Typical features in dark-skinned patients are:

- a. localized or widespread, severe and chronic itching;
- b. widespread and marked Köbner phenomenon;
- c. precocious appearance of brown-black plaques, nodules and verrucous lesions, mainly on knees, legs and dorsal surface of the feet (hypertrophic- verrucous lichen);
- d. annular lesions in inguinal folds and genitals;
- e. nail dystrophy, with irregular surface of the nail plate and hyperkeratosis of the bed;
- f. uncommon involvement of the oral cavity, although lips may be involved.

Lichen planus tropicus is of rather common observation in the Middle East, Arabian Peninsula and North-Eastern Africa, in particular Egypt. It is triggered by chronic exposure to sunlight: this is the reason for which it occurs typically on the face of adolescents and young adults.





PSORIASIS

Prevalence of psoriasis is lower in African, Asian and Latin American populations compared to Northern Europeans: however, underdiagnosis is not rare due to less visible erythema.

Psoriasis in Africans is less common in West African groups (e.g., Sudanese), but frequent in East and Southern African populations (Bantu, Hamitic, Nilotic). In Uganda, scalp involvement is more common than elbows and knees involvement. Psoriasis is rare in rural areas, but common in urban centers, particularly in HIV-positive patients, where it may present with atypical and sometimes severe clinical manifestations.

In Afro-Brazilians, psoriasis is rare, with atypical distribution and often intense pruritus. In South American natives, the incidence is very low. In Asians psoriasis has a lower frequency and milder clinical course compared to the so-called Caucasians.

Psoriasis on dark skin shows more severe pruritus and thicker plaques and exhibit greater pigmentary sequelae. Plaques appear grayish or copper-coloured; post-inflammatory hypopigmented macules are frequent. Scaling is whitish or grayish, often giving “ashy” appearance. Pruritus is often severe, being sometimes the dominant symptom. Koebner phenomenon is more striking: it may lead to linear hyperpigmented scars; scalp and trunk involvement are more frequent; face and flexures are occasionally affected.

Because erythema is often masked by baseline pigmentation, psoriasis in dark skin may initially appear as dry, thickened, hyperpigmented plaques. Consequently, early or mild cases may be mistaken for lichen simplex chronicus, chronic eczema, pityriasis rubra pilaris or cutaneous lupus erythematosus.



SARCOIDOSIS

This disease is of not rare observation among Afro-Americans.

A typical clinical manifestation is the slow appearance of mildly inflammatory, asymptomatic papules and plaques located on the eyelids. They are isolated or grouped, roundish or oval in shape, brown-violet in colour, with a smooth surface. These lesions are often diagnosed as xanthelasma.

Diagnosis of sarcoidosis may be very difficult when it appears as nodules located on the nose, cheeks and ears. A biopsy for histopathological examination, in order to distinguish sarcoidosis from tuberculosis and leprosy, is mandatory.

Both erythema nodosum and lupus pernio are difficult to observe on dark skin.

The possible involvement of eyes, salivary glands, lymph nodes, lungs, liver, spleen and bones is helpful for the final diagnosis.



SEBORRHEIC DERMATITIS

Incidence and prevalence of seborrheic dermatitis are similar in all ethnicities.

There is no evidence that individuals with dark skin have a greater number or size of sebaceous glands than the so-called Caucasians. Furthermore, no significant differences in sebaceous gland activity between Caucasian and pigmented skin have been recorded. It is possible that *Malassezia* sp. load is higher in African patients due to weather conditions, such as hot-humid climate.

Seborrheic dermatitis is a well-known marker of HIV infection or AIDS. Erythema is difficult to see (the darker the skin, the harder it is to see erythema), while scaling of the scalp and face is much more evident.



PARASITIC DISEASES

HEAD LICE

In collective dermatological imagination, head lice are considered as a rare infestation in African children. This is only partially true. In fact, except in Ethiopia, where head lice prevalence has been always high, ranging from 30 to 65%, in several other African countries it is low, from 0.7 to 8%. This lower prevalence among African children is due to the fact that the head lice “prefer” to live on straight hairs, usually typical of Caucasians, rather than on curly hairs, typical of Africans and Afro-Americans.

Predisposing factors of head lice in Sub-Saharan Africa are poor household hygiene, overcrowding, sharing of washing facilities, beds and combs, poor personal hygiene, malnutrition, long hair and hair plaiting. According to several epidemiological-clinical studies carried out in Africa, head lice are more common in females.

Head lice infestations in African children are very often chronic: this is the reason why squamous-crusts lesions are so common. Bacterial superinfections are also common.

The greatest concern of head lice infestation in African children is that these insects can be vectors of pathogenic microorganisms. In 2011, it was demonstrated the presence of *Bartonella quintana* in head lice of subjects living in different regions of Ethiopia. The presence of *B. quintana* DNA was detected in head lice collected in Senegal and the Democratic Republic of Congo. Other bacteria isolated from “African” head lice were *Yersinia pestis*, *Acinetobacter baumannii* and *Borrelia recurrentis*.

In conclusion, head lice are not so rare in African children as commonly thought. In addition, these insects can be vectors of pathogenic bacteria.

SCABIES

Scabies is the most common parasitic disease in the world: it affects over 200 millions individuals. It is traditionally associated with low socioeconomic status and overcrowding; however, its prevalence is increasing globally, including people living in Western affluent countries.

Scabies in dark-skinned individuals presents with the same lesions and locations as in the so-called Caucasian subjects, i.e., burrows, papules, vesicles and pustules on axillae, elbows, interdigital folds of hands, nipples, pubis, genitals and buttocks. However, late diagnosis and lack of proper treatment often lead to “chronic” scabies. It is characterized by widespread, mildly inflammatory, squamous-crusted-verrucous lesions. This “chronic” scabies is clinically similar to crusted (Norwegian) scabies, except for pruritus: it is severe in “chronic” scabies and mild or absent in crusted scabies.

In numerous poor countries, the problem of therapy of scabies is a true, dramatic problem: both permethrin and ivermectin are too expensive in several countries. The most used drug is sulphur: it is prepared at a concentration ranging from 3 to 20%, usually in petrolatum. Numerous clinical studies showed that sulphur is less effective in comparison with permethrin; in addition, it frequently causes an irritant contact dermatitis, with widespread erythema, itching and burning sensation.

In some countries of Central America the prevalence of scabies is 90-100%: in some villages, the entire population is infected. In these regions, a frequent cause of death in children is post-streptococcal renal failure, due to the almost total lack of dialysis centers. Post-streptococcal renal failure is often caused by chronic bacterial skin infections, which are in turn caused by chronic scratching.

In summary: in Central America, scabies is, albeit indirectly, an important cause of infant mortality! This series of events has been given the name of “scabies cascade.” Therefore: scabies -> chronic itching -> chronic scratching -> streptococcal superinfections of the skin -> acute glomerulonephritis -> chronic glomerulonephritis -> chronic renal failure -> death.



SKIN NEOPLASMS

BASAL CELL CARCINOMA

Basal Cell Carcinoma (BCC) is the most common skin cancer worldwide, but its incidence is significantly lower in individuals with darkly pigmented skin (Fitzpatrick IV–VI) due to the photoprotective effects of epidermal melanin, which absorbs and scatters UV radiation. In many African, Black, and Asian populations, BCC comprises only 15–30% of all skin cancers (compared with 70–80% in lighter-skin populations).

PIGMENTED BCC IN DARK SKIN

Pigmented BCC is disproportionately common in individuals with darker skin tones, representing 50–70% of all BCCs in these groups. Increased melanin deposition within the tumor explains the darker coloration and often mimics other pigmented lesions.

CLINICAL PRESENTATION IN DARK SKIN

In darkly pigmented skin, classic signs such as erythema and telangiectasia are often subtle or absent. Instead, clinicians should look for:

- Dark brown, blue-black, or violaceous plaque/nodule
- Pearly or shiny rolled border (may appear gray, purple, or jet black instead of pink)
- Central ulceration or depression is often present but may appear as a darker concavity rather than a red erosion
- Slow-growing lesion that persists or enlarges over months to years
- Surface smoothness or waxy quality, more noticeable on palpation than visual examination

Due to its pigmentation, BCC in dark skin is frequently misdiagnosed as:

- melanoma (especially superficial spreading or nodular).
- seborrheic keratosis.
- Dermatofibroma.
- pigmented Bowen disease.
- benign melanocytic nevus.

This leads to diagnostic delays, with dark-skinned patients often presenting at a later stage or with larger lesions.

SUBTYPE-SPECIFIC FEATURES IN DARK SKIN

Nodular BCC

- Typically presents as a pigmented, dome-shaped papule or nodule
- Often called the “pearly black nodule”.
- The classic rolled border may appear gray, purple, or dark brown.
- Ulceration produces a “punched out” darker central crater.
- Commonly located on the head and neck, but in darker skin more lesions occur on non-sun-exposed sites such as the back.

Superficial BCC

- Appears as a brown-red or hyperpigmented patch or thin plaque with mild scaling.
- Borders may be subtle and blend with surrounding skin.
- Often mistaken for eczema, psoriasis, tinea, or a post-inflammatory patch.
- More frequently occurs on the trunk in darker skin populations.

Morpheaform / Infiltrative BCC

- Rare in dark skin but when present may appear as a scar-like, ivory or hypopigmented plaque.
- Difficult to detect due to the lack of visible erythema.
- Often diagnosed late because it resembles a keloid, hypertrophic scar, or post-traumatic change.

DERMOSCOPIC FEATURES IN DARK SKIN

Dermoscopy remains helpful, though pigmentation may obscure vascular signs. Key findings include:

- Maple-leaf-like areas, blue-gray ovoid nests, spoke-wheel structures.
- Shiny white structures (polarized), indicating fibrosis.
- Absence of pigment network, helping differentiate from melanocytic lesions.
- Telangiectasia may be less visible due to background melanin.

DISTRIBUTION DIFFERENCES

Unlike in fair skin, where UV exposure is the major risk factor, BCC in darker skin occurs:

- more frequently on non-sun-exposed areas (trunk, genitalia, axillae).
- often associated with chronic scars, burns, or trauma.
- sometimes arising in areas of previous inflammation or irritation.

This pattern aligns more with an etiology linked to local injury or genetic predisposition rather than UV damage alone.

DIAGNOSTIC CONSIDERATIONS IN DARK SKIN

Because of reduced visibility of redness and surface vascularity, clinicians should maintain a high index of suspicion for any:

- persistent pigmented lesion with a pearly or shiny quality.
- non-healing ulcer or erosion.
- plaque that enlarges slowly over time.
- “seborrheic keratosis-like” lesion with unusual firmness or border change.

A low threshold for biopsy is recommended to avoid delayed diagnosis.



MELANOMA

Melanoma is far less common in dark skin but far more lethal when it occurs. Incidence rates: Sub-Saharan Africa: ~1–3 / 100,000 per year. African Americans: ~1/10 the incidence of Whites, but 5× higher mortality.

The disparity is due mainly to late diagnosis, non-sun-exposed sites, and low awareness among both patients and clinicians. UV exposure plays a minor role; instead, chronic trauma and mechanical stress are more relevant triggers.

Major Risk Factors are Acral trauma and chronic irritation (palms, soles, nail beds); Chronic scars or ulcers (post-burn, leprosy, trauma); Genetic predisposition (CDKN2A, BRAF, KIT mutations — distribution differs by ethnicity); Albinism: absent melanin → high susceptibility to UV-related melanoma and Immunosuppression (HIV, transplant).

Common presentations are Acral Lentiginous Melanoma (ALM) with Irregularly pigmented macule or plaque on palms/soles or under nail; In nail: longitudinal melanonychia that widens, irregular pigment, nail dystrophy (“Hutchinson sign”: pigment on periungual skin). Mucosal Melanoma with Pigmented plaque or mass on oral, genital, or anal mucosa and frequently ulcerated; metastasizes early. Amelanotic Melanoma: pink, red, or hypopigmented nodule—particularly treacherous in dark skin.

Melanoma in dark skin is less frequent but more aggressive due to delayed recognition. Acral Lentiginous Melanoma is the hallmark subtype and should be suspected in any irregular, enlarging, or ulcerated plantar or subungual lesion. Early biopsy is essential: *“Every chronic lesion on palms, soles, or nails deserves histology”*.



SQUAMOUS CELL CARCINOMA

Squamous Cell Carcinoma (SCC) is the most common skin cancer among individuals of African, Afro-Caribbean, and many South Asian populations, whereas basal cell carcinoma predominates in lighter-skinned groups. Although the overall incidence of SCC is lower in darker skin types due to photoprotective melanin, morbidity and mortality are significantly higher. This disparity is largely attributed to delayed diagnosis, atypical tumor appearance, frequent presentation on non-sun-exposed sites, and misinterpretation as benign or infectious conditions.

UNIQUE EPIDEMIOLOGY IN DARK SKIN

- UV radiation plays a smaller role because epidermal melanin reduces DNA damage.
- SCC commonly arises from non-UV-related pathways, especially chronic inflammation, scars, trauma, burns, chronic ulcers, and infections.
- Acral, mucosal, and subungual tumors are disproportionately common.
- Many patients present at advanced stages, sometimes with nodal or visceral metastasis, due to subtle early findings and limited awareness of cancer risk.

CLINICAL APPEARANCE IN DARK AND PIGMENTED SKIN

SCC in dark skin frequently diverges from the classic erythematous, scaly lesions seen in lighter skin:

- Lesions often appear gray-brown, violaceous, or hyperpigmented, with minimal or absent erythema.
- Tumors may be pigmented (dark brown to black), leading to confusion with benign melanocytic lesions or post-inflammatory hyperpigmentation.
- Crusting, hyperkeratosis, or verrucous texture are more prominent signatures than redness or inflammation.
- Borders are often indistinct, blending into background skin, particularly in chronic scars or ulcers.
- The lesion may present as a firm plaque or nodule, sometimes ulcerated or fungating, with variable pigmentation.
- Amelanotic tumors can appear dull gray or violaceous, mimicking tropical ulcers or chronic eczema.

TYPICAL SITES IN DARK SKIN

SCC in darker populations tends to arise in non–sun-exposed or previously damaged areas, including:

- Legs and feet—one of the most common sites, especially in women.
- Chronic ulcers (e.g., venous ulcers, sickle cell ulcers) → Marjolin’s ulcer.
- Scars from burns, trauma, or old infections (e.g., leprosy, osteomyelitis).
- Acral locations: soles, nail beds, palms.
- Mucosal sites: oral cavity, anogenital region.

This distribution reflects the strong association with chronic inflammation, wound healing disorders, and infection-related carcinogenesis rather than UV exposure.

COMMON DIAGNOSTIC PITFALLS IN DARK SKIN

Because erythema and inflammation are difficult to visualize, SCC may be mistaken for:

- Tropical ulcers or bacterial infections.
- Chronic eczema or lichen simplex.
- Tuberculosis verrucosa cutis.
- Chromoblastomycosis or deep mycoses.
- Post-traumatic scars.
- Verrucous lesions of HPV, including genital warts.
- Pigmented plaques assumed to be benign or residual PIH.

These misinterpretations contribute to delayed biopsy, sometimes allowing months or years of tumor progression.

PIGMENTARY FEATURES AND SEQUELAE

- SCC often shows peripheral hypermelanosis, making the lesion look deceptively benign.
- Post-inflammatory hyperpigmentation or hypopigmentation around the tumor may mask early growth.
- Surrounding dyschromia frequently hides ulceration or induration, key diagnostic clues.

PROGNOSTIC CONSIDERATIONS

In dark-skinned individuals, SCC is:

- More likely to present at advanced stage or with deep local invasion.
- Associated with higher rates of metastasis, especially when arising on scars, mucosa, or chronic ulcers.
- Often underrecognized by both patients and clinicians due to misleading color and non-classical morphology.

CLINICAL PEARLS FOR EARLY RECOGNITION IN PIGMENTED SKIN

- Focus less on color and more on texture, induration, hyperkeratosis, ulceration, fixation, and change over time.
- Any lesion on a chronic ulcer, burn scar, or non-healing wound warrants a biopsy.
- Pigmented plaques or nodules on acral or mucosal surfaces should be viewed with a high index of suspicion.
- Regular skin examinations are crucial, especially in patients with chronic leg ulcers, prior trauma, or persistent inflammatory dermatoses.



VIRAL DISEASES

HERPES ZOSTER

The first skin lesions of herpes zoster are clinically superimposable on Caucasian and dark skin. The final outcomes are different: frequent appearance of hypopigmented lesions and hypertrophic and/or atrophic, hypopigmented scars, because of the lack, in several poor countries, of antiviral drugs.

For the same reasons, herpes zoster ophthalmicus is frequently followed by chronic kerato-conjunctivitis, iridocyclitis, chorioretinitis, secondary glaucoma and unilateral blindness.

Also herpes zoster of the geniculate ganglion is often followed by sequelae; they are: chronic otalgia, headache, tinnitus, dizziness, deafness and facial palsy.



MOLLUSCUM CONTAGIOSUM

In many children living in Sub-Saharan Africa, molluscum contagiosum is a dramatic marker of HIV infection and AIDS.

On the face, in particular on the eyelids, dozens of roundish papules and nodules, isolated or confluent, with the same colour of the skin or pale pink, are present. The scalp and ears may also be infected. These lesions are asymptomatic.

Local treatment is very often impossible.



PITYRIASIS ROSEA

This infection is common in Sub-Saharan Africa.

Clinical picture is similar in all ethnicities. However, in African patients, not rare is the involvement of the face, in particular the forehead, and retroauricular folds. Not rare is also an enanthema of the oral cavity. Lesions of pityriasis rosea often heal with significant scaling; the latter sometimes causes severe itching.



OTHER DISEASES

CHLOASMA AND MELASMA

Both these conditions are very common in females with dark skin. They are the final clinical result of:

- a. previous or concomitant, long-lasting, inflammatory skin diseases;
 - b. irritant/allergic contact dermatitis caused by topical products and drugs;
 - c. chronic therapies with oestrogens and progestationals and d) chronic exposure to ultraviolet rays.
- Family predisposition is also considered as a possible risk factor.

The first lesions of chloasma and melasma appear at a young age. They are characterized by more or less numerous brown macules, of different morphology and size, with blurred borders, located mainly, bilaterally and symmetrically, on the cheeks. Also the forehead and the upper lip are often involved.





DERMATOSIS PAPULOSA NIGRA

Dermatosis papulosa nigra is very frequent in subjects with high phototypes.

It presents as more or less numerous brown-black, sessile and pedunculated papules occurring on cheekbones and cheeks from adolescence.

It is considered as a clinical variety of seborrheic keratosis.

Therapy (with liquid nitrogen, electrodesiccation and lasers) must be carried out with caution: the development of hyper- or hypopigmented lesions and hypertrophic or atrophic scars is common.



PITYRIASIS ALBA

Pityriasis alba is very common in children with dark skin. It is often characterized by multiple, hypochromic lesions, of different morphology and size, with a finely scaly surface and well-defined borders. No symptom is usually reported.

In addition to the typical bilateral location on the cheeks, the forehead, temples and nose may be involved. In not rare cases, pityriasis alba occurs also on shoulders, upper portion of the chest and back.

Differential diagnosis may be sometimes difficult: it includes post-inflammatory hypopigmentations (importance of medical history), vitiligo (bilateral and symmetrical hypochromic lesions, with smooth surface, located on the eyelids, around the mouth, elbows, hands, genitals, knees and feet) and leprosy (possible presence of systemic symptoms and signs).



SCARS AND KELOIDS

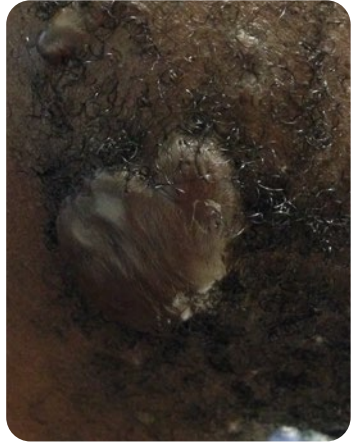
Scars and keloids are extremely common in individuals with pigmented skin: both genders are equally involved. An ethnic/genetic predisposition has been hypothesized.

Although ice picks, boxcars and rolling scars may occur on the face, in particular the cheeks, in patients with previous or concomitant acne, hypertrophic scars and keloids are more common. There is no location preference. The development of hypertrophic scars and keloids would be caused by the presence of more numerous, larger and often binucleated or multinucleated fibroblasts in the dermis and a reduced activity of collagenases.

The development of freakish hypertrophic scars and keloids is also due to other reasons:

- a. they may be the final clinical result of previous burns (the latter are much more common than people think);
- b. the lack of prompt and appropriate therapies (in Africa, the number of burn centres is extremely low);
- c. the frequent skin bacterial superinfections.





SEBORRHEIC KERATOSES

These lesions are common in subjects with pigmented skin. Ethnic and family predisposition, and chronic sunlight exposition are considered as risk factors.

Skin lesions are superimposable to those of the so-called Caucasian patients: more or less numerous, isolated or grouped, pigmented papules, roundish or oval in shape, with a smooth or hyperkeratotic surface. These lesions are especially located on the face, neck and trunk. Symptoms are rare and mild; however, irritation and bacterial superinfections are possible.

Therapy of seborrheic keratoses on dark skin is difficult. Electrodesiccation and curettage, cryotherapy and lasers frequently induce hyper- hypopigmented / atrophic or hypertrophic scars. In “patient” patients, topical 30% salicylic acid or 50% urea may be taken into consideration for a long period of time.

Dermatosis papulosa nigra is considered a clinical variety of seborrheic keratosis located on the face.



VITILIGO

Vitiligo is frequent in African populations. It is not characterized by different clinical features from those of subjects with low phototypes. However, vitiligo often appears at a young age. Furthermore, the first location of the disease may be the lips, and this is considered a prognostically negative factor. Acral or widespread involvement is common.

A post-traumatic clinical variety of vitiligo (for instance, footwear on the back of the feet) has been hypothesized.





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