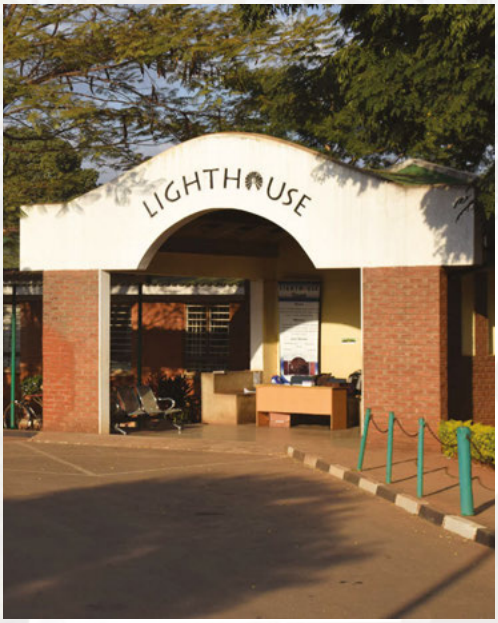

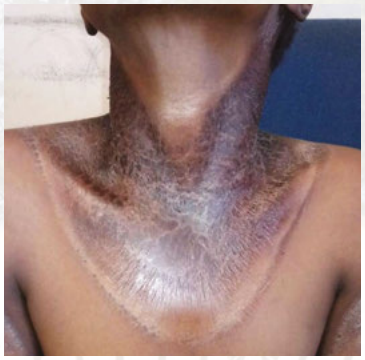
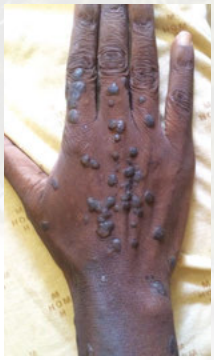
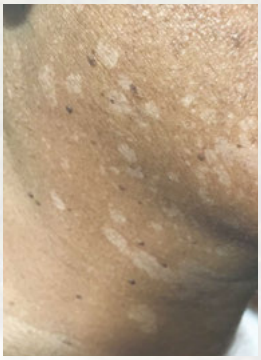
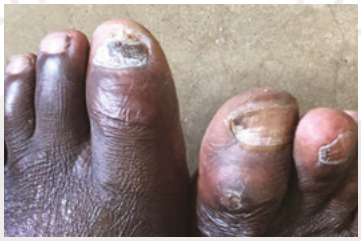
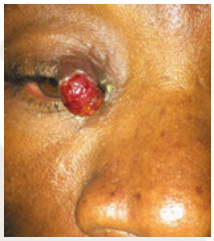
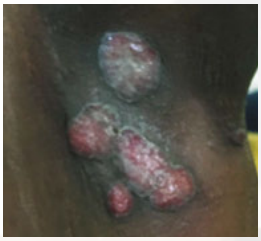

# LIGHTHOUSE

## SKIN MANUAL FOR HIV CLINICIANS

Claudia Wallrauch

Tom Heller

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# Preface

*…una lonza leggera e presta molto,*
*che di pel macolato era coverta;*
*e non mi si partia dinanzi al volto…*

…A panther light and swift exceedingly,
Which with a spotted skin was covered o'er!
And never moved she from before my face…

—Dante Aligheri, *The Divine Comedy*: Inferno, Canto I

With progress made towards and beyond the 90%–90%–90% goals in HIV care, a fourth 90% goal becomes more relevant: that at least 90% of those with viral suppression enjoy quality of life with respect to their health. This requires us to improve care and treatment of those conditions that may be considered less severe and less deadly.

As the spotted skin of a leopard shows—and can a leopard change its spots? (Jeremiah 13:23)—skin changes are often misinterpreted. In medieval Europe, the leopard's spots made him the representation of physical lust—possibly also because of the spots associated with secondary syphilis (see page 24). The high visibility of skin diseases increases the likelihood of stigmatisation, as skin changes are falsely associated with dirtiness, poor personal hygiene, and other 'sins of the flesh'.

Skin diseases are thus more than a cosmetic nuisance; they can produce anxiety, depression, and psychological problems that affect patients' lives. Patients with changes in important body image areas, such as the face, hair, hands, and genitals, are especially prone to distress. Thus, diagnosis and treatment are highly relevant to the patient.

In Africa, HIV has changed the landscape of skin diseases. The most common conditions stemming from HIV in adults are Kaposi sarcoma, papular eruptions, herpes zoster, fungal infections, and molluscum contagiosum; in children, tinea capitis, molluscum contagiosum, and warts are frequently seen. With widespread use of antiretroviral therapies, and drugs like cotrimoxazole and antituberculosis medications, we are seeing increased frequency of drug reactions and immune reconstitution inflammatory syndrome (IRIS) due to the high background prevalence of infection.

With this manual, we want to help our colleagues to recognise and treat some of the most frequently encountered skin conditions. Complicated cases will still need to be referred to dermatology departments and specialists, but for many diseases a diagnosis can be made, and treatment attempted, at the first point of care. We hope this manual proves helpful for that.

Claudia Wallrauch Tom Heller 2024

# Acknowledgements

We would like to acknowledge all the Lighthouse clinicians who help us daily in finding skin changes in our patients. In particular, we'd like to thank Prosperina Kapawata from Umodzi Family Clinic, Blantyre, for reviewing the treatment sections with a local dermatological eye.

Several of the images in this manual are taken from the manual *Common Skin Conditions in Africa*, by Drs Colette van Hees and Ben Naafs; we thank them for general permission to reproduce images from their manual without financial compensation for projects like ours. We highly recommend also reading their manual for more in-depth dermatological insights (https://plan-g.at/images/pdf/Common_skin_diseases_in_Africa_ver2017.pdf).

As with other Lighthouse manuals, we would like to acknowledge the excellent work and smooth cooperation of Kevin O'Conner, from Seattle. Without his efforts and input, this manual would have stayed a boring Word document.

Finally we'd like to thank our patients who consented to our taking pictures of their skin conditions. We hope we have been able to help them and others.

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**INTRODUCTION**
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Other images adapted from https://basicmedicalkey.com/alterations-in-the-integumentary-system/. Accessed 2023 Nov 7.

**PAPULAR PRURITIC ERUPTION/PAPULAR URTICARIA/EONOSOPHILIC DERMATITIS**
Pg. 3, bottom: image #32 from Hees C, Naafs B. Common Skin Diseases in Africa: An illustrated guide. 3rd revised edition. Stichting Troderma; 2014. ISBN 978-90-808016-0-8. [Hereafter referred to as *CSDIA*.]

**CUTANEOUS LARVA MIGRANS**
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**CUTANEOUS TUBERCULOSIS**
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*Mpox in HIV-positive patients* photos from Mitjà O, Alemany A, Marks M, Mora JIL, Rodríguez-Aldama JC, Silva MST, et al. Mpox in people with advanced HIV infection: a global case series. Lancet. 2023 Mar 18;401(10380):939-949. doi: 10.1016/S0140-6736(23)00273-8. Epub 2023 Feb 21.

**OTHER BLISTERING DISEASES**
Images from Tamazian S and Simpson CL. Autoimmune bullous disease in skin of color: A case series. JAAD Case Rep. 2020 Nov;6(11):11731178. doi: 10.1016/j.jdcr.2020.08.035. Epub 2020 Sep 9. [Hereafter referred to as *Tamazian and Simpson*.]

**PSEUDOFOLLICULITIS BARBAE**
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**SEVERE ACNE**
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**VITILIGO**
Patient before and after photos on [second page] from Onyekonwu CL, Chukuwuka CJ, Nwandu A, Patel D. JAAD Case Rep. 2018 Jan 16;4(2):145-148. doi: 10.1016/j.jdcr.2017.09.030. eCollection 2018 Mar. Images used under a Creative Commons license, CC BY-NC-ND (https://creativecommons.org/licenses/by-nc-nd/4.0/).

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**NECROTISING ULCERATIVE GINGIVITIS**
Images from Kman NE and Kumar VP. Acute necrotizing ulcerative gingivitis (ANUG). JETem. 2017;2(2):V1–2. doi: 10.5070/M522034558. Retrieved from https://escholarship.org/uc/item/21r2t29v. Used under a Creative Commons Attribution license, CC BY 4.0 (https://creativecommons.org/licenses/by/4.0/).

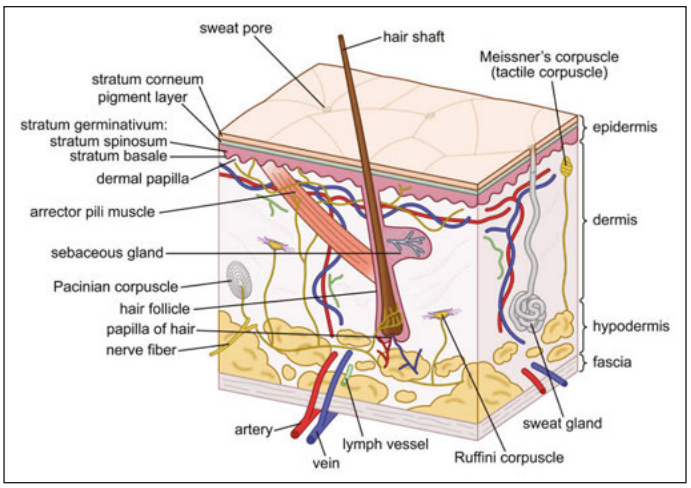
# Abbreviations

| | |
|---|---|
| ABC | abacavir |
| ART | antiretroviral treatment or therapy |
| CrAg | cryptococcal antigen |
| DPN | dermatosis papulosa nigra |
| EBV | Epstein–Barr virus |
| EDV | epidermodysplasia verruciformis |
| EVF | efavirenz |
| HPV | human papillomavirus |
| HS | hidradenitis suppurativa |
| HSV | herpes simplex virus |
| IRIS | immune reconstitution inflammatory syndrome |
| KOH | potassium hydroxide |
| KS | Kaposi sarcoma |
| KSHV | Kaposi sarcoma herpesvirus |
| MCV | molluscum contagiosum virus |
| MTB | *Mycobacterium tuberculosis* |
| NSAID | non-steroid anti-inflammatory drug |
| NUG | necrotising ulcerative gingivitis |
| NVP | nevirapine |
| PCT | porphyria cutanea tarda |
| PI | postinflammatory |
| PPE | papular pruritic eruption |
| SJS | Stevens–Johnson Syndrome |
| STI | sexually transmitted infection |
| TEN | toxic epidermal necrolysis |

# Introduction

## ANATOMY

The skin is made up of three layers: the epidermis, dermis, and the hypodermis. The layers vary significantly in their anatomy and function.




### EPIDERMIS

The epidermis is the thin outer layer of the skin. It consists of three types of cells:

**Squamous cells** form the outermost layer of the skin (the stratum corneum), which is continuously shed.

**Basal cells** are found just under the squamous cells at the base of the epidermis.

**Melanocytes,** which produce melanin, are also located at the base of the epidermis. Melanin gives the skin its colour.

### DERMIS

The dermis is the middle layer of the skin and contains:

**Blood vessels** for nutrition of the skin.
**Lymph vessels** for immune function.
**Sweat glands** to cool the skin (temperature control).
**Hair follicles** to preserve heat.
**Sebaceous glands** for production of oily substances.
**Nerves and sensory corpuscles** for sensing pain and touch.

The dermis is held together by **collagen**, a protein formed by fibroblasts. This layer gives the skin flexibility and strength.

### SUBCUTANEOUS HYPODERMIS

The subcutaneous hypodermis is the innermost layer of the skin; it consists of collagen and fat cells. Subcutaneous fat helps to conserve the body's heat by providing insulation, and protects the body from injury by acting as a shock absorber.

## BASIC FUNCTIONS OF THE SKIN

### BARRIER FUNCTION

The skin provides a physical barrier that regulates water loss and protects against mechanical, chemical, and microbiological insults.

### IMMUNOLOGICAL FUNCTION

The skin senses and responds to pathogens.

### TEMPERATURE REGULATION

The skin helps to maintain a constant body temperature through the insulating properties of fat and hair, and by dissipating heat through sweat production and microvasculature.

### NERVE SENSATION

Sensory receptors allow the skin to constantly monitor the environment. They are also important for interaction with physical objects.

### INJURY REPAIR

Skin wounds heal in four phases: coagulation, inflammation, proliferation (tissue formation), and remodelling (scarring).

### PROTECTION AGAINST RADIATION

The dark pigment melanin protects cells against the UV radiation in sunlight, which can cause cancer.

### APPEARANCE AND SOCIAL FUNCTION

Skin defects and even physiological ageing can cause considerable stress, as the skin is also an indicator of health and status to our fellow human beings.

## THE DERMATOLOGICAL EXAM

### REQUIREMENTS

**Adequate light**. Sunlight is the best; otherwise, try to get as much light as possible.

**Privacy**. Because the patient needs to undress—you cannot diagnose what you cannot see! (For example, patients often report 'ulcers in the private parts'—which upon examination turn out to be HPV-associated warts, not ulcers.)

**A prepared mind (knowledge)**. Because we can only see what we know to look for.

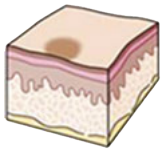
# Introduction

## MORPHOLOGY OF LESIONS

### PRIMARY LESIONS

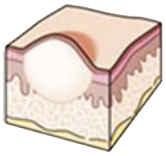
**Macule**

A **macule** is a flat (non-palpable) lesion. By definition, maculae are smaller than 1 cm; larger ones would more properly be called **patches**.

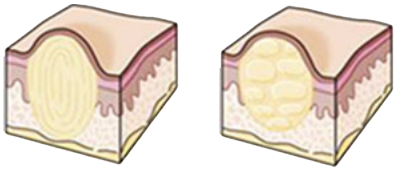


**Papule, nodule, and tumour**

A **papule** is a raised, palpable lesion caused by proliferation in the epidermis or superficial dermis. By definition, papules are smaller than 0.5 cm. They can be classified as itchy or non-itchy, and are seen in a variety of HIV-associated skin conditions.

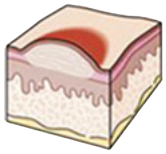


Nodules and tumours more often originate from the mid-deep dermis. A **nodule** (left) is 0.5–2 cm in size; a **tumour** (right) is larger than 2 cm.



**Plaque**

Plaque can be felt on palpation, like a papule. By definition, however, plaque is larger than 1 cm, often covering an even larger surface area, with little elevation. Plaque is also caused by proliferation in the epidermis or superficial dermis.

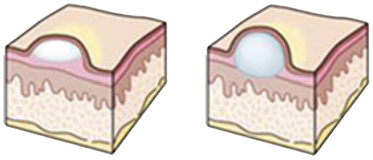


**Weal (wheal)**

A type of plaque resulting from transient oedema in the dermis.

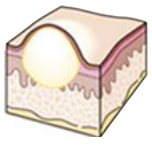
**Vesicle or bulla**

A circumscribed, elevated lesion filled with fluid. A **vesicle** (left) is smaller than 1 cm; a **bulla** (right) is larger than 1 cm.



**Pustule or abscess**

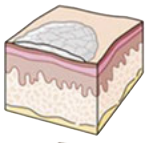
A circumscribed, elevated lesion filled with pus. A **pustule** is smaller than 1 cm; an **abscess** is larger than 1 cm.



### SECONDARY LESIONS

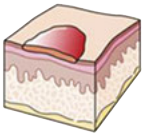
**Scales**

Flakes of cornified skin cells.



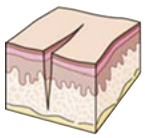
**Crust**

Dried exudate (serum or blood) on the skin.



**Fissures**

Cracks in the skin.

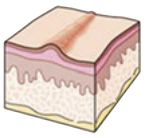


**Ulcer**

An area of destruction of the epidermis.

**Scar**

Excess collagen after an injury has healed.



## PAPULAR PRURITIC ERUPTION
## PAPULAR URTICARIA
## EOSINOPHILIC FOLLICULITIS

### CAUSE

The cause of **papular pruritic eruption (PPE)** and **papular urticaria** is not completely understood. These conditions are most likely the result of hypersensitivity to insect bites and chronic reaction to insect antigens due to HIV-associated immune dysregulation. **Eosinophilic folliculitis** is a non-infectious eosinophilic infiltration, mainly of the hair follicles.

### HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?

With all three conditions, the lesions consist of papules and small nodules, all of which are extremely itchy and cause (post-) inflammatory hyperpigmentation. Initially, they may appear as pustules or vesicles; because they are very itchy, we may also see scratch marks and ulcerations.

Where they differ most (though there is some overlap) is in the distribution of lesions:

**PPE** is likely one of the most common skin conditions among HIV patients; numerous studies indicate that it is present in as many as three-quarters of patients with CD4 counts below 200. It mainly occurs on the upper and lower limbs.

**Papular urticaria** mainly occurs on the face, arms, and legs—i.e., exposed areas that are most susceptible to insect bites. If caused by fleas or bedbugs, there may be a linear distribution of lesions that is not seen with the other conditions.

**Eosinophilic folliculitis** occurs where hair follicles are present, mainly in the face, neck, scalp, upper chest, and trunk.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

It is difficult to differentiate among the conditions—histology may help, but it is not very relevant, as the treatment is similar for all three. Scabies is the main differential diagnosis to be identified, as it requires different treatment.

### WHAT IS THE TREATMENT?

**Antihistamines**

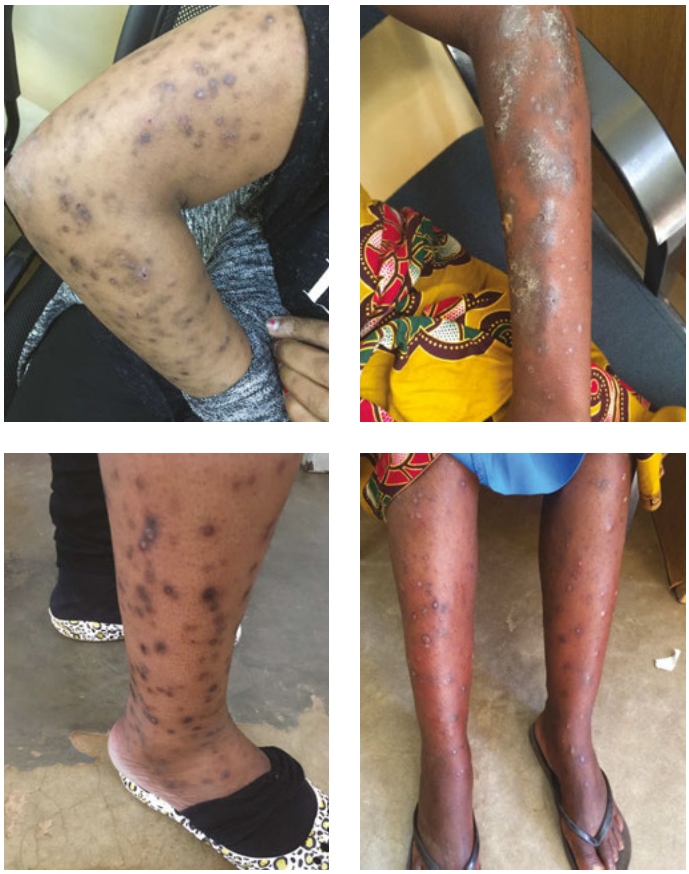
- Chlorpheniramine 4 mg tds
- Phenergan 25 mg bd

**Topical steroids**

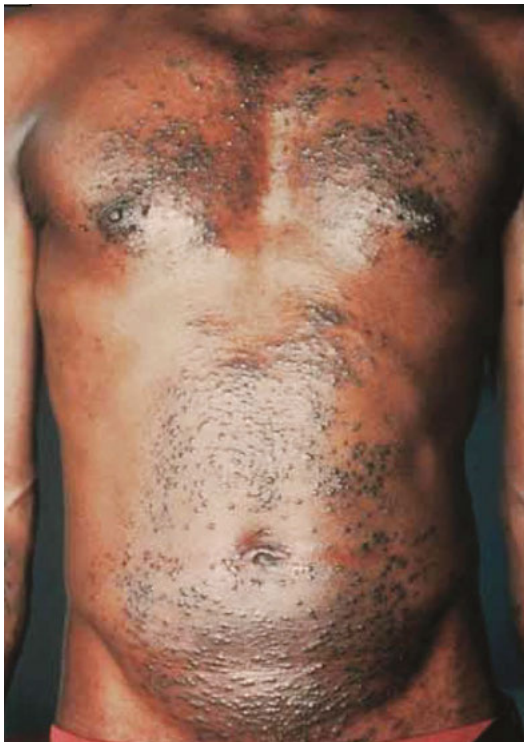
- Hydrocortisone cream 1% (for the face)
- Betamethasone valerate 0.025–0.1% (trunk, extremities)

### PEARLS AND HIV-RELATED FACTS

**Papular urticaria as a reaction to insect or flea bites is also seen among HIV-negative patients; it is more common in children than in adults. It is also referred to as a persistent insect bite reaction.**



Examples of papular pruritic eruption (PPE).



Folliculitis in an HIV-positive male.

## SCABIES

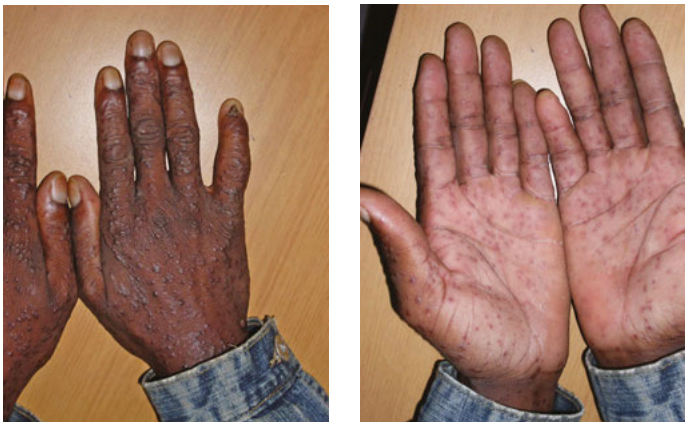
### CAUSE

Mites (*Sarcoptes scabiei*). Normally, 15–30 mites live on one human. Overcrowding, poor hygiene, delayed diagnosis, and delayed treatment contribute to their spread, especially among families or in institutions (e.g., barracks, prisons). Mites can live for three days separated from human skin; therefore, infected clothing and bedding can be a source of infection in addition to direct contact.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

During the first month, the patient remains asymptomatic. Immune reactions then develop and skin lesions form. Typically, papules occur on the hands (between the fingers, on the wrists), feet, ankles, elbows, axilla, groin, and genitals.

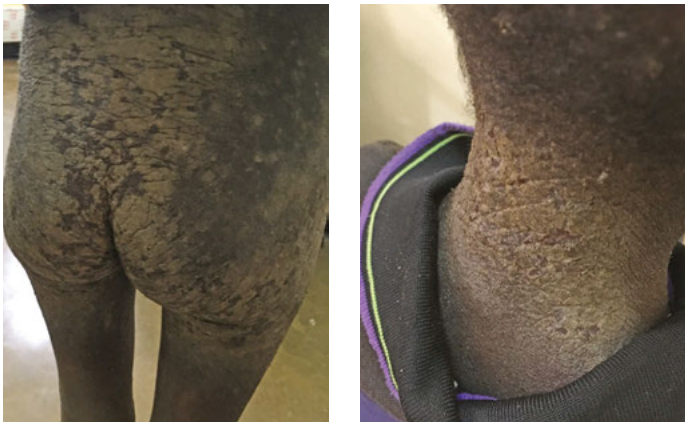
The head, face, neck, palms and soles are often involved in infants and very young children, but not in older children and adults.



Examples of scabies.

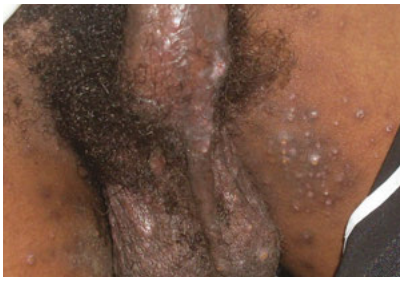
Burrows are the telltale sign of scabies; they represent tunnels in the skin caused by movement of the mites. They show as linear, thread-like papulous elevations 3–10 mm long that may be curved. The lesions are very itchy, especially at night when it is warm.

With advanced HIV disease, thickened, crusted lesions occur; this is called **crusted scabies** (formerly known as Norwegian scabies). The lesions can become more widespread over the body (e.g., including the buttocks or the face). Crusted scabies can affect the whole body (a form of erythroderma). These lesions are less itchy, since there is less of an immune response to the mites. However, they are highly infectious, as by this point there are thousands of mites present.



Crusted scabies.

Lesions can be complicated by bacterial superinfection, mainly staphylococci and streptococci. This is referred to as **impetiginisation**.



Scabies affecting the genital area.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Other itchy papules, like PPE, eosinophilic folliculitis, or papular urticaria—the distribution of the lesions is different. KOH examination of skin scrapings can be done to find mites, or parts of the mites, but this is rarely available in our setting.

### WHAT IS THE TREATMENT?

- Benzoyl benzoate 25% lotion. Repeat after 1 week—for example, on days 1–3 and 8–10.
- Permethrin 5% cream
- Ivermectin tablets 200 µg/kg with food (give only to patients weighing more than 15 kg)

  Treatment should be repeated after 1–2 weeks to prevent reoccurrence.

  With crusted scabies, ivermectin needs to be combined with a topical agent. Multiple doses of ivermectin may be needed here, depending on the severity of the disease.

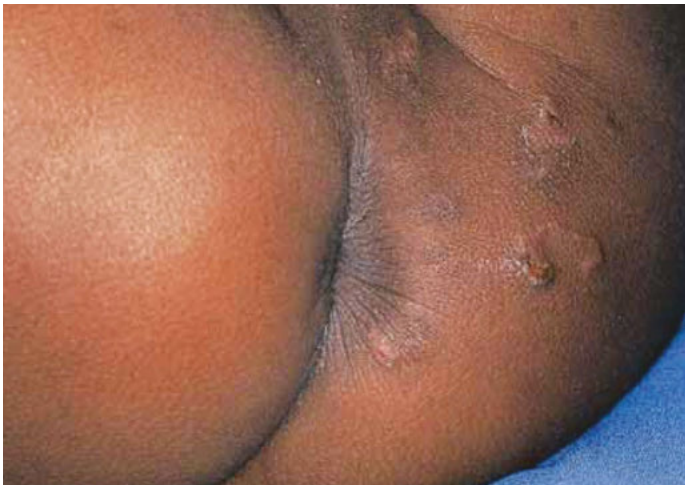
- Antihistamines (for itching)
- Antibiotics (for local superinfection)

**General measures:**

- Wash contaminated bed linen and blankets with hot water.
- All sexual contacts and other persons with prolonged skin contact need to be treated concomitantly.

### PEARLS AND HIV-RELATED FACTS

- **Patients may continue to experience pruritus for up to two weeks after successful treatment.**
- **Always ask about or check for infected family members who should be treated at the same time.**



## CUTANEOUS LARVA MIGRANS

**CAUSE**
Larvae of animal hookworms, which penetrate the skin. The larvae are shed by animals (e.g., dogs) on soil or sand and accidentally infect humans when they are sitting or lying on the ground.

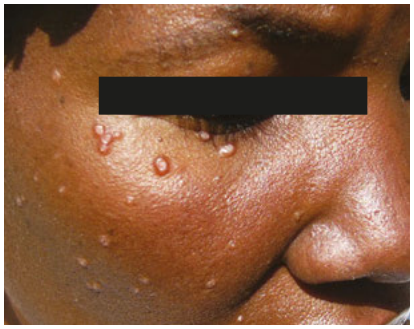
**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Hookworm larvae enter through intact skin and stay confined to the upper layer. Migration of the larvae through the skin causes very itchy linear or serpiginous papulous tracks (also known as **creeping eruption**). The tingling starts as soon as an hour after penetration of the larvae. Lesions are typically located on the feet (including the backside of the feet), but can also occur on the buttocks, hands, and knees.

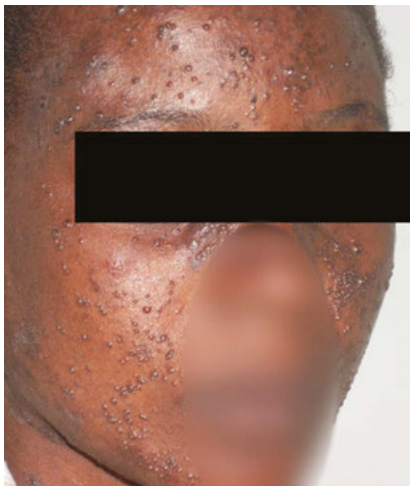
**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Scabies. Other itchy skin diseases.

**WHAT IS THE TREATMENT?**
- Ivermectin single dose (200 µg/kg, patients weighing more than 15 kg). In some cases, a second dose after 8 days may be needed.
- Albendazole 400 mg/day for 3 days
- Antihistamines (for itching)
- Cryotherapy is obsolete. Not only does it not kill the larvae, it can also cause severe scarring.

**PEARLS AND HIV-RELATED FACTS**
- **Creeping eruptions are seen more often in children than in adults, most likely because they are more likely to play barefoot.**
- **A patient's HIV status does not appear to affect the frequency or symptoms of larva migrans.**

## MOLLUSCA CONTAGIOSUM





### CAUSE

Molluscum contagiosum virus (MCV), a member of the family Poxviridae

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Typically, umbilicated papules (papules with a central dimple) are seen in mollusca contagiosum. The lesions (which are not itchy) are similar in colour to the surrounding skin, and are 1–3 mm in size. Fewer lesions are seen among HIV-negative children; immunosuppressed patients (including adults) tend to have larger lesions (up to 10 mm), and more of them.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Cryptococcoma of the skin are similar, but tend to be larger and may involve central necrosis. In case of doubt, perform a cryptococcal antigen (CrAg) test in serum—and consider a skin biopsy.

### WHAT IS THE TREATMENT?

- Lesions may be expressed using a sharp curette.
- Often the best treatment is ART; as the CD4 count improves, the lesions may flare up (IRIS), but will eventually disappear.

### PEARLS AND HIV-RELATED FACTS

**In immune-competent patients, the lesions heal spontaneously in 6–9 months.**

# Papules & Nodules Non-itchy Conditions

## WARTS

**CAUSE**
Human papillomavirus (HPV)

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Common warts (also called **verruca vulgaris**) present as cauliflower-like papules with a hard, rough surface, ranging in size from 1 mm to 1 cm or larger. They may be solitary or multiple, and are usually skin-coloured or hyperpigmented. They can become very large, especially in the genital or anal regions (where they are called **condylomata acuminata**). They may also affect the mucosa of the mouth. When transmitted by scratching, they can have a linear arrangement.

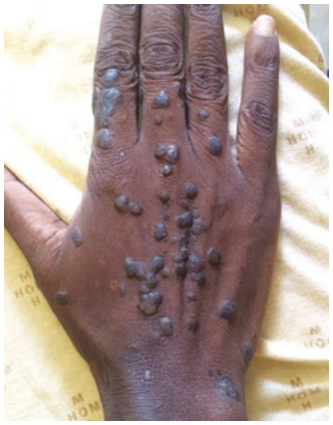
**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Other non-itching papulous diseases.

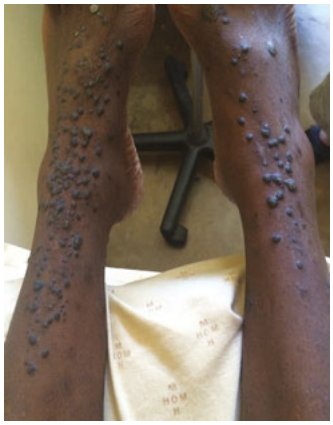
**WHAT IS THE TREATMENT?**

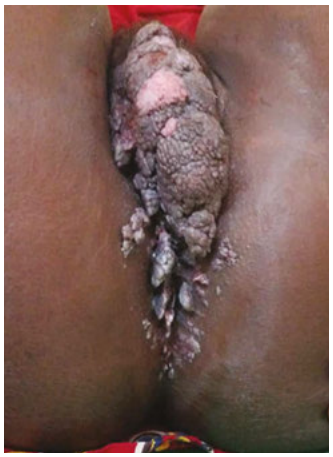
- Topical podophyllin 20% 1x or 2x per week for 4 hours each. The solution is applied with a cotton swab, and washed off with water afterwards. Treatment can be repeated as necessary, up to 4 or 5 times. Normal skin should be protected by applying Vaseline between treatments.
- In dermatology, the warts may be frozen using liquid nitrogen.
- Due to the risk of malignancy, larger (anogenital) lesions may require surgical intervention.
- Initiation of active ART will help to reduce the lesions.

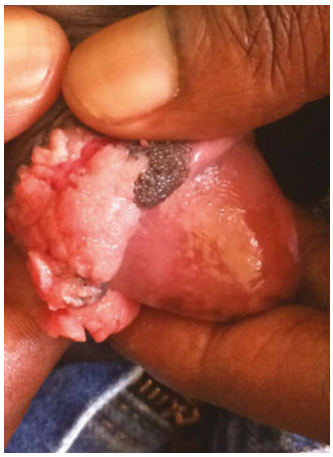
**PEARLS AND HIV-RELATED FACTS**

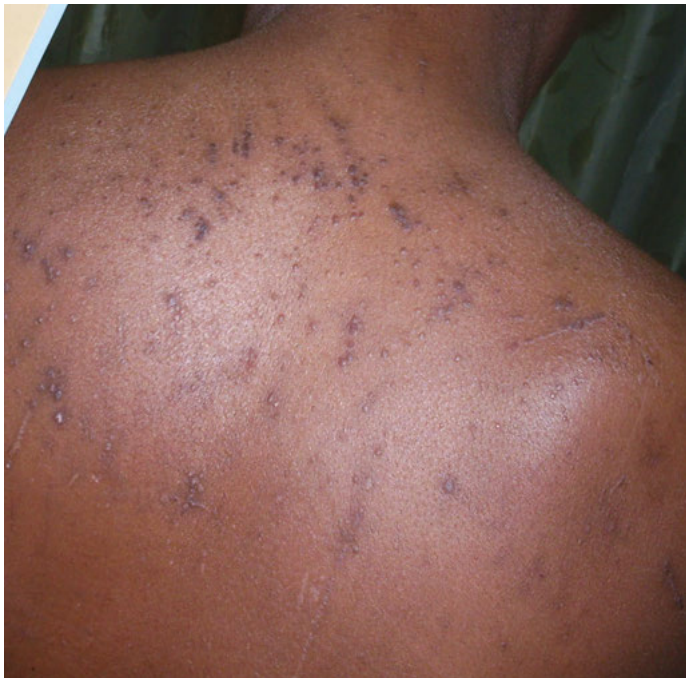
- **HPV is spread by sexual contact, other direct skin-to-skin contact, and self-inoculation, thus carrying the virus from an existing wart to healthy skin areas.**
- **In immune-competent patients, warts are particularly common among school-aged children; however, they may occur at any age.**
- **A special form of HPV skin infection seen in HIV patients is epidermodysplasia verruciformis (EDV). EDV is characterised by thin, flat-topped, hypopigmented papules coalescent to lines or plaques. The papules present on the chest, abdomen, and back, but can also appear on the arms and legs. Fine scaling can be noted, especially when the lesions are rubbed—this makes it possible to mistake EDV for tinea versicolor. Skin biopsy can aid in the diagnosis.**











Epidermodysplasia verruciformis (EDV).

## SYPHILIS

**CAUSE**
*Treponema pallidum*, a spirochaetal gram-negative bacteria

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Syphilis is a sexually transmitted infection that has three clinical stages.

Stage 1 is characterised by a painless ulcer in the genital region, which may be accompanied by mild local lymphadenitis.

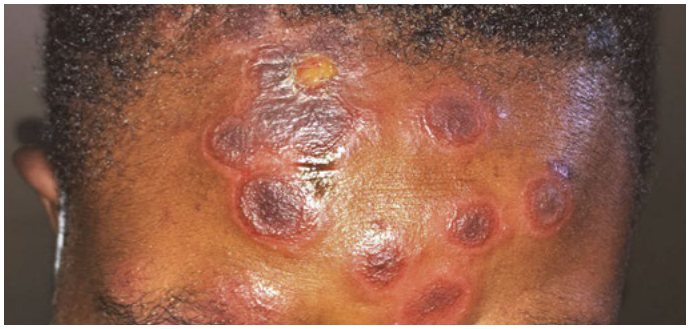
Stage 2 shows more generalised involvement of the skin (and of the inner organs, causing fever, headache, sore throat, malaise, or generalised lymphadenopathy). Papular lesions occur, which can be scaly on the periphery. When lesions involve warm, moist intertriginous areas, they do not show scales, but rather a moist appearance (called **condyloma lata**), and are very infectious. Syphilis lesions typically occur on the palms and soles, but can also involve the face, trunk, and extremities. Lesions close to the angles of the mouth and on the oral mucosa are also seen. The lesions are usually described as only mildly itchy.

Stage 3 affects mainly the inner organs, blood vessels, and the brain. There are no specific skin changes.

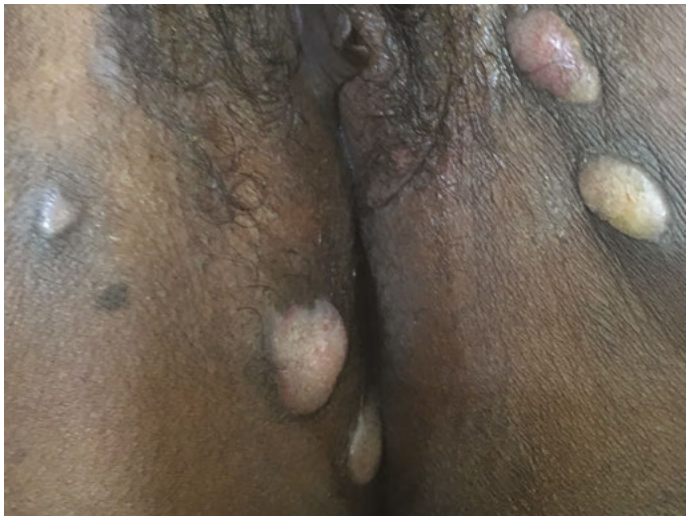
A syphilis rapid test should be done with unclear skin changes. A positive result confirms that the patient was infected at some stage, which makes the diagnosis more likely. Remember, however, that the syphilis rapid test is NOT really a VDRL test (although it is often considered as such)—it will continue to return a positive result after successful treatment.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Other maculopapular skin diseases.

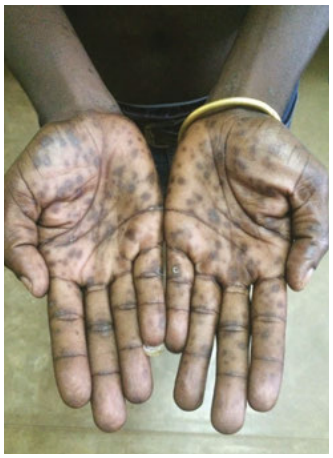
**WHAT IS THE TREATMENT?**
Benzathine penicillin im 2.4 MU weekly for 3 weeks.

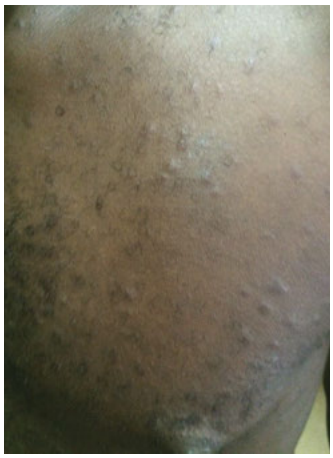


Lesions on the forehead.



Condyloma lata.





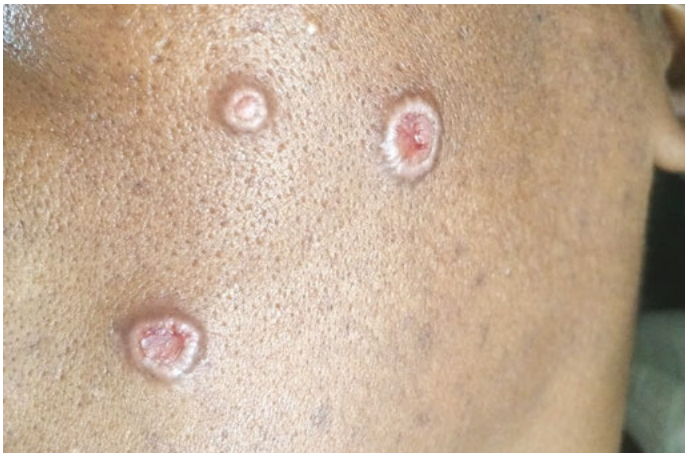
**PEARLS AND HIV-RELATED FACTS**

- **Because its lesions can also have pustular, nodular, lichenoid, follicular, psoriasiform, and annular appearances, syphilis is also called 'the great imitator'.**
- **A patient's HIV status does not affect the appearance of skin lesions in secondary syphilis.**
- **If any neurological symptoms are present, a lumbar puncture should be performed to evaluate (and/or treat) the patient for neurosyphilis.**
- **Sexual partners should be tested and treated according to the results. Persons who had sexual contact within the last 3 months with someone who was diagnosed with primary or secondary syphilis should be treated presumptively.**
- **During treatment, the patient may experience a reaction, with fever and a morbilliform rash as well as a brief worsening of skin lesions (Jarisch–Herxheimer reaction).**

## CRYPTOCOCCOSIS

**CAUSE**
*Cryptococcus neoformans* (fungus)

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Disseminated cryptococcosis can involve many other organs in addition to the skin. Infection of the brain is especially dangerous.



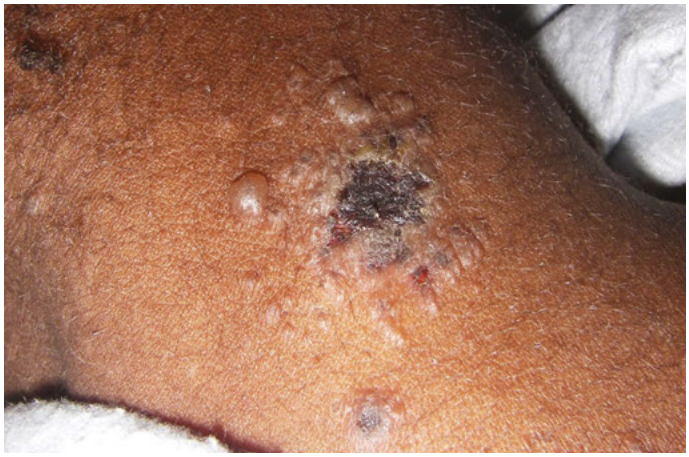
The lesions typical of cryptococcosis are skin-coloured papules or nodules, which are umbilicated (they have a central dent or dimple) and not itchy. They may become ulcerated with a black necrotic centre; frequently, you will see a larger lesion surrounded by smaller satellite lesions. Sometimes the ulcer can become large and walled; diagnosis in these atypical cases may depend on histology. The CrAg test in serum is usually positive; a lumbar puncture should be done to rule out cryptococcal meningitis.

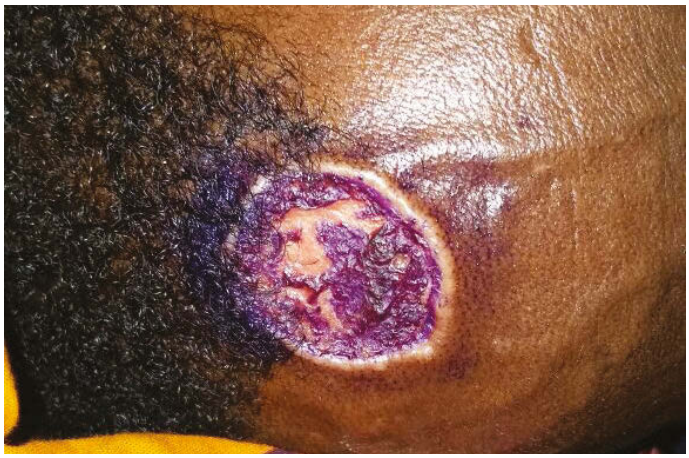
**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Mollusca contagiosum has a similar dimpled appearance.

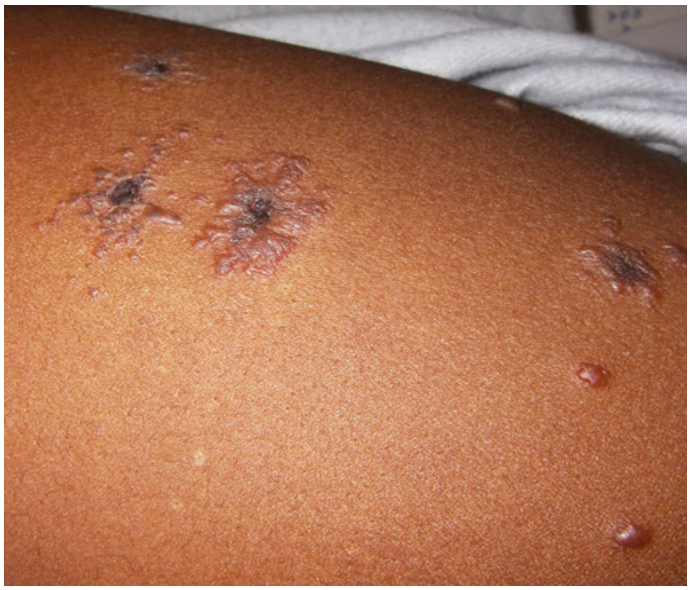
**WHAT IS THE TREATMENT?**
Follow the national guidelines for treatment of cryptococcemia and meningitis.

**PEARLS AND HIV-RELATED FACTS**

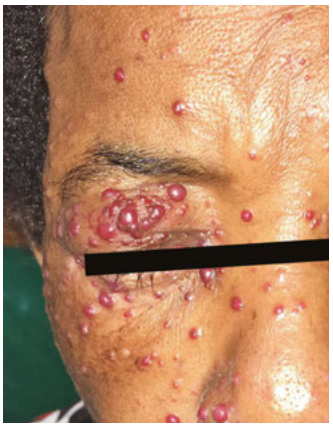
**Disseminated cryptococcosis is usually associated with advanced HIV disease (CD4 < 200 cells), but cases are sometimes seen with higher CD4 counts, and even among patients who are HIV-negative.**

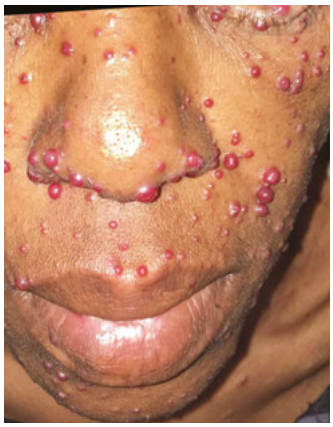


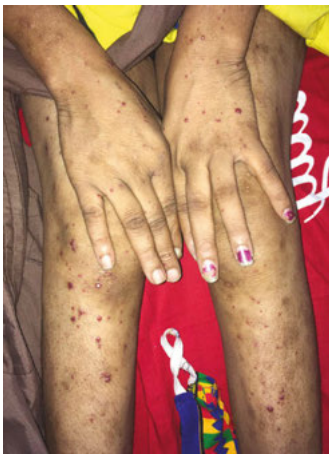


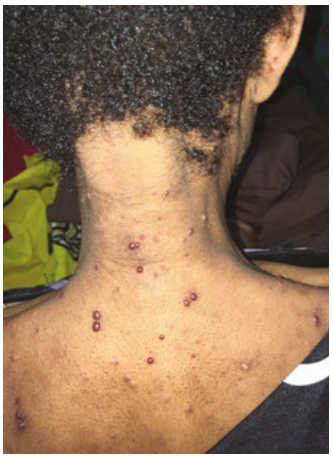


## BACILLARY ANGIOMATOSIS









**CAUSE**
*Bartonella henselae* (gram-negative intracellular bacterium)

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
This rare disease presents with purple papulonodular lesions due to vascular proliferation in the skin. These lesions are neither painful nor itchy. The condition can also infect inner organs, such as the liver and spleen (peliosis), where it appears as ill-defined, hypoechoic lesions on ultrasound.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Cryptococcoma, KS.

In case of doubt, a skin biopsy can be done to diagnose; the pathologist should be informed of the suspected diagnosis to search for basophilic granular material representing the bacteria.

**WHAT IS THE TREATMENT?**
Doxycycline 100 mg bd for 14 days.

**PEARLS AND HIV-RELATED FACTS**

**Bacillary angiomatosis is associated with severe immune suppression; it usually occurs only in patients with CD4 counts < 50 cells/mL.**

## KAPOSI SARCOMA

### CAUSE

Kaposi sarcoma (KS) is caused by human herpesvirus-8 (HHV-8). It is a vascular neoplastic disease affecting the skin, oral mucosa, lymph nodes, and visceral organs, with the skin being the most common site.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Dark purple macula, nodules, and plaques presenting on the skin are the hallmarks of KS. Early lesions can resemble bruises. The lesions often involve the feet and legs; inguinal lesions are common. Lesions found on the trunk can appear parallel to skin tension lines.

Swollen lymph nodes are often also present; obstruction of the lymph channels can cause woody lymphoedema (especially of the legs and genitals).

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

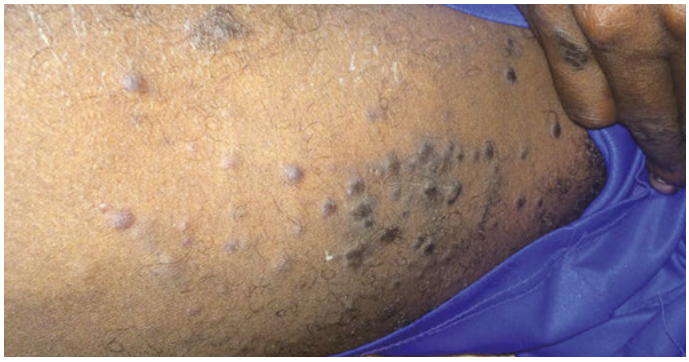
Warts, bacillary angiomatosis, chronic venous disease with induration and hyperpigmentation of the legs, other diseases with inflammatory hyperpigmentation. Ideally, diagnosis should be confirmed by skin biopsy.

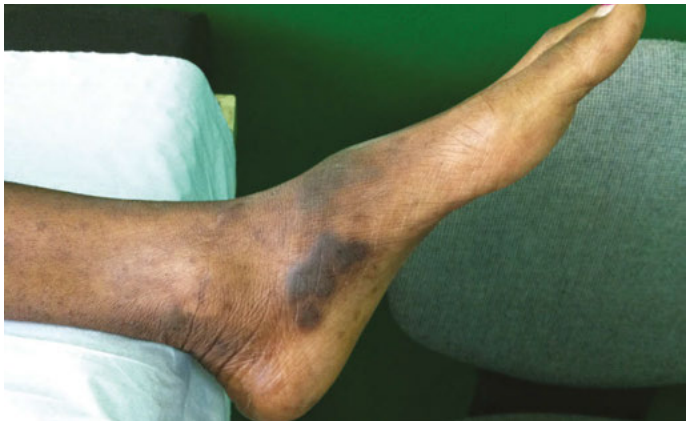
### WHAT IS THE TREATMENT?

Refer to the national guidelines for treatment of KS.

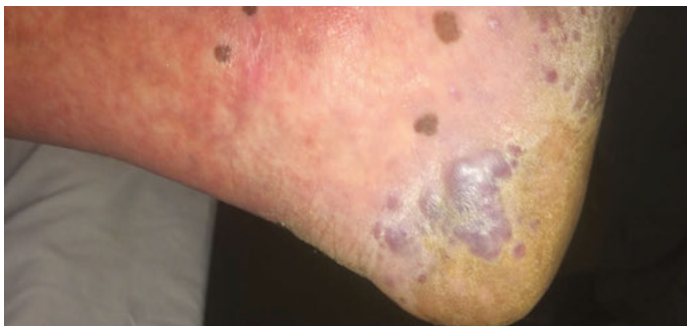
### PEARLS AND HIV-RELATED FACTS

- **KS is the most common cancer found in HIV patients. It is an AIDS-defining disease.**
- **KS can occur regardless of the CD4 count, but is more frequent and aggressive with lower counts.**
- **Systemic involvement is frequent; particularly affected are the stomach and bowel, lungs, and lymph nodes. Lesions in the spleen and liver are also seen on ultrasound.**
- **Systemic KS without involvement of the skin is rare, accounting for probably less than 1% of all cases.**

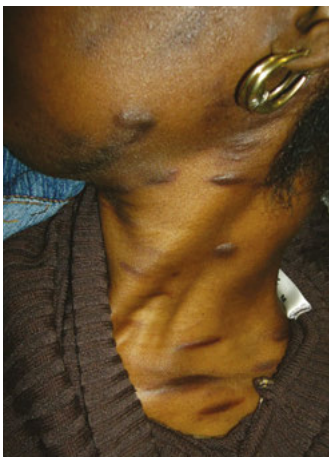


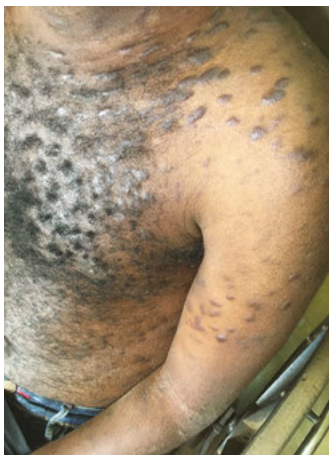


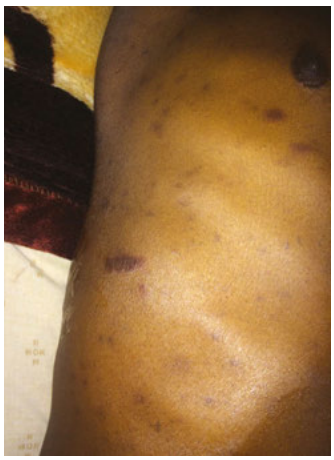
KS lesions predominantly develop on the legs and feet, and in the groin.

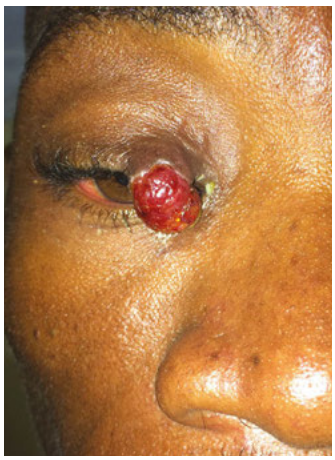


The violaceous colour of KS is more visible on the depigmented skin of vitiligo.









## CUTANEOUS TUBERCULOSIS

### CAUSE
*Mycobacterium tuberculosis* (MTB)

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?
Three different types of skin involvement can be differentiated according to the pathomechanism.

**Exogenous infection**
MTB enters the skin from outside, either through traumatic injuries or surgical procedures performed with unsterilised material. This causes a local infection with small nodules and ulcers (TB chancre). The most common form of exogeneous TB is **tuberculosis verrucosa cutis**, which has a warty appearance. This infection proceeds from an injured dermal layer; in the tropics, it most frequently occurs among barefooted children—but it can occur also on the hands (e.g., among surgeons) or face. Typically the lesions are singular and painless.

**Endogenous infection**
Most often this is seen as **scrofuloderma**, the result of direct spread from an underlying infected lymph node, bone, joints, or testicles. A less common form is **orificial TB**, which presents as ulcers around the nose and the mouth, but can also occur in the perianal region in patients with advanced disseminated TB (sometimes with advanced HIV), caused by autoinfection of the mucocutanous border.

**TB hypersensitivity reaction of the skin**
The form seen most often is **erythema nodosum**, which presents with bruise-like intradermal nodules that are painful to the touch.

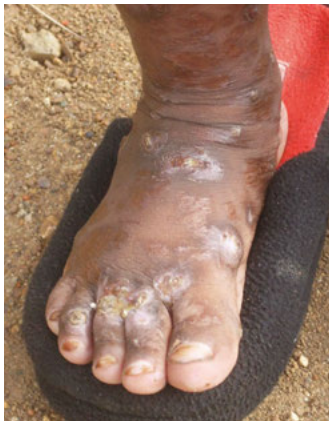
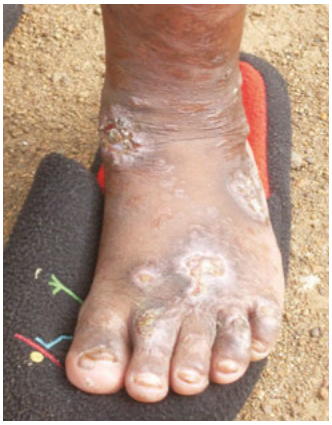
An associated, but slightly different disease is **BCGitis**, which occurs as a complication of the intradermal injection of BCG vaccine (an attenuated live strain of *Mycobacterium bovis*). It presents as local abscess and regional lymphadenitis.

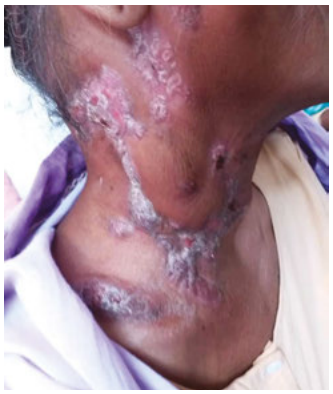
### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?
Other bacterial skin infections (e.g., syphilis, leishamiansis, atypical mycobacteria).

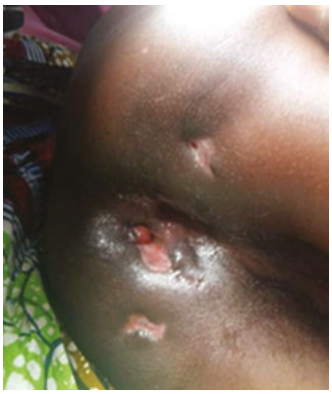
### WHAT IS THE TREATMENT?
- All forms of skin TB are treated with the standard 6-month course of RHZE.
- With BCGitis, the usual therapy is drainage (e.g., needle aspiration). Systemic treatment has little effect, but may be required when treating immunosuppressed children with disseminated disease.

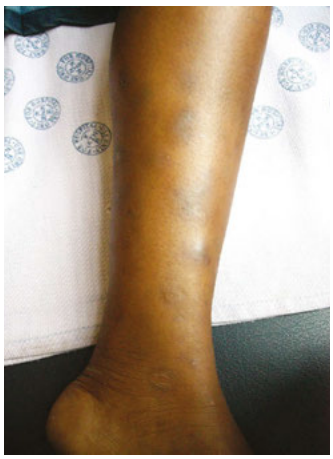
**PEARLS AND HIV-RELATED FACTS**

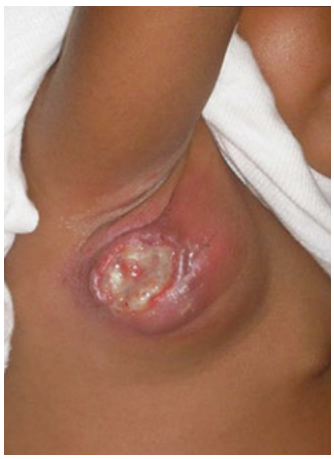
**Hypersensitivity reactions occur more frequently in patients with good immunity (HIV-positive patients with good CD4 counts and HIV-negative patients). The same is true for tuberculosis verrucosa cutis, which requires a good immune response to the mycobacteria, and is usually associated with a positive skin test.**



Tuberculosis verrucosa cutis.

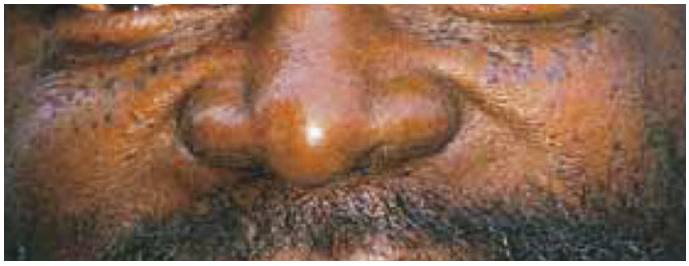

Scrofuloderma.


Orificial perianal TB.


Erythema nodosum.


BCGitis.

## DERMATOSIS PAPULOSA NIGRA



### CAUSE

The cause of dermatosis papulosa nigra (DPN) is unknown, but there may be a familial tendency. The condition appears more frequently on areas of the skin that are exposed to sunlight.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

The lesions are dark brown to black in appearance, and are usually flat-topped or pedunculated 1–5 mm papules. They typically appear around the eyes and on the cheeks, but can also occur on other areas of the face, neck, and upper trunk. The lesions start to develop as early as the mid-20s, and increase with age.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Warts, but DPN lesions usually have a distinctive appearance.

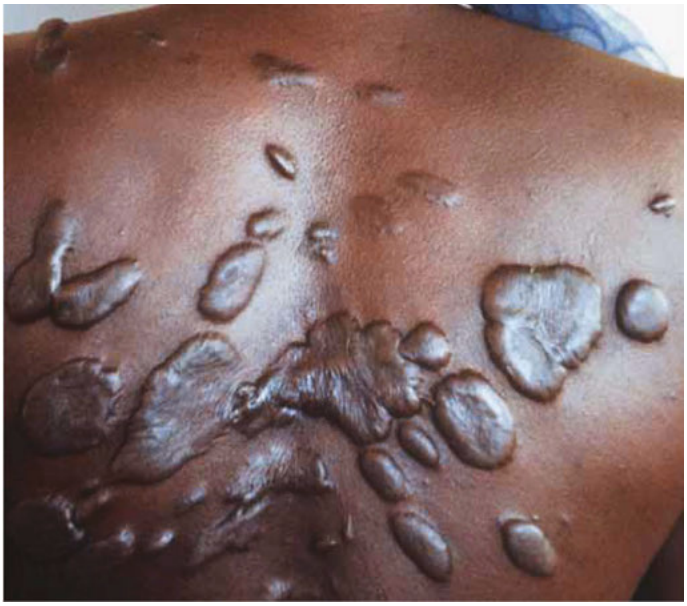
### WHAT IS THE TREATMENT?

No treatment is necessary, unless the patient requests it for cosmetic reasons. Cauterisation is usually successful, but carries the risk of hypo- or hyperpigmentation.

**PEARLS AND HIV-RELATED FACTS**

**DPN is frequently seen in up to 40% of Africans above 30 years of age; it has no association with HIV.**

## KELOIDS



### CAUSE

Overgrowth of fibroblasts and connective tissue in damaged skin areas

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Keloids usually occur around injury sites or scars, but can also be triggered by acne; spontaneous development has also been described. They usually develop between the ages of 20 and 40, rarely occurring in children or the elderly. Keloid growth starts weeks or even months following an injury, and spreads beyond the area originally injured. Firm, skin-coloured nodules or plaques form, most often on the head, neck, shoulders, chest, and upper extremity; these nodules can be intensely itchy, and even painful. They can persist for years, though some may eventually become flattened.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

KS and other skin tumours should be considered—but keloids are usually very obvious.

### WHAT IS THE TREATMENT?

Treatment of keloids is difficult, as simple surgical excision usually leads to regrowth. Intralesional steroid injections are sometimes helpful. Often, surgery and local steroid injections are combined.

### PEARLS AND HIV-RELATED FACTS

**Apart from well-described cases of keloid development around areas of skin injury on the trunk and face due to herpes zoster, keloids do not appear to be associated with HIV.**

# Papulosquamous Lesions

## RINGWORM (TINEA)

### CAUSE

Infection of the skin by dermatophytes (skin-linked fungi). The variations of tinea are named according to the body parts affected: **tinea capitis** affects the head (scalp), **tinea faciei** the face, **tinea corporis** the body, **tinea cruris** the inguinal folds, and **tinea pedis** the feet.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Tinea is characterised by well-demarcated, round, scaly patches on an erythematous ground. The lesions are itchy, and often there is a central clearing and a slightly raised border due to the active inflammation.

Tinea capitis, which can also be seen in HIV-negative children, affects the head and hair; it is often followed by hair loss in the affected area, with broken hair, pustules and scales, and even abscesses (due to bacterial superinfection).

Tinea faciei and corporis can affect any area of the face and body.

Tinea cruris (sometimes referred to as jock itch) affects the inguinal folds, extending to the upper inner thigh and, in severe cases, to the lower abdomen. Fine scaling is seen at the border; often, inflammatory hyperpigmentation results in only the hyperpigmentation being visible. It is usually itchy, especially in warm environments, and is more common in males.

Tinea pedis affects the area of the foot that a flat shoe would cover. It usually presents with scaling, hyperkeratosis, and fissuring of the soles and the skin between the toes.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Psoriasis mainly affects the extensor surfaces and nails. Seborrhoeic dermatitis mainly affects the scalp, face, and skin folds (axilla). The ring-shaped elevated border is usually not present with either of these conditions.

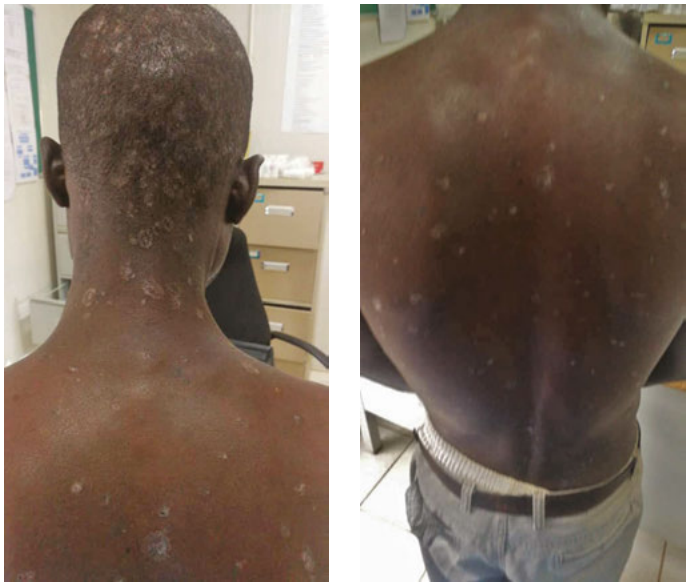
In cases of severe hyperpigmentation, tinea can be confused with KS.

### WHAT IS THE TREATMENT?

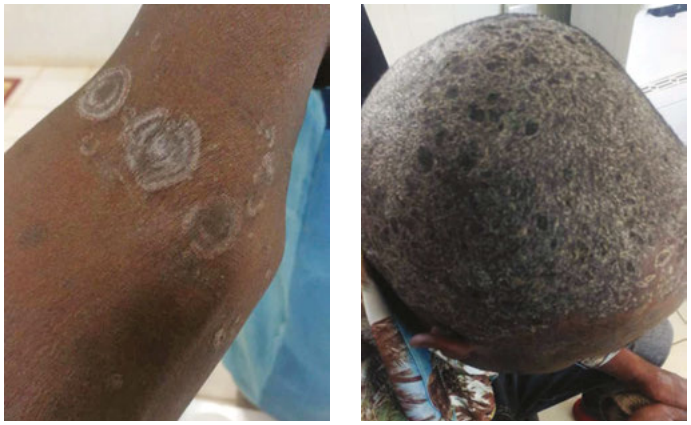
- Clotrimazole cream or other topical antifungals 2x/day for 2–4 weeks
- Oral fluconazole 3–6 mg/kg/day for 6 weeks
- Tinea capitis requires oral antifungals to penetrate the hair shaft—e.g., fluconazole 8 mg/kg per week for 4–8 weeks or griseofulvin (20–25 mg/kg/d) od for 4–6 weeks.

**PEARLS AND HIV-RELATED FACTS**

**Tinea may flare up as part of IRIS when ART is started.**



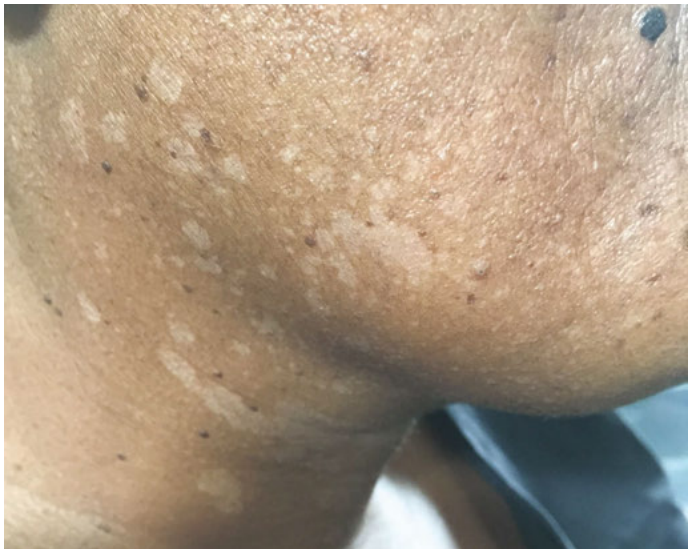
Tinea capitis with tinea corporis (left), tinea corporis (right).



Tinea corporis (left), tinea capitis (right).

# Papulosquamous Lesions

## TINEA VERSICOLOR



**CAUSE**
Common benign fungal infection of the skin with *Malassezia spp.*

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Tinea versicolor is characterised by round, hypopigmented macula with very fine scales, which become more visible when you stretch the skin. It is found most frequently on the chest and back. The lesions are not itchy. Tinea versicolor can occur regardless of HIV status; in HIV-positive patients, it is usually more extensive and more often involves the face.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Other fungal skin infections, vitiligo.

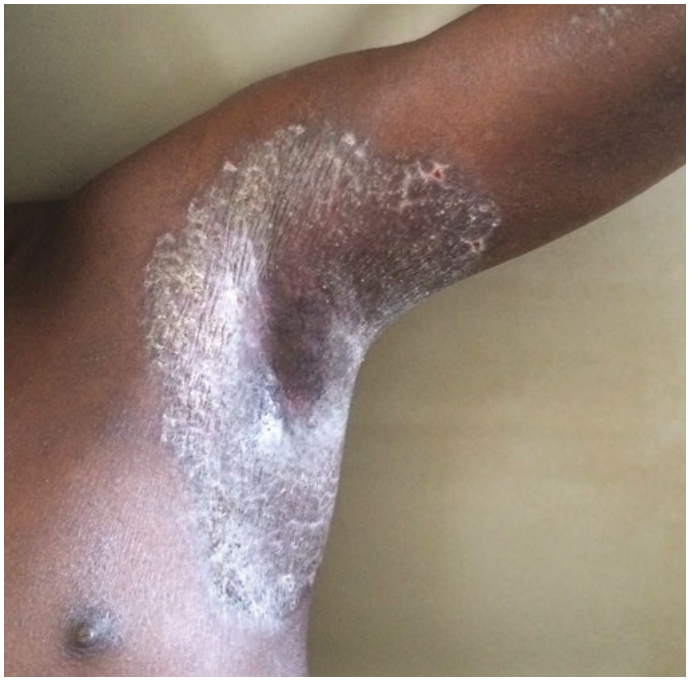
**WHAT IS THE TREATMENT?**
- Clotrimazole cream or other topical antifungals 1x or 2x/day for 2–4 weeks
- Oral fluconazole 300 mg/week for 2–4 weeks

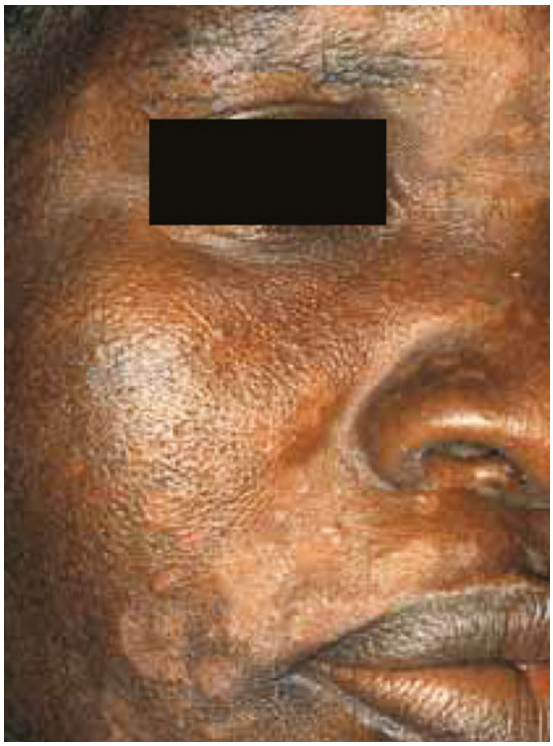
Note that pigmentation will increase with exposure to sunlight after treatment.

**PEARLS AND HIV-RELATED FACTS**
- **Metabolites of *Malassezia* inhibit melanocyte growth, which explains the hypopigmentation. With stronger inflammation, the lesions may be reddish in colour, or even hyperpigmented.**
- **In patients who are more susceptible to tinea versicolor, recurrences may be chronic. In some cases, prophylactic treatment (e.g., 1 tablet of fluconazole per month) can be given.**

# Papulosquamous Lesions

## SEBORRHOEIC DERMATITIS





### CAUSE

Seborrhoeic dermatitis is an inflammatory skin disorder likely associated with lipophilic fungus (*Malassezia*). Although the exact mechanism of the disease is not clear, it seems to involve sebum and the sebaceous glands (oil glands) of the skin.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Seborrhoeic dermatitis presents with thin, scaly erythematous plaques. The scales are yellowish and greasy; on the scalp, they can present as dandruff. Pruritus is usually mild. The condition affects mainly those areas of the skin that have oil glands: the scalp, areas of the face (the eyebrows, nasolabial folds, and behind the ears) and the intertriginous body folds (axilla, groin, and under the breast). Severe cases can present as erythroderma, which involves more than 90% of the body surface and may require hospital admission. Relapses and remissions are seen; these may require long-term treatment.

Lesions can be complicated by bacterial superinfection (mainly staphylococci and streptococci), resulting in impetigo; and by secondary herpes simplex virus (HSV), resulting in the sudden appearance of painful vesicles (eczema herpeticum).

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Psoriasis, especially as it also affects the scalp. However, the scales associated with psoriasis are finer and silvery, whereas with seborrhoeic dermatitis they are thicker and an oily yellow. In cases of erythroderma, it may be difficult to differentiate. Tinea is another differential diagnosis.

### WHAT IS THE TREATMENT?

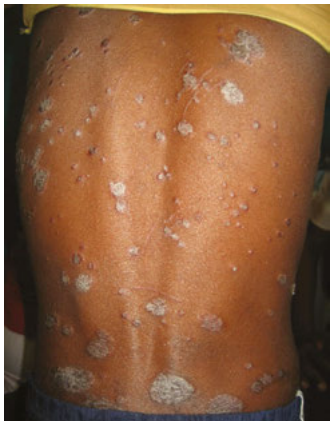
- Clotrimazole or miconazole cream or ointment, possibly in combination with short-term topical steroids, e.g. hydrocortisone 1% (not recommended for long-term use)
- Oral antifungals, e.g., fluconazole 300 mg/week for 2–4 weeks; or itraconazole 200 mg daily for 1 week, followed by 200 mg once every 2 weeks
- Treat secondary infections with antibiotics (impetigo) or acyclovir (HSV).

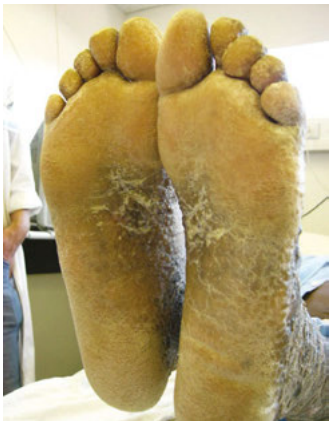
### PEARLS AND HIV-RELATED FACTS

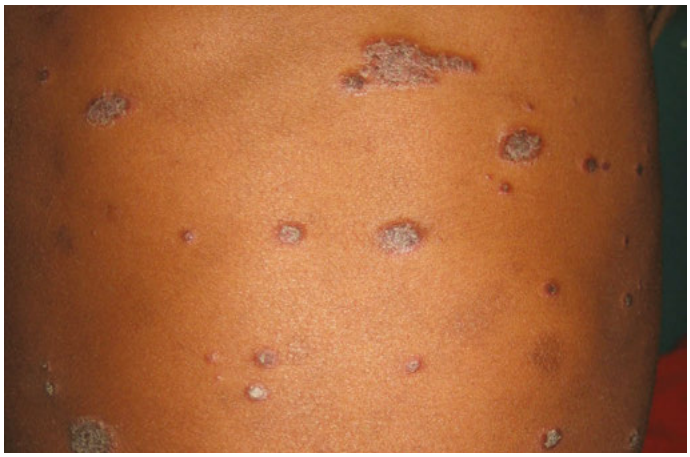
**The extent of seborrhoeic dermatitis inversely correlates with the CD4 count: the lower the count, the more extensive the rash.**

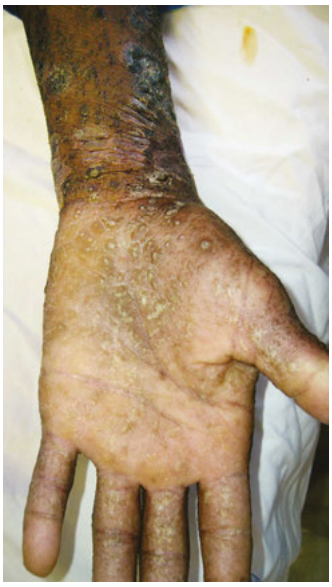
# Papulosquamous Lesions

## PSORIASIS









**CAUSE**
Psoriasis is an inflammatory autoimmune disorder of the skin caused by dysregulated T-lymphocytes.

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Psoriasis presents as well-defined, thickened plaques with silvery scales, typically involving the scalp and the extensor sides of extremities (e.g., elbows, knees); the palms, soles, and nails are also affected. Among HIV-positive patients, atypical forms with drop-like lesions disseminated over the trunk, legs, and arms are also seen (guttate psoriasis). Severe cases can present as erythroderma, which involves more than 90% of the body surface; in such cases, hospital admission may be required.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Seborrhoeic dermatitis, tinea corporis.

**WHAT IS THE TREATMENT?**
**Scalp**
- Tar or ketoconazole shampoo
- Seborrhoeic scalp ointment (5% coal tar solution + 2% sulphur + 2% salicylic acid)

**Body**
- Topical steroids (1% hydrocortisone cream)

**Palms and soles**
- 5–10% salicylic acid
- Topical steroids

In severe cases, systemic steroids may be required.

In other settings, phototherapy with UV light is recommended; because this is not available in our setting, exposure to natural sunlight for short periods of time can help. Be cautious of overexposure, though; sunlight also harms black skin, and can cause sunburn

**PEARLS AND HIV-RELATED FACTS**

**Psoriasis is generally rare in African patients; when it does appear, it can be a sign of underlying HIV infection. More severe presentations are associated with low CD4 counts.**

# Papulosquamous Lesions

## PHOTOTOXIC REACTIONS (PHOTODERMATITIS) AND PELLAGRA

### CAUSE

**Photosensitivity** is an inflammatory reaction caused by an abnormal response to sunlight. **Pellagra** is a disease caused by deficiency of vitamin $B_3$ (niacin); phototoxic skin reaction is part of the disease.

### HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?

Phototoxic reactions occur as non-scarring, erythematous, pruritic papules, vesicles, plaques, or nodules. They usually involve the face, the V and nape of the neck, the arms, and the shins and feet.

Phototoxic drug reactions can be caused by a variety of medications. In our setting, the likely causative agents are sulpha drugs (CPT), tetracycline, anti-TB drugs, and NSAIDs, but also EFV.

Pellagra is a systemic disease with skin changes in the areas described. In addition to dermatosis, it is characterised by diarrhoea and dementia (together referred to as DDD). INH is a well-known cause of pellagra. It interferes with the activity of vitamin $B_6$ (pyridoxine); the suppressed $B_6$ activity in turn reduces the body's ability to produce vitamin $B_3$. Pellagra is therefore seen in patients who have borderline low niacin levels when they start INH.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

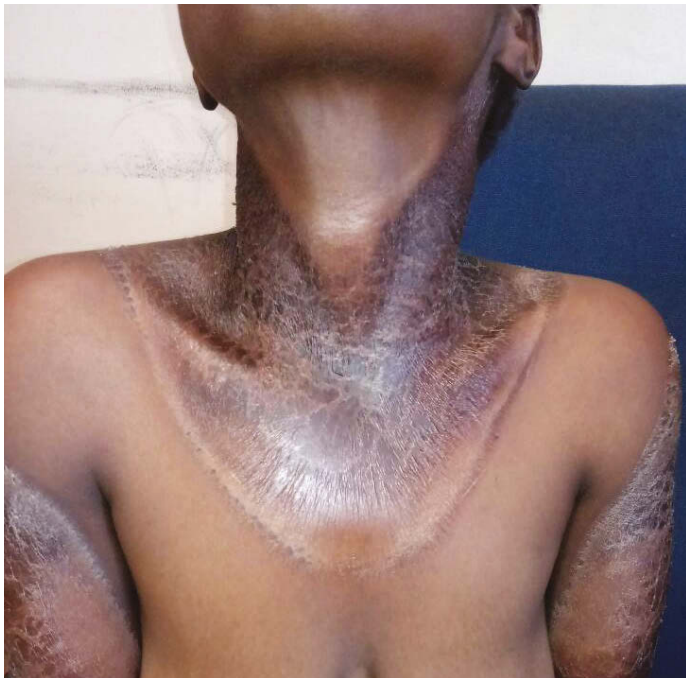
Less common diseases observed with phototoxicity are porphyria cutanea tarda (PCT) and pseudoporphyria. Actinic lichenoid leucomelanoderma of HIV is a rare photosensitive dermatological condition that has been specifically described in South Africa.

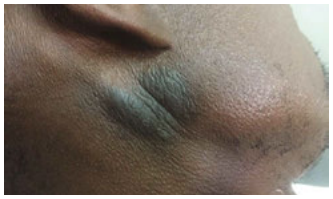
### WHAT IS THE TREATMENT?

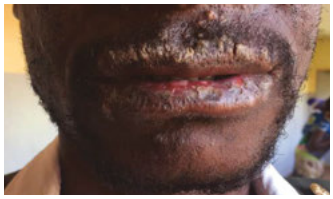
- Stop use of the offending drug.
- Avoid sunlight; use protective clothing and hats, and possibly sunscreen lotion.
- **Pellagra**: 300 mg nicotinamide po daily for 3–4 weeks

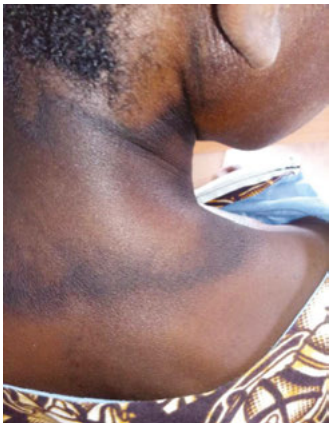
### PEARLS AND HIV-RELATED FACTS

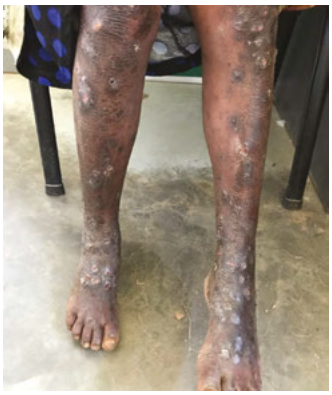
- **Photosensitive disorders are common, affecting up to 5% of HIV-positive patients. Pellagra is also more common among patients with HIV.**
- **Photosensitivity typically occurs in patients with CD4 counts below 200 cells/mL, and more often when the count is below 50.**

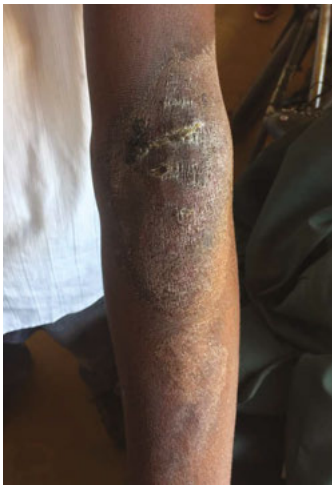












# Blisters & Ulcers

## HERPES SIMPLEX AND GENITAL HERPES

**CAUSE**
HSV-1 and HSV-2, transmitted through direct skin contact with a lesion or infected secretions. Asymptomatic virus shedding between episodes is possible.

**HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?**
Herpes simplex presents initially with small grouped vesicles, which are (very) painful. Often the pain starts as a burning, itching, tingling sensation hours to days before the blisters become visible. After a while, the vesicles burst and ulcers (which may have a crust) develop.

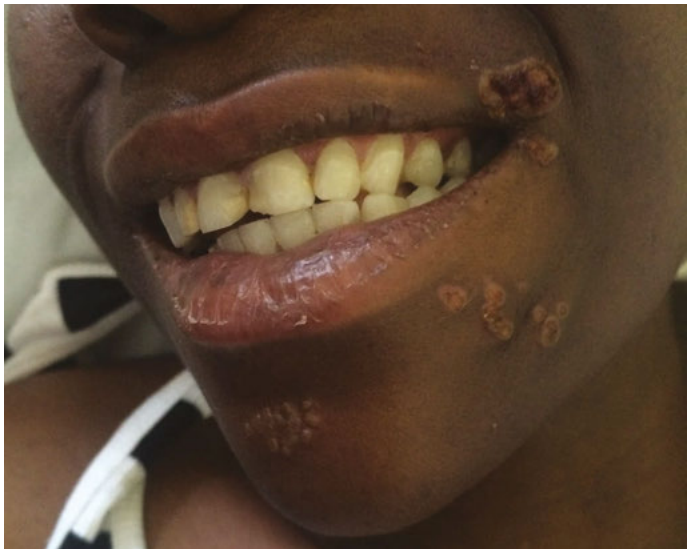
Lesions are commonly found around the mouth (perioral, associated more with HSV-1), and in the genital and anal areas (associated more with HSV-2). The lesions tend to heal, but often recur in the same location, as chronic HSV infection is present in the nerve root. Recurrence and the severe pain associated with the lesions are clues to the diagnosis. (Confirmation by histology and viral culture is possible, but not readily available in our setting.)

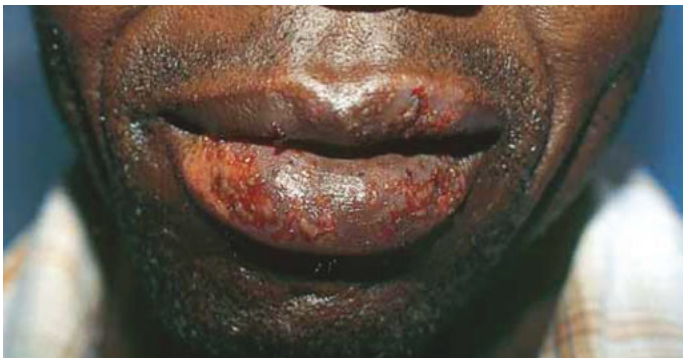
**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Ulcerations in the genital area should make you think of other STIs, such as primary syphilis (which has painless ulcers), chancroid, and lymphogranuloma venerum (LGV) (characterised by marked inguinal lymphadenopathy).

**WHAT IS THE TREATMENT?**
- Acyclovir 400 mg 3x/day for 7–10 days; start as soon as possible.
- An alternative to acyclovir with a lower pill burden is valacyclovir 1000 mg bd for days (2 g po bd for 1 day for herpes labialis); however, this is rarely available in our setting.
- Start or continue ART (check viral load and CD4).
- Acyclovir cream may be helpful in cases of perioral disease, but it is not highly effective.
- In addition to analgesics (paracetamol, ibuprofen), topical antiseptics, antibiotics, and calamine can be given.

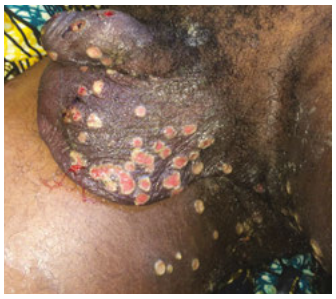
If the patient has frequent attacks of genital HSV (i.e., 8–10 or more per year), they should receive chronic suppressive therapy (e.g., acyclovir 400 mg bd for a longer period).

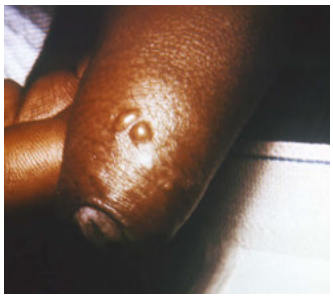
**PEARLS AND HIV-RELATED FACTS**
- **HSV also affects immune-competent hosts, but the infection is usually self-limiting, healing within a week or two. With HIV patients, lesions may be more frequent, more severe, and persist for longer. (HSV becomes AIDS-defining when the lesions are present for longer than one month.) HSV can manifest as IRIS after the patient is started on ART.**
- **Lesions may leave behind a residual postinflammatory hyperpigmentation, which can last for months after the ulcers have resolved**
- **Patients not responding to acyclovir should be referred for further examination and possible skin biopsy.**





Herpes lesions around the mouth.

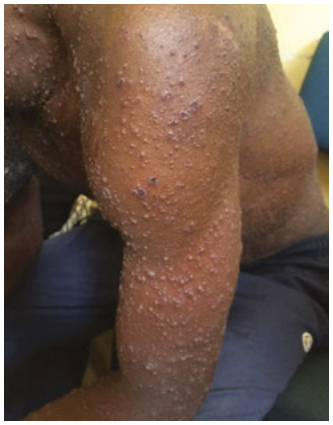


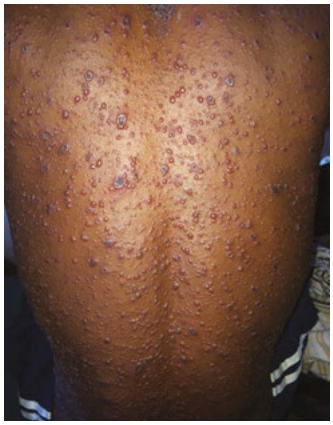


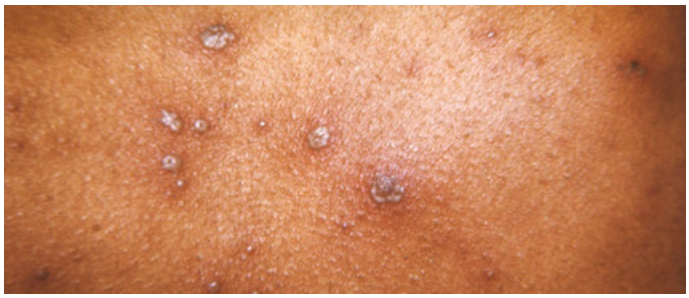
Examples of HSV-2 lesions (genital herpes).

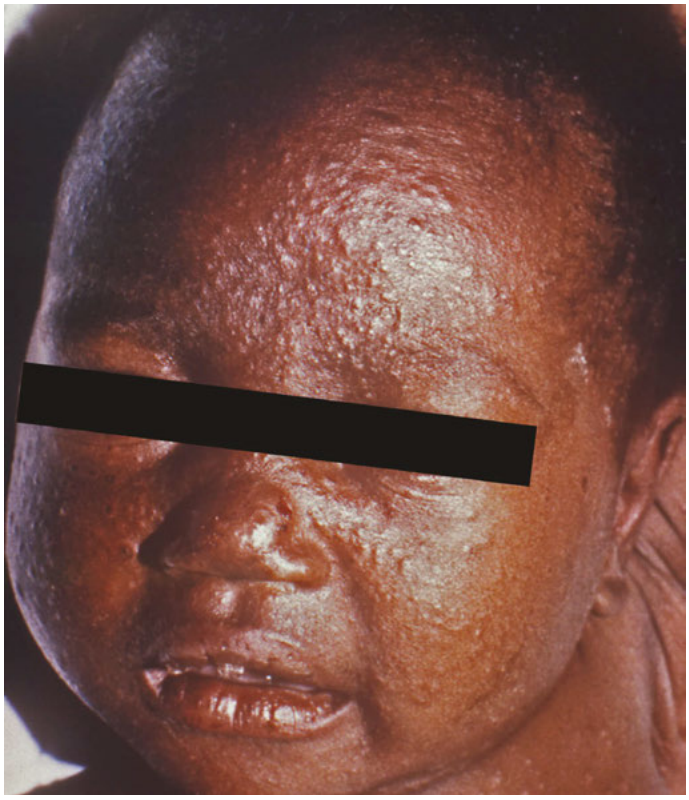
# Blisters & Ulcers

## CHICKENPOX









**CAUSE**
Varicella-zoster virus (another human herpesvirus—the same virus that causes herpes zoster)

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Chickenpox presents with papules, which become vesicles and pustules; they then form small ulcers and become crusted. The rash involves the face and trunk, and (less often) the extremities.

Typically, lesions are seen in all stages of development simultaneously, meaning that active vesicles and pustules, superficial erosions, and crusted lesions are all present at the same time. Hyperpigmentation may be seen around the vesicles.

Chickenpox is typically seen in children, but also occurs in adults, especially with immune suppression. With HIV-positive patients, it is more severe and extensive, and may involve internal organs, e.g., the lung (pneumonitis) and liver (hepatitis).

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
With severe immune suppression, disseminated cases are seen, which makes it difficult to distinguish from other generalised blistering diseases.

**WHAT IS THE TREATMENT?**

- Acyclovir 800 mg 5x/day for 5–7 days
- In cases of systemic involvement, IV administration may be preferred: 10–20 mg/kg infused over 1 hour every 8 hours.
- An alternative to acyclovir with a lower pill burden is valacyclovir 1000 mg tds for 5–7 days; however, this is rarely available in our setting.
- Start or continue ART (check viral load and CD4).

**Co-treatment**

- Similar to herpes zoster.

**PEARLS AND HIV-RELATED FACTS**

- **With HIV patients, the interval between chickenpox and herpes zoster can be short.**
- **On skin of colour, lesions may leave behind post-inflammatory hyperpigmentation, which can last for months after the original rash resolves.**

# Blisters & Ulcers

## HERPES ZOSTER

### CAUSE

Varicella-zoster virus (a human herpesvirus). The virus reactivates from a chronic neural root infection obtained during chickenpox.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Zoster presents with small, painful vesicles, which are distributed unilaterally according to a dermatome. They typically do not cross the midline of the body. Mainly affected are the head, neck, and trunk. Vesicles can burst and crusted ulcers develop. Lesions tend to heal with significant scarring, and often have post-herpetic neuralgia, which can last for weeks to months. The dermatomal distribution is the hallmark for making the diagnosis, although it can affect multiple dermatomes.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

With severe immune suppression, disseminated cases are seen, which makes it difficult to distinguish from other generalised blistering diseases.

### WHAT IS THE TREATMENT?

- Acyclovir 800 mg 5x/day for 7–10 days; start as soon as possible.
- In cases of ophthalmic involvement, IV administration may be preferred: 10–20 mg/kg infused over 1 hour every 8 hours.
- An alternative to acyclovir with a lower pill burden is valacyclovir 1000 mg tds for 5–7 days; however, this is rarely available in our setting.
- Start or continue ART (check viral load and CD4).

### Co-treatment

Analgesics:

- Paracetamol 500 mg 3–6 times per day or ibuprofen 600 mg 2–3 times per day
- Amitriptyline 25–75 mg at night
- Tramadol, and even morphine, may be needed.

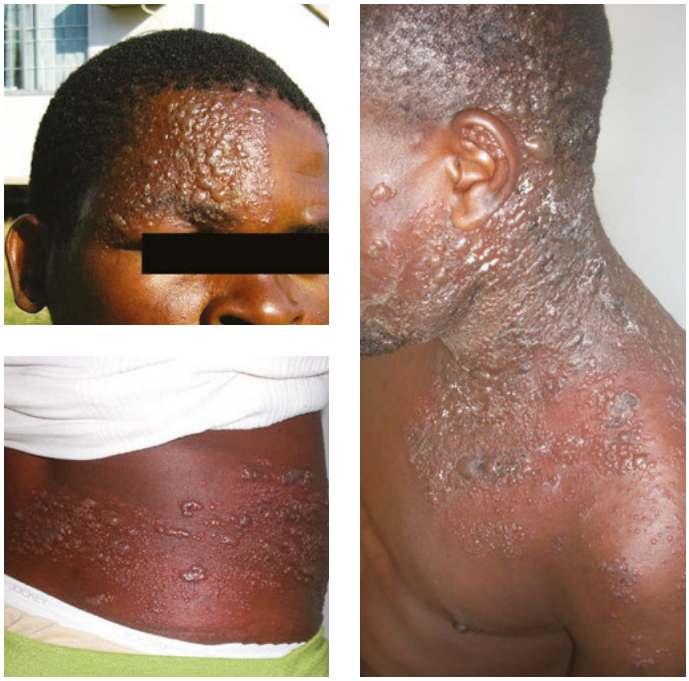
Antibiotics:

- Fucidin or mupirocin cream 3x/day
- Systemic antibiotics (as needed)

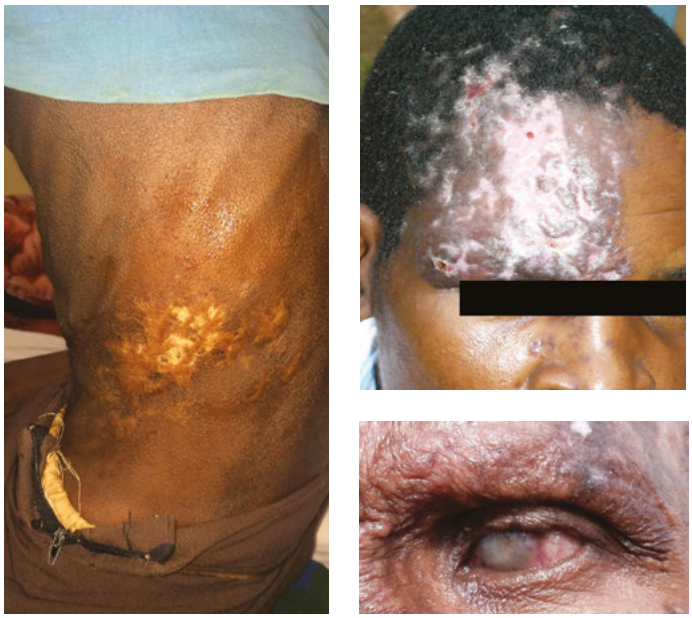
Topical calamine lotion.

**Post-herpetic neuralgia**: Similar pain medications as above can be used, especially in combination with amitriptyline (tricyclic antidepressant as co-analgetic).

Local therapy with capsaicin cream or other heat rubs may ameliorate the pain. Steroids do not appear to be effective.



Acute herpes zoster.



Sequelae of herpes zoster.

### PEARLS AND HIV-RELATED FACTS

- **Herpes zoster is common among HIV patients, and can occur early in the course of the disease. It can also manifest as IRIS after starting ART.**
- **Among non–HIV-infected patients, it is mainly seen in the elderly.**

# Blisters & Ulcers

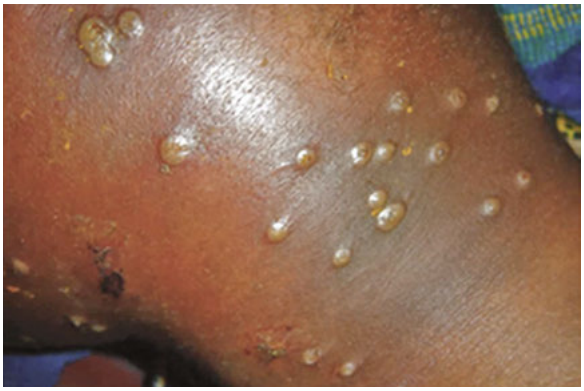
## MPOX (MONKEYPOX)

### CAUSE
Mpox virus (formerly called the monkeypox virus)

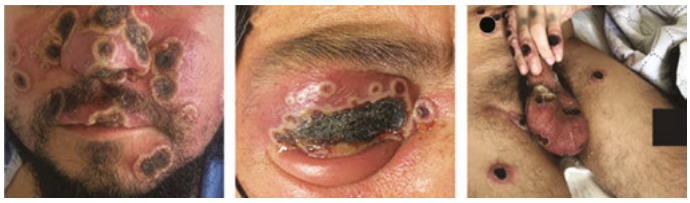
### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?
Patients may initially have general symptoms, such as headache, fever, and sore throat. Mpox presents with a rash that forms vesicle and pustules, then later becomes ulcerative and crusted; all lesions follow the same progression. The average number of skin lesions is around 15, but can be greater than 100. Swollen lymph nodes are also seen. The ulcers can become haemorrhagic, and necrotic lesions can be seen, especially among patients with low CD4 counts. Mpox usually lasts 20–40 days. Systemic involvement with respiratory and neurological symptoms frequently occurs among HIV-positive patients.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?
The main differential diagnosis is chickenpox. However, with chickenpox the lesions are present in various stages at the same time; to be Mpox they must be more uniform, and at the same stage of development.

### WHAT IS THE TREATMENT?
Antiviral treatments (tecovirimat, cidofovir) exist, but are not available locally.



Mpox in an HIV-negative child.



Mpox in HIV-positive patients.

### PEARLS AND HIV-RELATED FACTS

- **In the non-sexually transmitted form of the disease (in West and Central Africa), the rash is most pronounced on the head and face; children are frequently affected.**
- **Vaccination: Two different vaccines (Mvanex and Jynneos) are internationally available. Vaccination can also be given as postexposure prophylaxis.**

# Blisters & Ulcers

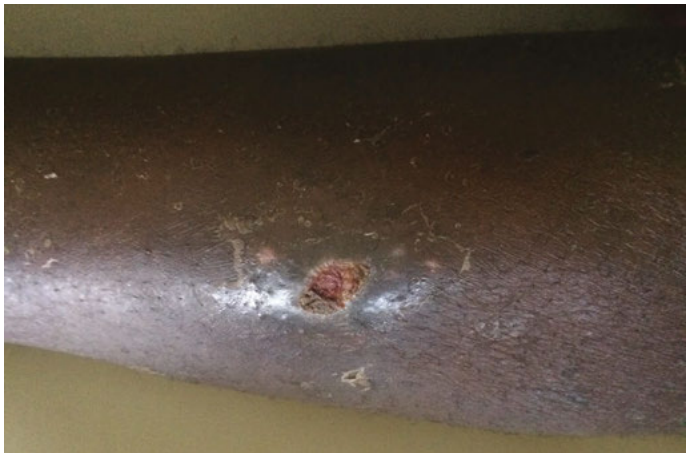
## ECTHYMA

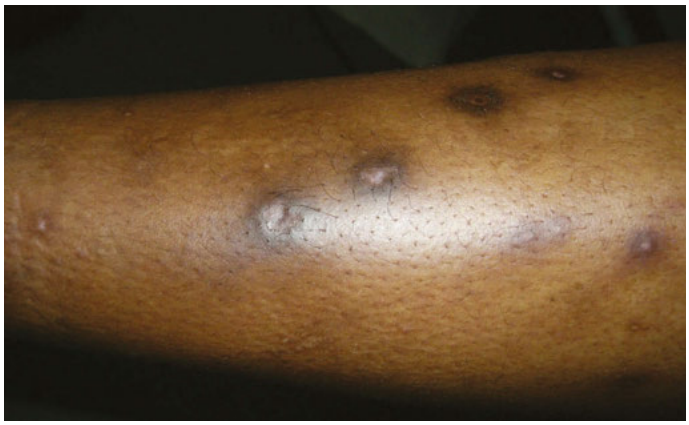
**CAUSE**
An ulcerating form of skin infection, ecthyma is usually caused by *S. aureus* and streptococci.

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Ecthyma typically presents as punched-out, shallow ulcers occurring mostly on the extremities, or at sites of insect bites or previous trauma. Ecthymata are painful, and frequently leave scars and hyperpigmentation after healing.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Ecthyma is usually obvious from the clinical picture.

**WHAT IS THE TREATMENT?**
- Topical application of mupirocin 2% or Fucidin cream
- Amoxy-clav (amoxicillin and clavulanate) 625 mg po bd for 5 days





# Blisters & Ulcers

## DRUG REACTIONS

**CAUSE**
Allergy to drugs; especially sulpha drugs (CPT) and anti-TB drugs, as well as ART (especially NVP and EFV) and anti-epileptics.

**HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?**
The initial morphology of the lesions is varied; it can comprise maculopapular target lesions and blistering. The blisters become larger bullae, which erode, revealing the pink, moist, erythematous dermis. SJS/TEN usually develops 2–3 weeks after starting the offending drug, but later appearance (at six weeks or more) is also seen. If severe, it can involve the mucosal membranes in the mouth, genitals, and eyes.

The percentage of body surface area affected by the bullae determines the classification of the condition. If less then 10% of the body is affected, it is **Stevens–Johnson Syndrome (SJS)**; if more than 30% is affected, it is **toxic epidermal necrolysis (TEN)**; and if coverage is between 10–30%, it is **SJS/TEN overlap**.

The acute phase of SJS/TEN lasts about 10 days; skin eruption and mucosal lesions develop, often accompanied by fever. The re-epithelialisation phase can last as much as a month or more. Patients may also have renal and liver function abnormalities; hepatic involvement is frequently seen with NVP and EVF reactions.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Other generalised bullous conditions.

**WHAT IS THE TREATMENT?**
Immediately stop use of all drugs that may be causing the condition.

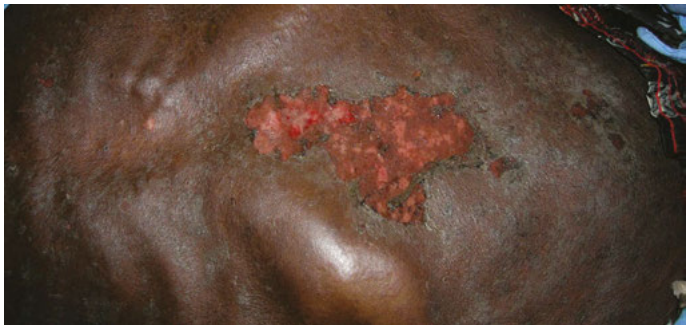
**Co-treatment**

- Analgesics, especially when painful, burn-like lesions are present. In severe cases, morphine may be needed.
- Antibiotics. Local topical antibiotics may be helpful, but be vigilant for sepsis, and start systemic antibiotics early.
- Antiseptic mouthwash
- Eyedrops containing erythromycin and steroids are helpful. (Avoid silver sulfadiazine topical eye drops, as these can worsen SJS/TEN). Consult ophthalmology early.
- Fluid replacement (high fluid loss occurs through exposed skin)

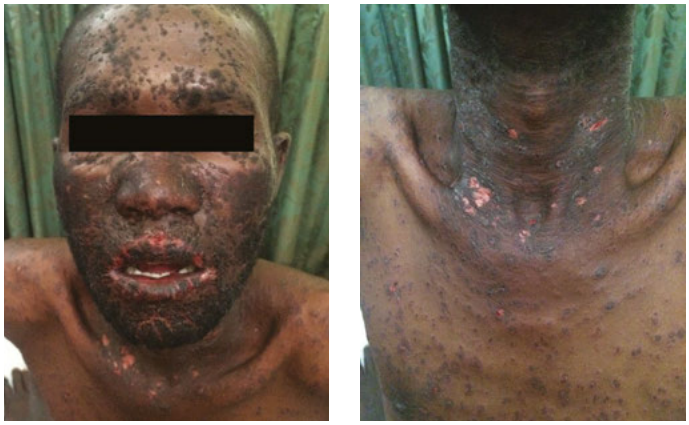
Steroid therapy for SJS/TEN is a controversial topic, as studies have shown conflicting results. One needs to balance the reduction of the skin reaction (positive) against immune suppression, which allows bacterial entry and sepsis (negative).

**PEARLS AND HIV-RELATED FACTS**

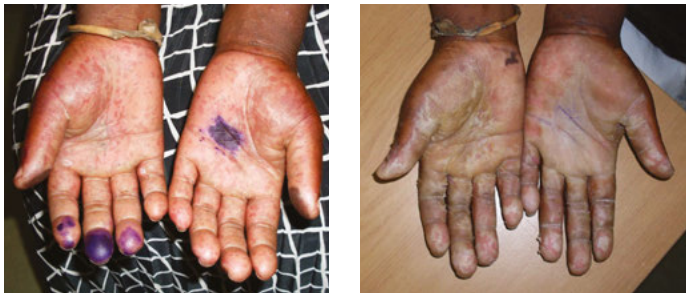
**HIV-positive patients have a much higher incidence of allergic drug reactions than non-infected patients, possibly as much as 100 times higher. This is due to immune deregulation, which HIV causes on top of simple immune suppression.**



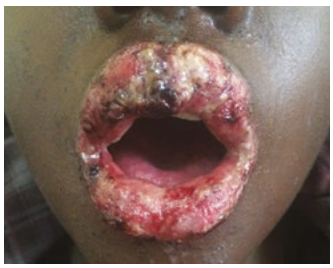
Burn-like lesions.



Male SJS patient.



NVP reaction (left); two weeks later (right).



Female TEN patient.

# Blisters & Ulcers

## OTHER BLISTERING DISEASES

### CAUSE

Acquired blistering diseases are usually caused by autoantibodies against intradermal (pemphigus group) or subepidermal (pemphigoid group) structures.

### HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?

The typical skin lesions of **pemphigus foliaceus** are blistering erosions with overlying cornflake-like scales. Pemphigus foliaceus occurs mainly on the scalp, face, and upper trunk, with no involvement of the mucosa.

The lesions associated with **pemphigus vulgaris**, a related condition, are also flaccid blisters or erosions, with overlying crust, that heal with hyperpigmentation. Pemphigus vulgaris affects the mucosa, and often involves the mouth; oral lesions may be the first lesions seen.

The characteristic clinical feature of superficial, intradermal pemphigus blisters is that they rupture easily and show the Nikolsky sign. The **Nikolsky sign** refers to the production of new blisters or the extension of existing ones under lateral pressure with the finger on normal skin. Pemphigus is most often seen in older adults, but it can occur also in younger patients.

**Bullous pemphigoid** lesions start as itchy, eczematous papules or plaques. Later, tense blisters arise, often still itchy, which do not rupture easily. The Nikolsky sign is negative; the blisters are located deeper in the skin. Erosions, crusting, and secondary bacterial infections are seen. Medications possibly associated with pemphigoid are spironolactone, phenothiazine, furosemide, antibiotics, angiotensin-converting enzyme inhibitors, and metformins.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Other conditions rarely occur; patients should be referred for dermatological examination and skin biopsy.

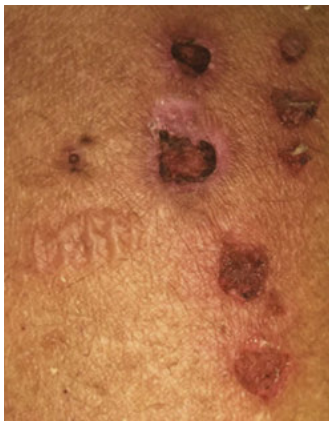
### WHAT IS THE TREATMENT?

**Pemphigus foliaceus**

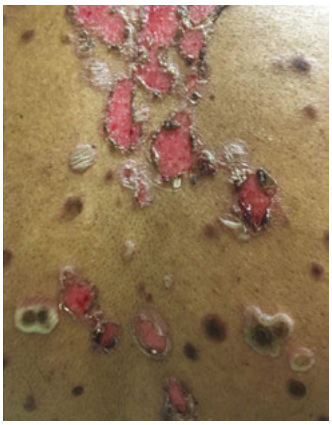
Oral glucocorticoids: prednisone 0.5 mg/kg in mild cases, 1 mg/kg or more in severe or rapidly progressing cases.

**Bullous pemphigoid**

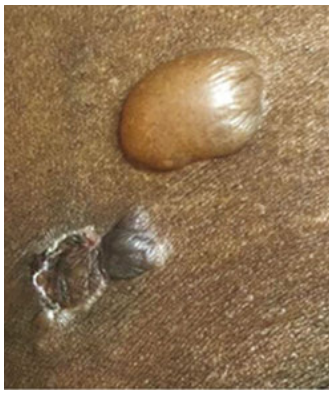
Potent topical steroids or possibly systemic prednisolone (usually in lower doses than with pemphigus).



Pemphigus foliaceus.



Pemphigus vulgaris.



Bullous pemphigoid.

### PEARLS AND HIV-RELATED FACTS

- **Blistering diseases in adults are predominantly acquired autoimmune diseases. Although children can also have inherited blistering conditions (e.g., PCT), here as well, acquired diseases are more common.**
- **Little is known about autoimmune blistering diseases in HIV patients, except that they seem to affect male patients more frequently, and that the age of onset is younger (30s to 50s) than in the general population (50s to 70s).**

# Pustules & Abscesses

## FOLLICULITIS AND FURUNCULOSIS

### CAUSE

*S. aureus* is the most common bacterial cause of skin infections.

**Folliculitis** refers to superficial bacterial infection and inflammation, with pus present only in the upper dermis and epidermis.

A **furuncle** (or boil) is a bacterial infection of the hair follicle, in which pus extends through the dermis into the subcutaneous tissue, with formation of a small abscesses. If the lesion is draining from multiple hair follicles, it is called a **carbuncle**.

### HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?

The lesions of folliculitis are small, discrete pustules around hair follicles; they are often present on the scalp, buttocks, and extremities.

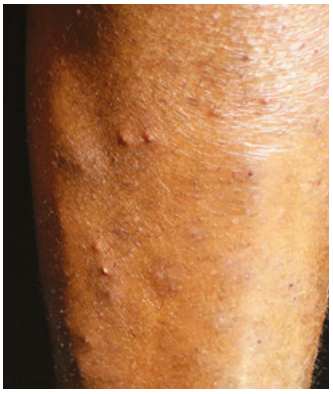
Furuncles are painful, larger papules with walled-off pus. A lesion eventually develops into a fluctuant abscess and opens to the skin surface. Furuncles (and carbuncles) occur mainly in skin areas with hair, such as the face, axillae, and buttocks.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

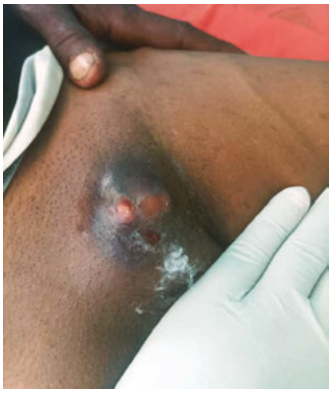
Acne, pustular drug reactions.

### WHAT IS THE TREATMENT?

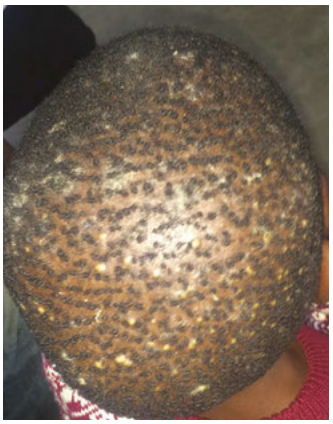
- Topical antibacterial ointments, e.g., Fucidin and mupirocin creams
- Severe and recurrent cases: flucloxacillin 500 mg tds for 10 days. An alternative is amoxi-clav 625 mg bd for 10 days.
- Large or deep nodular lesions: incision and drainage, with antibiotic coverage.

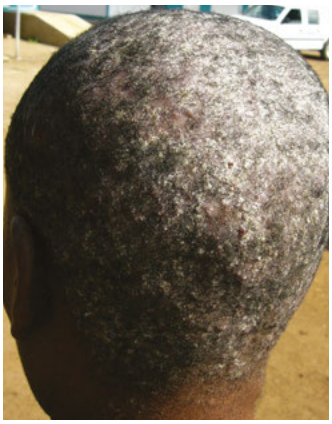


Folliculitis on the leg.



Furuncles.





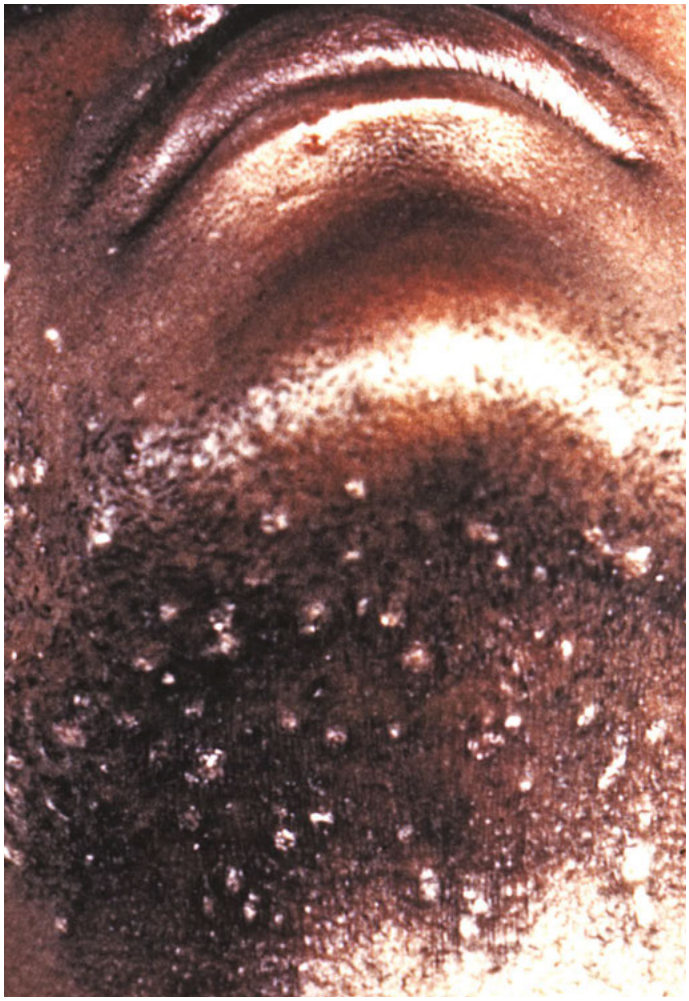
Examples of scalp folliculitis.

### PEARLS AND HIV-RELATED FACTS

- **Folliculitis and furuncles occur frequently in immune-competent patients, but with HIV coinfection they are more extensive and recur more often.**
- **Bacterial folliculitis can recur as IRIS after ART is initiated.**
- **In recurrent cases, consider nasal carriage of *Staph aureus*. In these cases, application of nasal mupirocin cream for 10 days may reduce both colonisation and recurrence.**

# Pustules & Abscesses

## PSEUDOFOLLICULITIS BARBAE



**CAUSE**

Pseudofolliculitis barbae (also called **razor bumps**) is an inflammatory condition affecting skin areas with shaved hair. The causative step is the penetration of the shaved hair shaft into the dermis or epidermis. This occurs frequently among Africans because the hair follicles are oriented more obliquely and hair growth is more coiled.

**HOW DOES IT PRESENT?**
**HOW IS IT DIAGNOSED?**

Pseudofolliculitis barbae typically presents with follicular-based papules; erythema or pustules may also be seen. Razor bumps are mildly painful or itchy. Scars can form as depressed grooves or even as keloids; hyper- and hypopigmentation are also seen. The changes commonly develop in the region of the beard, but also in the axillae or suprapubic regions, if these are shaved.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**

The condition is usually obvious from its appearance.

**WHAT IS THE TREATMENT?**

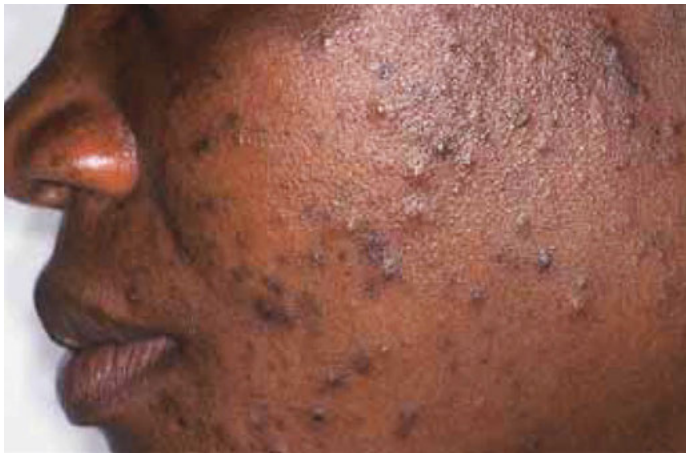
- Razors (or clippers) which prevent close shaves are recommended. If traditional razors are used, it is better to use single-blade razors, shaving in the direction of hair growth without stretching the skin. Reducing the frequency of shaving to two or three times a week should reduce the occurrence of ingrown hairs.
- Low- to medium-potency topical corticosteroids
- In cases of secondary infection: topical and even oral antibiotics

**PEARLS AND HIV-RELATED FACTS**

**As many as 60% of black males are estimated to develop pseudofolliculitis barbae; the condition develops mostly between the ages of 14 and 25. As the condition is frequent in general, it is seen in HIV patients, but no further association with HIV has been described.**

# Pustules & Abscesses

## SEVERE ACNE



### CAUSE

Androgen hormones stimulate sebaceous glands to increase sebum production, resulting in oily skin. This can lead to formation of **comedones** (also known as **blackheads**), which can rupture and be superinfected by *Propionibacterium acnes* bacteria. The subsequent inflammation then leads to the full clinical picture.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Typical lesions include comedones, inflammatory papules, and pustules. In more severe cases, there may be nodules and cysts. Acne affects mainly the face, upper chest, and upper back.

Postinflammatory (PI) hyperpigmented dark spots, often lasting for months, are common in dark-skinned individuals; they may be more distressing to the individual than the acne itself. Other sequelae are pitted scarring, and hypertrophic scars and keloids.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Papules and pustules can also suggest folliculitis; if they are itchy and associated with HIV, they may suggest eosinophilic folliculitis.

### WHAT IS THE TREATMENT?

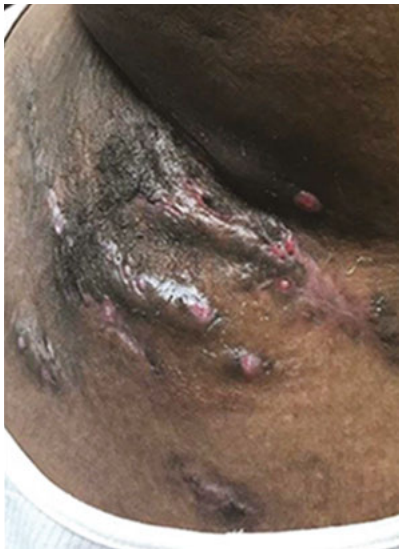
- Stop all use of Vaseline and oily or greasy cosmetics.
- **Comedones**: peeling with benzoyl peroxide 5–10% (apply at night to avoid phototoxicity); an alternative is salicylic acid 10% solution.
- **Pustular lesions**: clindamycin 1% lotion. If severe, doxycycline 100 mg bd until improvement (which may take weeks or even months).
- Sometimes retinoids, such as tretinoin or adapalene, are prescribed (available from private pharmacies).

### PEARLS AND HIV-RELATED FACTS

- **Acne occurs frequently in the 12–24 age group, but many adults are affected even in their thirties.**
- **The characteristics of acne are similar regardless of HIV status. Age is the most important factor; the severity of acne lesions does not appear to be affected much by HIV infection or CD4 count.**

# Pustules & Abscesses

## HIDRADENITIS SUPPURATIVA



### CAUSE

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disease affecting hairs and their associated (androgen-dependent) glands.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

Also referred to as **acne inversa**, hidradenitis suppurativa typically affects the axillae, the inguinal and anogenital regions, and the areas under the breasts. It presents as very painful papules, subcutaneous nodules, and sinus tracts. The lesions may spontaneous discharge serous, purulent, or bloody material. Lesions can progress to form interconnecting sinus tracts or larger abscesses.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Acne, folliculitis, furuncle/carbuncle, granuloma inguinale, lymphogranuloma venereum.

### WHAT IS THE TREATMENT?

**Mild forms**

- Fucidin or mupirocin cream plus benzoyl peroxide washes

**Severe forms**

- Oral antibiotics, e.g., doxycycline 100 mg bd
- Refer to dermatologist or surgeon for excisional surgery.

### PEARLS AND HIV-RELATED FACTS

- **Hidradenitis suppurativa is strongly associated with smoking and high BMI, with 38% of patients reporting a positive family history.**
- **It is thought to be more prevalent in individuals of African descent.**
- **Some authors believe HIV-positive patients are more susceptible to developing HS, and may show involvement of atypical sites, such as the face or thighs.**

## MORBILLIFORM DRUG REACTION

### CAUSE

Immunological reaction to drugs, leading to generalised skin changes that look similar to generalised viral rashes like measles or rubella. The main causative drugs are Bactrim, TB drugs, and ART drugs, but many others may also cause similar skin changes.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

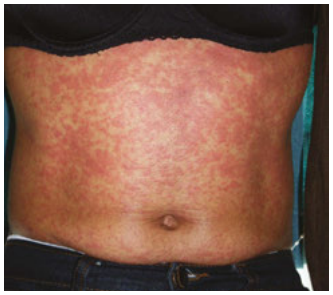
Morbilliform rashes are the most common type of drug-related skin reaction. They are characterised by a maculopapular appearance, often appearing initially on the head, neck, and trunk before spreading to the distal limbs. Pruritus is a common complaint. The mucous membranes are typically not involved.

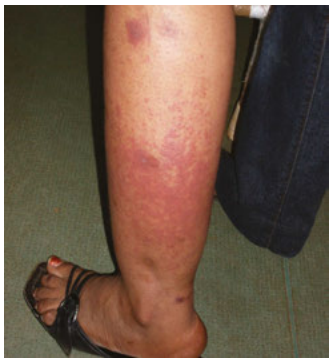
A special case is abacavir (ABC) hypersensitivity, which is fortunately rare in patients of African descent. ABC hypersensitivity reaction is a multiorgan process manifested by at least two of the following:

- Fever
- Rash
- GI symptoms
- Respiratory symptoms

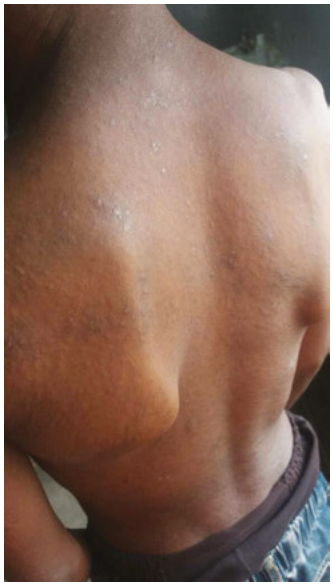
Usually in the first two months of ABC treatment, rash, mild flu-like symptoms, and fever start. Over the following days or weeks, fever and malaise increase; nausea and vomiting or respiratory symptoms may follow. The most important diagnostic clues to ABC hypersensitivity are that: a) the symptoms are most prominent several hours after taking the medication in the morning, and b) they seem to worsen with each day on ABC.

Other drug reactions of the skin are fixed drug eruptions that often present as local target lesions:

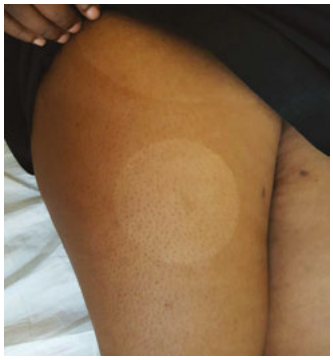


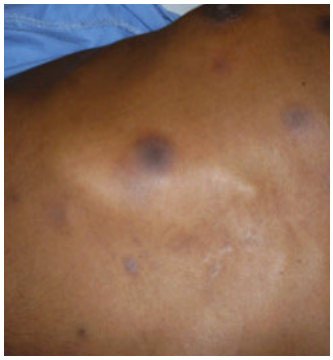


Morbilliform rashes caused by Bactrim.



ABC reaction.





### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

HIV seroconversion rash, other viral diseases (e.g., EBV, measles, rubella).

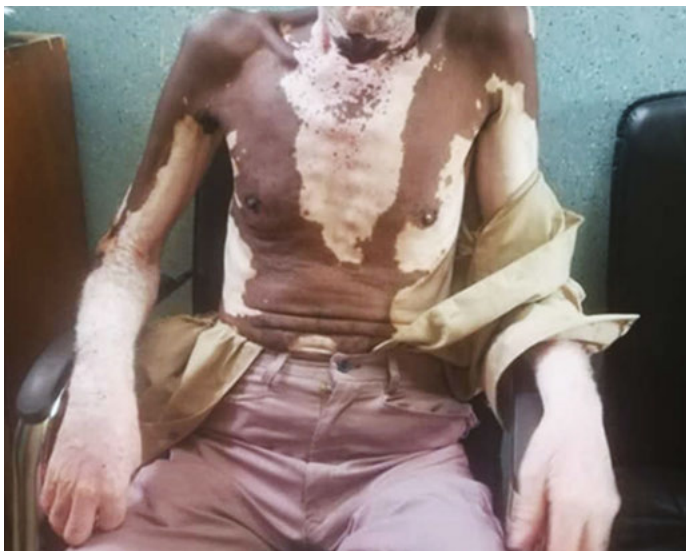
### WHAT IS THE TREATMENT?

- Stopping use of the offending drug treats the cause.
- Antihistamines and steroids may help to curb the symptoms.

### PEARLS AND HIV-RELATED FACTS

- **ABC hypersensitivity is linked to the gene HLA-B*5701. In Caucasians, it occurs in 6–8% of patients; therefore, genetic testing is required before starting ABC. However, studies have confirmed a far lower risk of ABC hypersensitivity reactions among Africans; it is therefore possible to start our patients on ABC without genetic testing. However, clinicians must still be vigilant for suggestive symptoms, and should always check for signs of hypersensitivity.**
- **HIV seroconversion rash is probably more frequent than we think. Because the symptoms are generally mild, patients do not seek medical care. The generalised morbilliform rash is associated with headache, sore throat, fever, and painful lymph node swellings. It resolves spontaneously after one week, with the HIV rapid test turning positive a few weeks later. By that time, HIV DNA may already be positive.**

## VITILIGO

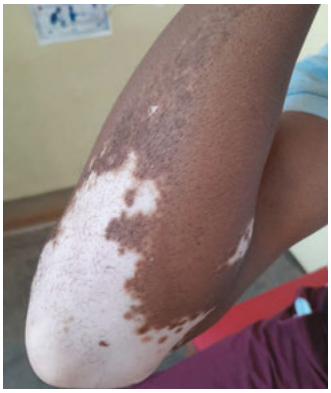


### CAUSE

Vitiligo is a chronic condition characterised by skin depigmentation due to an autoimmune reaction that destroys pigmented melanocytes.

### HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?

White or depigmented patches on the skin are the major clinical finding; they usually start small and then grow. The patches frequently occur in areas regularly exposed to sunlight, such as the backs of the hands and fingers, as well as the areas around the eyes, nose, and mouth. Other areas affected include the armpits, navel, and genitals. Half of vitiligo cases start before the age of 20, with the vast majority (95%) starting before the age of 40; there is no gender predilection. The course of the condition is highly unpredictable; depigmented patches may resolve, spread progressively, or remain stable and persist indefinitely.

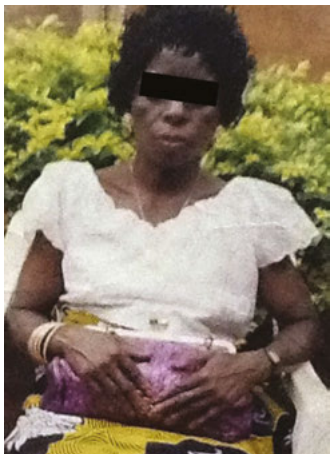


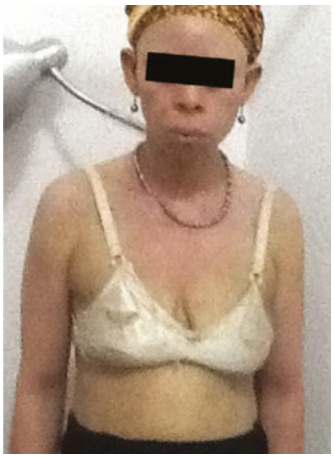
### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Tinea versicolor, PI hypopigmentation.

### WHAT IS THE TREATMENT?

- There is no satisfactory treatment; instead, reassure that patient that the condition is only a change in skin colour, not an indication of further disease. Psychosocial counselling may be needed, as the skin changes can cause significant emotional stress.
- Sometimes a combination of topical steroids and sun exposure may lead to repigmentation—but the risk of sunburn and sunlight-induced skin cancer must be considered.





Patient before and after starting ART, developing vitiligo as a manifestation of IRIS.

### PEARLS AND HIV-RELATED FACTS

- **Vitiligo has been reported in HIV patients; here it is believed to be an autoimmune disease triggered by viral infection (possibly through polyclonal B-cell activation against melanocytes, and changes in the balance between helper and suppressor T cells).**
- **In some patients, vitiligo starts within three months after initiation of ART, suggesting an IRIS-like immune dysregulation. Cases of generalised depigmentation have also been seen.**
- **Be aware that HIV-associated skin diseases (e.g., Kaposi sarcoma) look different on depigmented skin. In these cases, biopsy may be needed to confirm diagnosis.**

## POSTINFLAMMATORY HYPO- AND HYPERPIGMENTATION

### CAUSE

Melanocytes can react with increased, normal, or decreased melanin production in response to cutaneous inflammation or trauma.

### HOW DO THEY PRESENT? HOW ARE THEY DIAGNOSED?

**Postinflammatory (PI) hypopigmentation** is characterised by patches of colour, ranging from light brown to white.

**PI hyperpigmentation** commonly appears on the skin as darker brown to grey macules. The changes appear in areas of prior inflammatory disease, infection, or allergic and irritant reactions; the borders are often hazy.

PI hypo- and hyperpigmentation can be emotionally distressing for patients, affecting both their personal and professional lives.

Minimal hypopigmentation may resolve within a few weeks, but severe cases may be permanent or take years for pigmentation to return.

Excess epidermal pigment may take 6–12 months to fade.

### WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?

Inflammatory diseases of the skin.

### WHAT IS THE TREATMENT?

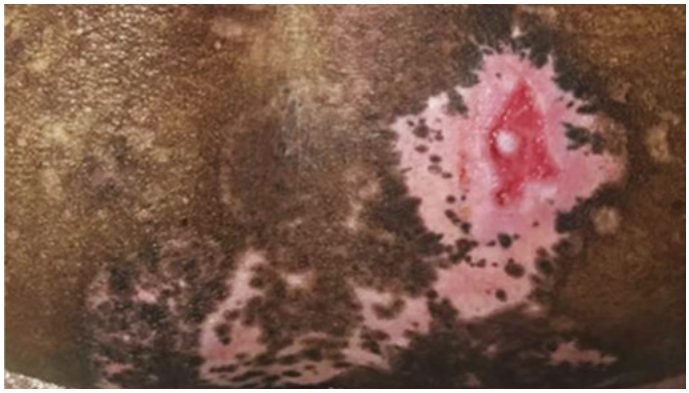
Underlying untreated inflammatory conditions could result in new areas of PI hypo- or hyperpigmentation; these should therefore be treated first.

**Hyperpigmentation**

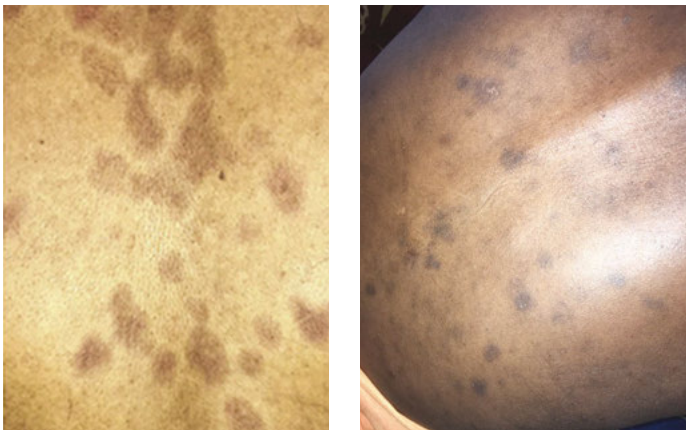
Topical hydroquinone has been used for treating hyperpigmentation. A triple combination cream—containing hydroquinone 4%, a steroid (fluocinolone acetonide 0.01%), and a form of vitamin A (tretinoin 0.05%)—is one possible treatment for hyperpigmentation. However, it should NOT be used for more than 4–8 weeks.

**Hypopigmentation**

Topical steroids combined with sun exposure may help to accelerate repigmentation. Treatment should be limited to 4–8 weeks at most.



PI hypopigmentation.



PI hyperpigmentation.

### PEARLS AND HIV-RELATED FACTS

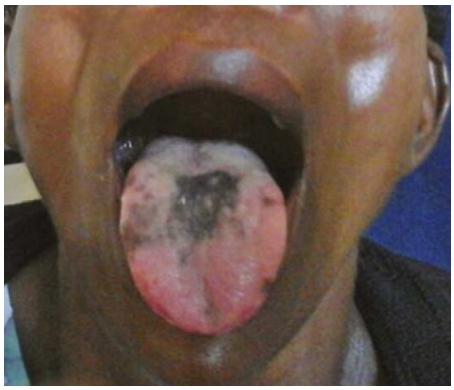
- **HIV infection is associated with both hyper- and hypopigmentation. Nail and skin hyperpigmentation has been reported in patients infected with HIV, and in patients treated with AZT.**
- **PI hyperpigmentation is a major concern in patients who have facial involvement of PPE, eosinophilic folliculitis, and papular urticaria.**

## DISCOLOURATION (MELANONYCHIA)

**CAUSE**
Dark nail discolouration can have multiple causes, including HIV and drug treatment (AZT).

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Darkening of the nail.

AZT-induced discolouration may not only affect the nails, but can also be seen on the mucosa or the skin.

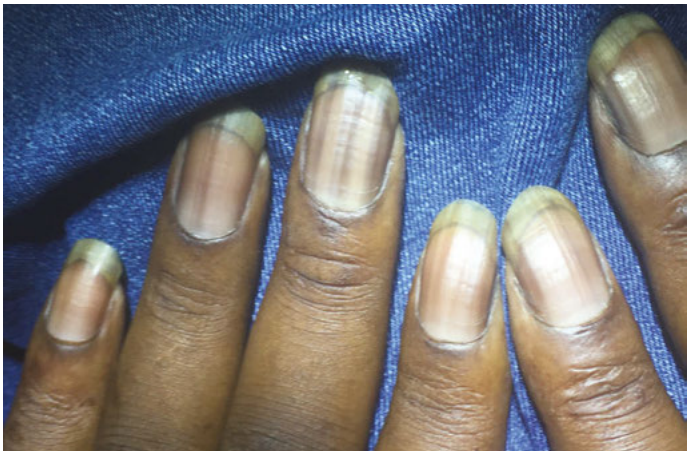


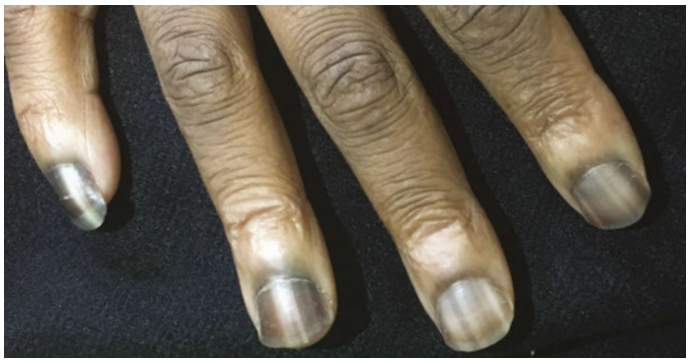
Discolouration of the tongue caused by AZT.

**WHAT IS THE TREATMENT?**
There is no specific treatment.

**PEARLS AND HIV-RELATED FACTS**

- **Nail discolouration is common among the general population, but when several nails are affected, an association with HIV is likely.**
- **Drugs associated with nail discolouration include AZT, fluconazole, antimalarial therapy, cancer chemotherapy, clofazimine, and tetracycline (and other drugs rarely used in our setting).**





Discoloration of the hands due to AZT. The patient's sister's hands are shown for comparison.

## ONYCHOMYCOSIS

**CAUSE**
Fungal infection of the nail

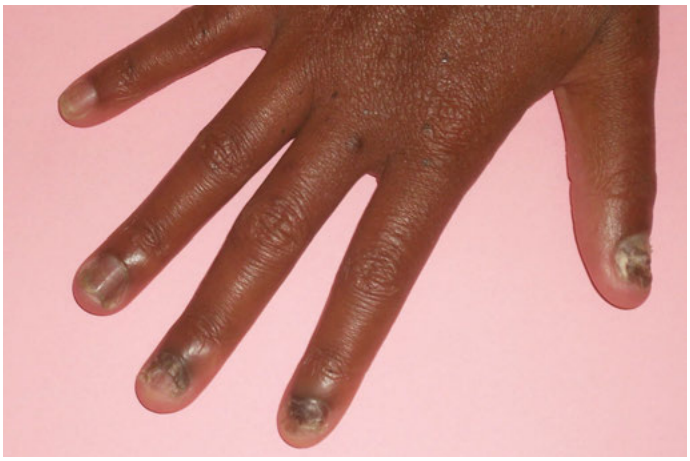
**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Discolouration, thickening, crumbling material, and deformity of the nail are typical signs of fungal infection.

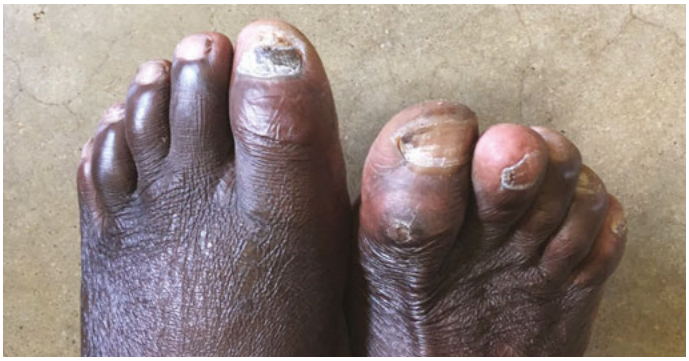
**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Psoriasis and eczematous conditions are often clinically indistinguishable from fungal nail infection; therefore, the rest of the skin should be checked for lesions. Onychomycosis can also be caused by trauma and iron deficiency; ideally, a nail sample should be submitted for KOH microscopy.

**WHAT IS THE TREATMENT?**
Fluconazole, e.g., 200 mg once per week for 8–12 weeks.

**PEARLS AND HIV-RELATED FACTS**

- **The prevalence of fungal nail infection increases with age, possibly due to vascular insufficiency, trauma, suboptimal immune function, or insufficient foot care.**
- **Onychomycosis is more prevalent among HIV patients and other persons suffering from immunodeficiency.**





## HAIR STRAIGHTENING

**CAUSE**
With uncontrolled HIV infection, the hair's natural curly architecture is lost, causing the hair to straighten. This may be due to caloric and protein malnutrition, as well as to deficiencies in minerals (such as copper, zinc, and selenium) that affect hair growth.

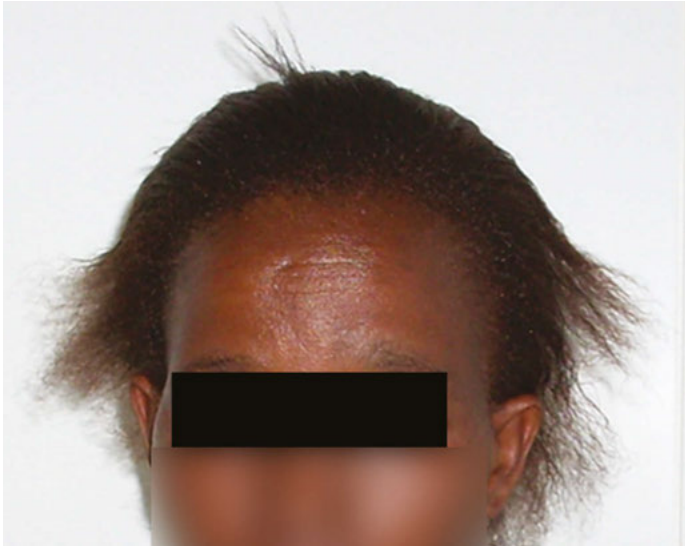
**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Hair becomes less curled, and thus longer, lighter, softer, and often brittle. Discolouration is occasionally seen.

**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Other causes, such as malnutrition, cachexia, and vitamin and mineral deficiency.

**WHAT IS THE TREATMENT?**
Treat the underlying condition.

**PEARLS AND HIV-RELATED FACTS**

**Hair straightening was observed in as many as 50% of HIV-positive black patients during the early days of the HIV pandemic; it was referred to as the 'straight hair sign'. It was later found in other patient groups also.**



Example of hair straightening.

## HAIR LOSS AND ALOPECIA

**CAUSE**
Many acute or chronic illnesses can lead to hair loss, but it can also be caused by surgical operations, accidents, and childbirth.

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
It presents as increased shedding of hair and thinning of hair on the scalp. Itching and other symptoms are generally absent unless the hair loss is caused by an accompanying inflammatory skin disease.

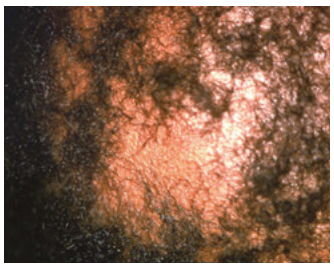
**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
**Alopecia areata** is an autoimmune condition in which hair follicles fall out in a localised area, resulting in the appearance of bald spots. This condition is more prevalent in patients with HIV.

**Traction alopecia** is the result of excessive pulling of the hair during braiding and styling.

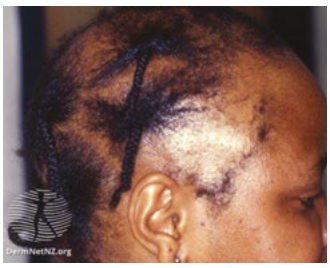
**WHAT IS THE TREATMENT?**
Treat the underlying conditions.

**PEARLS AND HIV-RELATED FACTS**

- **HIV is not strongly associated with hair loss. Although hair loss may occur in people with HIV, it is usually either the result of the natural ageing process (male and female pattern baldness), or a side effect of co-occurring conditions.**
- **Hair loss is described as a side effect of AZT.**



Alopecia due to syphilis.



Traction alopecia.

Alopecia areata.

# Other Conditions Oral

## KAPOSI SARCOMA

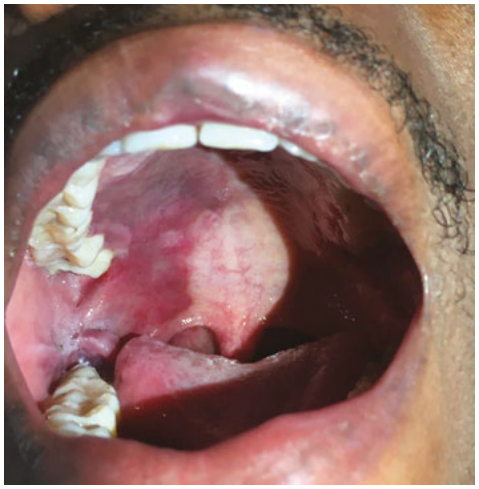
**CAUSE**
Kaposi sarcoma herpesvirus (KSHV), also called human herpesvirus 8 (HHV-8)

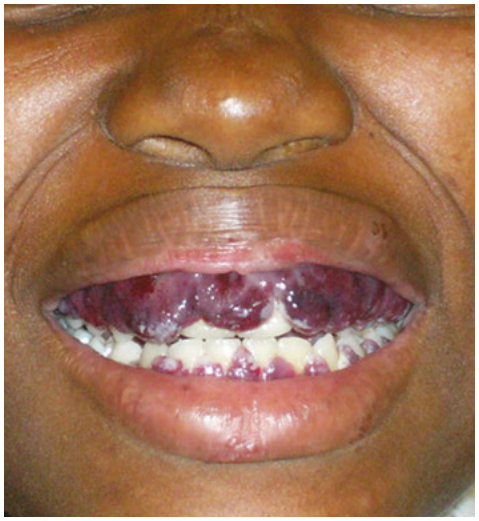
**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Oral KS lesions usually occur on the hard palate, but may be found on any part of the mucosa of the mouth (tongue, buccal pockets, gingiva, uvula). Initially, they are asymptomatic, but can bleed, cause difficulty in swallowing and/or breathing.

**WHAT IS THE TREATMENT?**
Refer to the national guidelines for treatment of KS.

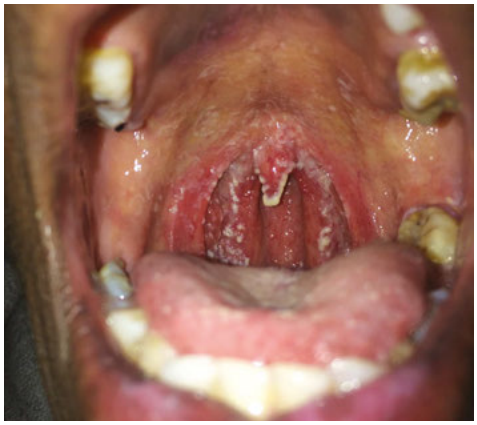
**PEARLS AND HIV-RELATED FACTS**

**Oral KS lesions are often a sign of GI involvement and other systemic KS disease. They can be considered as the visible tip of the KS iceberg.**



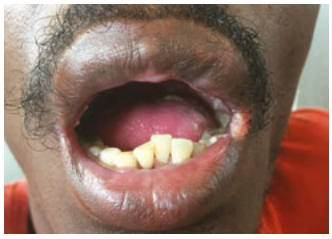


## CANDIDIASIS

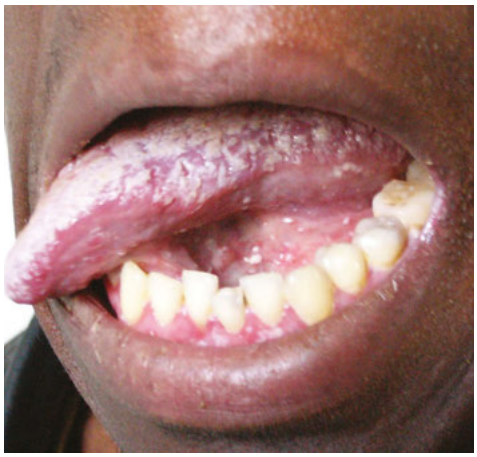


**CAUSE**
Fungal infection of the oral mucosa by *Candida albicans*

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Oral candidiasis most often presents with creamy white plaques that resemble cream cheese (thrush). These pseudomembranes can vary in size, from a few millimetres to large enough to cover the entire mouth or tongue. Sometimes, only red maculae are seen on the tongue or the palate; these are easily missed. Oral lesions usually do not cause many symptoms, but burning sensation, pain, and changes in taste have been reported. Painful swallowing (odynophagia) is the most important symptom to ask about, as it suggests extension of the disease into the oesophagus.



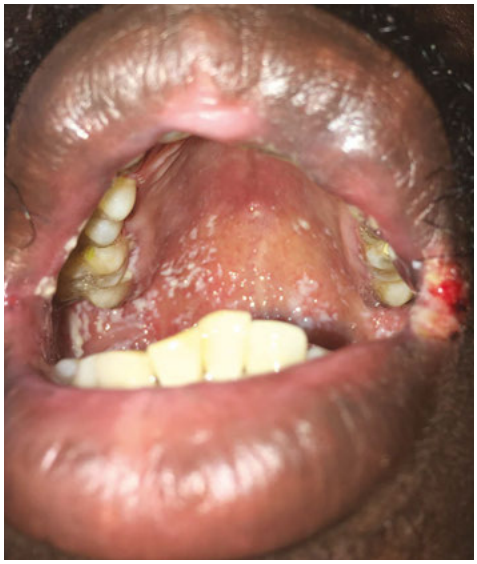
A rarer form, **angular cheilitis**, is characterised by linear ulcers and cracks at the angles of the mouth, with crusting. These may be painful or burning. It can also be caused by iron or vitamin $B_{12}$ deficiency.



**WHAT OTHER CONDITIONS SHOULD BE CONSIDERED?**
Usually, the typical symptoms are definitive; further tests are rarely done.

**WHAT IS THE TREATMENT?**

- Nystatin suspension 4 mL every 6 hours for 7–14 days. Keep in the mouth as long as possible.
- If the condition is severe, there is no response to nystatin, or the oesophagus is involved, an alternative regimen is fluconazole 200 mg every 24 hours for 14 days.

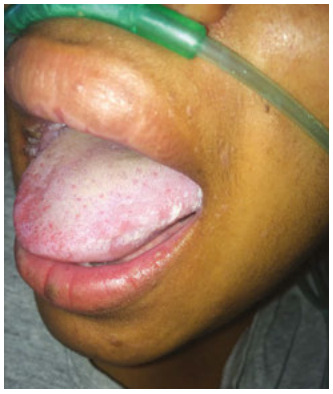


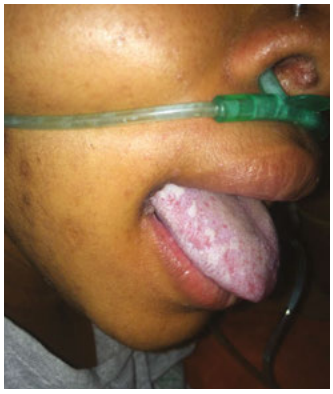
**PEARLS AND HIV-RELATED FACTS**

**The incidence and extent of oral and oesophageal candidiasis increases as CD4 count falls. Oral candidiasis is seen in 20–40% of patients with AIDS.**

# Other Conditions Oral

## HAIRY LEUKOPLAKIA





**CAUSE**
Epstein-Barr virus (EBV) infection of the tongue epithelium

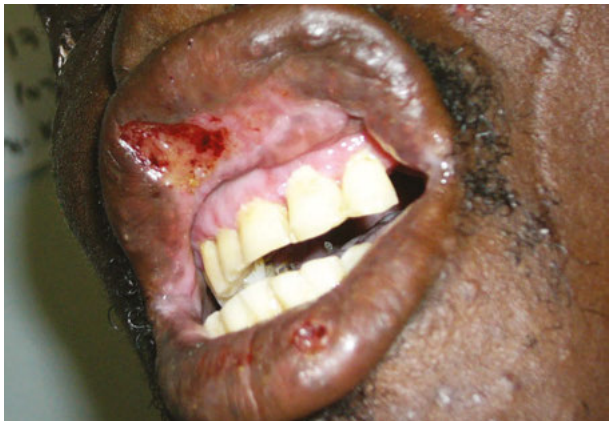
**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Oral hairy leukoplakia presents as white plaques on the lateral tongue that do not wipe off. The plaques may be unilateral or bilateral, and are usually asymptomatic.

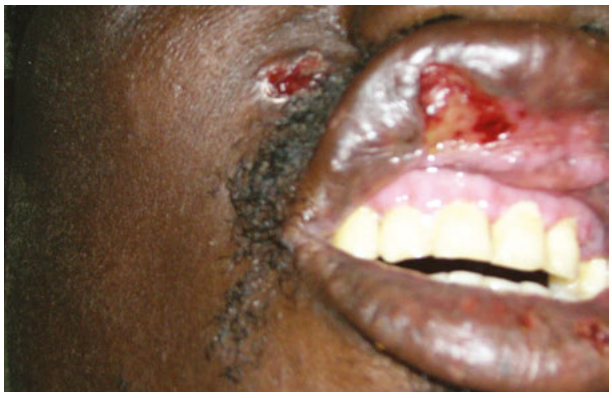
**WHAT IS THE TREATMENT?**
Treat the HIV infection with ART.

**PEARLS AND HIV-RELATED FACTS**

**Oral hairy leukoplakia itself is usually not treated; it may resolve spontaneously when CD4 improves. It does not progress to oral cancer.**

## APHTHOUS ULCER





**CAUSE**
Unclear

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Painful ulcers, often recurrent, develop in the mucosa of the mouth (tongue, lips). They have a whitish base, surrounded by a red, hyperaemic area. They are mostly smaller than 1 cm, but in some cases can be larger. They can also appear in a herpetiform arrangement (multiple ulcers grouped together).

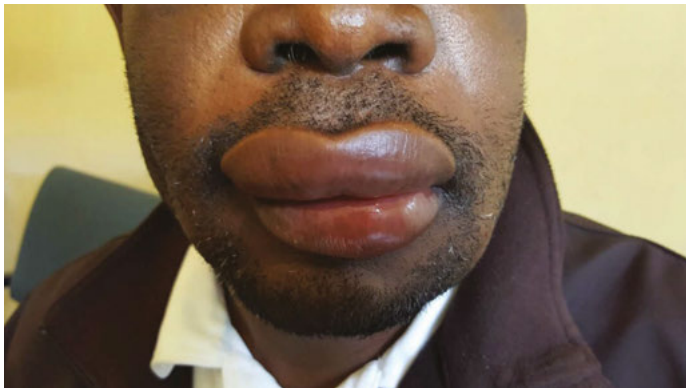
**WHAT IS THE TREATMENT?**
Because it is a likely differential diagnosis, the first treatment would be for herpes simplex: acyclovir 400 mg 5x/day for 7–10 days. Recurrent aphthous ulcers are more difficult to treat.

- Antiseptic mouthwash (chlorhexidine 0.2% or polyvidone-iodine 1%)
- Local steroids (cream or inhaler)

**PEARLS AND HIV-RELATED FACTS**

**It is recommended to first treat with acyclovir to rule out HSV infection, as this can be exacerbated by steroid use.**

## ALLERGIC OEDEMA/ANGIO-OEDEMA OF THE LIPS



**CAUSE**
Allergy or reaction to offending drugs, most often seen with ACE inhibitors (e.g., enalapril).

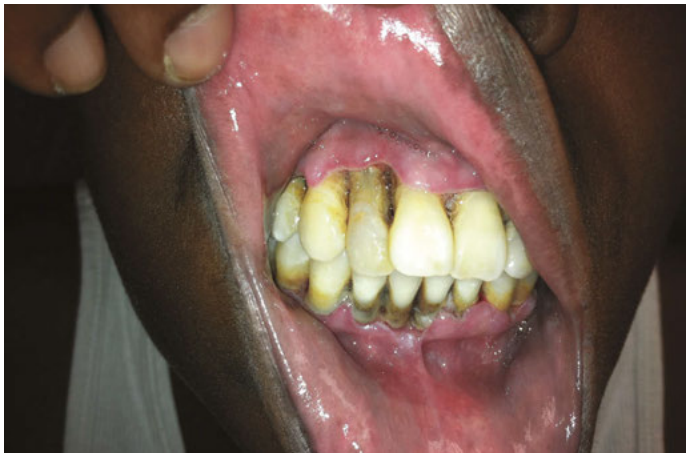
**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Acute oedematous swelling of the lip (weal). In severe cases, problems with swallowing and breathing can occur.

**WHAT IS THE TREATMENT?**
- Stop use of the offending drug—check for enalapril or other ACE inhibitors!
- Anti-allergic treatment using steroids (e.g., prednisolone 30 mg od for 3 days) and antihistamines (cetirizine....)

**PEARLS AND HIV-RELATED FACTS**

**Allergic oedema occurs when contact with a specific allergen or closely related chemical substance elicits an immunologic inflammatory response, usually between 24 and 72 hours after exposure.**

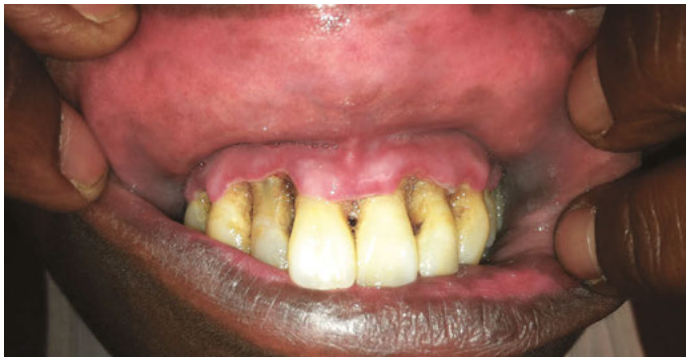
## NECROTISING ULCERATIVIE GINGIVITIS



**CAUSE**
Multibacterial infection due to poor oral hygiene and immune suppression

**HOW DOES IT PRESENT? HOW IS IT DIAGNOSED?**
Necrotising ulcerative gingivitis (NUG) presents as rapid onset of gingival necrosis between the teeth, gingival pain, bleeding, and bad breath.

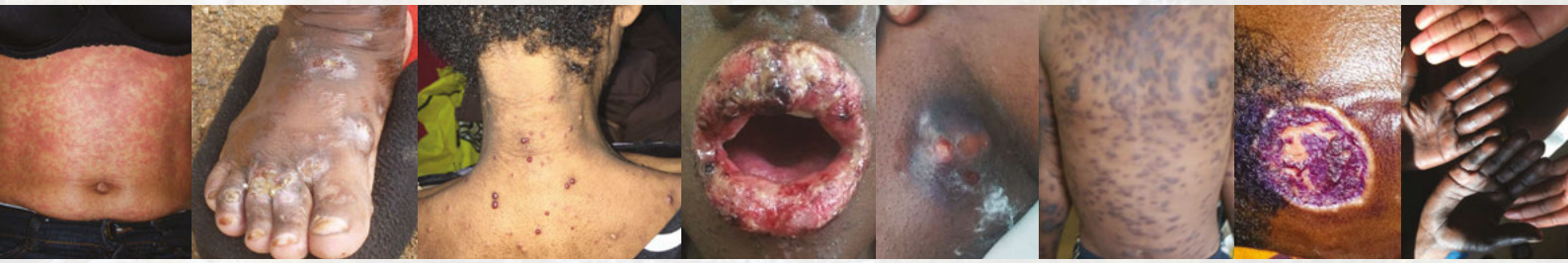
**WHAT IS THE TREATMENT?**
- Metronidazole 250 mg tds for 5–10 days. Amoxy-clav or clindamycin may be used as an alternative.
- Refer the patient to a dentist for possible debridement and local peroxide application.



**PEARLS AND HIV-RELATED FACTS**

**NUG was originally seen among military personnel during World War I, presumably caused by multiple risk factors, including poor oral hygiene, intense psychological stress, and malnutrition.**

With this manual, we want to help our colleagues to recognise and treat some of the most frequently encountered skin conditions. Complicated cases will still need to be referred to dermatology departments and specialists, but for many diseases a diagnosis can be made, and treatment attempted, at the first point of care. We hope this manual proves helpful for that.



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