

# Tropical Skin Conditions - a short overview

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Many of the skin conditions met in medical practice in the tropics are related to poverty, malnutrition, poor housing, water and soap shortage. In high income countries, of the over 1000 named skin conditions, 70% are in 9 conditions or groups: **acne, bacteriological, viral, fungal infections, tumours, dermatitis, psoriasis, leg ulcers and warts**. This list is not representative for low-income-countries. The focus in this lecture is not on the conditions readily recognised from medical practice in Sweden but those that are common and should be recognised in Africa, some of which are never seen in Sweden.

The lack of diagnostic and therapeutic resources and lack of hygiene make the situation worse. Many are due to infectious agents that are more common in warmer climates and others are due to strictly tropical diseases that are not seen outside of the tropics.

## 1. What do you see and feel?

**Which layer of the skin is affected? Symmetry? colour? Nodules, rashes, eruptions, distribution:** 2<sup>0</sup> syphilis: symmetrical, eczema: flexures, psoriasis: extensors. Distribution and colour of pellagra: skin exposed to sunshine, Eschars: often on lower legs where ticks bite.

**Two large groups of skin conditions are seen:**

A. **Ulcers:** here important conditions worth identifying are the following: **Tropical ulcers** (by far the commonest in many poor communities); **trophic ulcers** due to leprosy; tuberculous ulcers (not common); **Buruli ulcers** (rare except for certain areas e.g. in Uganda); **cutaneous Leishmaniasis** (seen in parts of East Africa); Tertiary syphilis (gumma) now rare; sickle cell disease in adults (used to be rare since so few survived to adulthood but now less rare); dracontiasis - Guinea worm (now almost gone); Edges of **syphilitic chancres** vs chancroid.

## 2. What do patients feel?

**B. Itching:** The most common are **Scabies, Infestations from insects** such as mosquitoes, fleas, lice including bedbugs (vägglöss), **Tumbu fly** etc., **creeping eruption** (*Ancylostoma braziliensis* or *A. caninum*), Swimmers' itch (*Schistosomiasis*), Eczema including pompholyx, Contact dermatitis, onchocerciasis, fungi (tinea etc.), urticaria, drug eruptions, HIV related itch probably due to *Pitosporem orbiculare*.

**C. Pain:** Herpes zoster, Tropical ulcers, **Tunga penetrans, chancroid, herpes genitalis, cutaneous myiasis,**

**D. Loss of sensation, Pain:** Leprosy, Diabetes. Lack of pain in syphilitic chancres, tertiary syphilis. **Buruli Ulcer**

## Causative descriptions

### Viral causes

#### 1. Measles,

The incubation period is 10-14 days (median 11 days) and the child is contagious from around 4 days prior to the rash appearing until 4 days after the rash disappears.

A generalized rash and fever with one of the following: cough, runny nose or red eyes. When measles occurs linked to HIV the symptoms may be hidden or less apparent.

The child usually has 3-4 days of fever, coryza, cough, malaise, conjunctivitis. Then **Koplik's spots**

appear on the **3rd. or 4th. day of the illness**: these look like tiny grey-white grains of sand on a red background on the posterior buccal mucosa.

**The rash usually appears on the 14th day after exposure to the virus** when the fever is high (39° or more). The rash usually starts **behind the ears and the hairline**, on the forehead, then to the face, trunk, arms, buttocks, and reaches the legs after 3 days. **the rash lasts on average for 4 days** and then starts to fade as the temperature goes down with wide-spread desquamation. By the time the rash is generalized, the child has a raw red mouth. The younger the child the longer it is infective for others.

## 2. Dengue

The illness shows as fever with severe headache, retro-orbital pain and intense myalgia. A **blanching macropapular rash appears after a few days** especially in infants and young children and the fever and illness, when uncomplicated, lasts for 4-7 days usually. Platelet counts are low.

## 3. HIV

### Skin changes

The commonest of all HIV-related skin conditions is **herpes zoster** and in Africa a single episode gives high predictive value (over 90% have HIV). The herpes may be very extensive involving several dermatomes, may even be bilateral, and may be aggressive locally and leave considerable scarring. Another very common skin problem is a **generalized pruritic reaction**. There is Folliculitis with severe itching probably caused by *Pitosporum orbiculare*. Difficult to cure but get some relief with calomine lotion. In many African HIV+ patients, this is often the first sign of HIV infection and in certain high-risk groups is almost specific for HIV. Papules, scratch marks and hyperpigmented macules are symmetrically distributed over the body especially over the extensor arm surfaces, back of hands, ankles and dorsum of feet. They last throughout the illness. **Kaposi sarcoma** is somewhat less likely in Africa than in North America and Europe but is seen quite often. A single lesion of **molluscum contagiosum** in an adult is almost diagnostic for HIV. Genital herpes simplex may spread and be very aggressive around the perineum and anus. Very florid **seborrheic dermatitis** and **aggressive psoriasis** are also very suggestive of HIV. Marked **skin reactions to pharmaceuticals** are more common especially to sulfa drugs and to thiacetazone (an old TB drug which is now hardly used because of this side-effect in HIV+ patients).

## 4. Herpes genitalis

The first episode causes painful small vesicles in and around the vagina, cervix, penis, anus and on the inner thighs. There is dysuria, pain at intercourse, vaginal discharge, headache, backache, fever and nausea. 70% have no symptoms. During the recurrent flare-ups the vesicles are often smaller and fewer with milder generalized symptoms. Around 70% of the first episode are caused by HSV-2 and the rest HSV-1 but the vast majority of chronic recurrent episodes are caused by HSV-2. If there is recurrence with HSV-1 this occurs in the first year only. Previous oral HSV-1 gives protection against the genital form of HSV-1 but not against HSV-2 although it reduces the symptoms and frequency of recurrences. It has now become the leading cause of genital ulcer disease and in sub-Saharan Africa 30-80% of women and 10-50% of men are infected.

## Bacterial causes

**1. Pyogenic organisms.** In impetigo the common organisms are the same as in Sweden, staphylococci and streptococci. However they are often more florid and can more often develop cellulitis, folliculitis, furuncles and carbuncles to become life-threatening septicaemias when no treatment is available. This is due to unhygienic conditions, malnutrition and various immune

suppressing conditions especially malaria, measles and especially HIV / AIDS.

Two special conditions are worth noting:

**2. Tropical ulcer:** this condition sometimes comes in epidemic form especially where there is an acute shortage of soap. It is often called the “**f**” condition linked to **f**usiformis bacteria (Vincent’s organisms are fusiformis and a spirochaete as well as streptococci), **f**riiction (any local trauma), **f**lies, **f**ilth. More than 90% are below the knee. It starts as a papule or blister and then breaks down to form an ulcer which is painful and rapidly extends over the next few weeks. The edges of the ulcer are typically slightly raised swollen, red and tender and not undermined. The floor of the ulcer which penetrates the superficial fascia is composed of granulation tissue and is covered with pus. There may be only one or several ulcers but these are not grouped together. After a few weeks the inflammation subsides and the ulcer becomes static with a diameter of 1-10 cms. Treatment is with penicillin and, after cleaning with copious water, local antiseptics including **honey under a paraffin gauze dressing**. Once the infection is under control dress with a non-adherent dressing and leave if possible without disturbance for 10-14 days. If necessary surgery, including finally a skin graft, in the large ones, may be required if the ulcer invades deeper layers. This condition was common in the prisoner of war camps in Burma and the tropical Far East.

**3. Pyomyositis:** a muscle infection usually due to staphylococci causing widespread necrosis and often a massive collection of pus that finally erupts onto the surface. It becomes even more extensive in HIV infection

**4. Treponemal infections.** Here **syphilis** dominates in many regions with the classical picture of secondary syphilis. 1 - 6 months after the primary infection the secondary stage often begins with fever, malaise a symmetrical rash over the whole body without any itching. This is a maculo-papular rash which is typically seen on the palms of the hands or the soles of the feet and over the trunk. In the perineum this rash can develop into flat warts (Condylomata lata) (highly infectious). There can also be snail-like ulcers in the mouth or on the tongue. There are often enlarged regional lymph nodes, sore-throat or loss of hair etc. Penicillin usually given as benzathine penicillin is highly effective in earlier stages.

**5. Yaws** is a less common treponemal infection (caused by *treponema pallidum pertenue*) manifesting on the feet and perioral skin of the face. It affects mainly the male rural population in South America, sub-Saharan Africa and South-East Asia. It is associated with a humid tropical climate and characteristically shows with plantar keratosis. Late tertiary infection results in asymptomatic palmoplantar keratoderma that develops nodular hyperkeratotic lesions leading to painful disability, with a classical walk known as “crab yaws”. Penicillin usually given as benzathine penicillin is highly effective in the early stages but later tissue damage can be so great with face deformations that penicillin has only a limited effect. Huge campaigns with sulfa injections in the early days of antibacterial treatment were highly effective in reducing the prevalence in many parts of the tropics.

**6. Pinta** is another treponemal infection reported mainly from the Philippines, Mexico and Colombia usually in children and young adults. It usually show with papular eruptions or more commonly with progressive dyschromic patches and plantar keratoderma. Hypo- and hyperpigmented skin patches affect the limbs, trunk neck and face and may show as frank leucoderma. Penicillin is the treatment of choice as Benzathine penicillin in a single dose.

**7. Endemic syphilis (Bejel)** Uncommon: usually shows as painless ulcers of lips and oropharynx. Later there may be osteoperiostitis, condylomata lata, angular stomatitis and rarely a

rash similar to 2<sup>0</sup> syphilis. Treatment: Benzathine penicillin as a single dose.

## 8. Typhoid Fever

### Symptoms and Signs

This presents as a fever which after a week leaves the patient looking sick, there is no response to anti-malarials and there is no evidence of urinary tract infection, pneumonia or ear or throat infections. You must think of typhoid fever as a possibility.

The symptoms that make you suspicious are a **fever that has come on slowly in a step-wise manner** up to 39-40<sup>0</sup> without rigors but after one week with headache and often abdominal pain.

There may appear in people with pale skin at the end of one week's illness **rose spots (pink sparse maculopapular spots on the chest or abdominal wall that fade on pressure with a glass slide).**

**Cough and chest signs** with a normal Xray make you even more suspicious.

**Relative bradycardia** (not corresponding to the fever) and splenomegaly and hepatomegaly are seen. In the third week the patient looks more toxic with **mental dullness or a delirious confusional state**. Early constipation in the first week followed by loose stools (pea soup stools) in 2nd or 3rd week is a classical story but not seen in so many. The most commonly affected group are young adults and older children.

## 9. Mycobacterial infections

**Mycobacterium marinum** or fish tank granuloma is usually difficult to diagnose by someone who has never seen it before and if the history of exposure to a fish tank is not forthcoming. It usually affects the fingers and dorsum of the hand with locally progressing swelling with variable pain and within a few weeks nodular or verrucous skin lesions which may ulcerate and bleed when traumatized. Treatment is with doxycycline or minocycline or rifampicin + ethambutol.

**Mycobacterium leprae** is the most widespread of all the skin conditions with a mycobacterial cause but is less common after multidrug therapy was introduced in 1983.

The symptoms and signs depend on the body's immune response to bacilli. The range is from tuberculoid to lepromatous.

<b>Tuberculoid</b>		<b>Borderline</b>		<b>Lepromatous</b>
<b>TT</b>	<b>BT</b>	<b>BB</b>	<b>BL</b>	<b>LL</b>

TT Good immune response. Lepromin test strongly positive. Few lesions. Dense nerve damage early. Very few bacilli. No public health danger.

LL Poor or absent immune response. Lepromin test negative. Many lesions. Weak but progressively more serious nerve damage late. Many bacilli. Can shed 10<sup>8</sup> bacilli in 24 hours. Untreated cases can spread disease.

The very earliest sign in indeterminate leprosy is a skin patch that has lost its ability to sweat or nodules, erythematous plaques or hypopigmented patches. Other later signs are:

1. Asymmetrical anaesthetic skin lesions without hair in TT.

Small symmetrical numerous infiltrates with normal sensation in the early stages in LL.

2. Enlarged often tender nerves, especially the cooler parts of the nerves where they are near the surface e.g. superficial radial nerve at wrist (most lateral point at wrist), ulnar nerve at the elbow, median nerve at the wrist (under medialis tendon), great auricular nerve behind the ear, the lateral popliteal (or common peroneal) nerve near the knee (lateral aspect around neck of fibula), and the posterior tibial nerve just behind the medial malleolus.

3. Search for the bacilli from the lobe of the ear, scrapings from the nose and the edge of a skin lesion. Never found in paucibacillary leprosy.

Treatment is with multidrug therapy with rifampicin plus dapsone for 6 months in paucibacillary (2 - 5 lesions only) and rifampicin + clofazimine + dapsone in multibacillary (more than 5 lesions) leprosy for 12 months.

**Mycobacterium tuberculosis** has many different skin manifestations which frequently affect the lower limbs and particularly one or both feet. Lupus vulgaris and papulo-necrotic tuberculide are more common in females. Tuberculosis verrucosa cutis is the commonest TB skin manifestation and affects the adult foot. Lupus vulgaris is often on the face. Dry patches of atrophic skin, pigmentary changes, nodules and plaques of verrucous lesions are commonly seen. Diagnosis is by biopsy, bacteriology and PCR investigations. Treatment is with multi-drug therapy usually with rifampicin, pyrazinamide and isoniazid.

**Mycobacterium ulcerans (Buruli ulcer)** is the third commonest mycobacterial skin disease and is a highly destructive ulcerating condition. It is seen especially in Uganda, West Africa and neighbouring countries. It affects mainly the limbs and starts as a firm painless nodule which may be itchy. The overlying skin breaks down and an ulcer forms and often spreads in all directions. It is painless with undermined edges and satellite ulcers form with a subcutaneous tunnel communicating with the original ulcer. Scraping of the ulcer base show acid-fast bacilli. Widespread excision and skin grafting is the best treatment with rifampicin and clarithromycin treatment before, during and after surgery. Heating the ulcer to 40<sup>0</sup> is a useful adjunct. BCG gives a short-lived protection.

### 3. Actinomycoses

Here a combination of Nocardia, Actinomadura and Streptomyces may cause Madura foot or actinomycetoma. This form of mycetoma occurs in tropical countries with high prevalence in Sudan, Senegal, Nigeria, Sindh and Mexico. It is caused by direct inoculation of the bacteria into the skin often during agricultural workers without shoes. The bacteria are capable of blocking the killing mechanisms by the cells of the infected host. There is a chronic course with inflammation, formation of sinus tracks discharging "grains" and progressive deformity of the foot. The chronic infection often heals for a while and then breaks down and sinuses are formed. There is often periosteal involvement and osteomyelitis. Effective drugs are streptomycin, dapsone and cotrimoxazole but more recently a combination of cefotaxime, amikacin and immunomodulators has shown promise. However treatment has to be for many months and even then the final results are doubtful. When osteomyelitis is shown amputation of the foot may be the best answer.

## Rickettsial skin conditions

**General summary of rickettsial diseases:** Clinical presentations vary with the causative agent and patient; however, common symptoms that typically develop within 1–2 weeks of infection include fever, headache, malaise, and sometimes nausea and vomiting. Most symptoms associated with acute rickettsial infections are nonspecific. Many rickettsioses are accompanied by a maculopapular, vesicular, or petechial rash or an eschar at the site of the tick bite. **African tick-bite fever should be suspected in a patient who presents with fever, headache, myalgia, and an eschar (tache noir) after recent travel to southern Africa.** Mediterranean spotted fever should be suspected in patients with rash and fever after recent travel to northern Africa or the Mediterranean littoral. Scrub typhus should be suspected in patients with a fever, headache, and myalgia after

recent travel to Asia; eschar, lymphadenopathy, cough, hearing difficulties, and encephalitis may also be present. Patients with typhus usually present with a severe but nonspecific febrile illness. Most symptomatic rickettsial diseases cause moderate illness, but epidemic typhus and Rocky Mountain spotted fever can be severe and may be fatal in 20%–60% of untreated cases. Treatment of patients with possible rickettsioses should be started early and should not await confirmatory testing. **Treatment** usually involves doxycycline or chloramphenicol; azithromycin, fluoroquinolones, and rifampin may be alternatives, depending on the scenario. Expert advice should be sought if these alternative agents are being considered.

**Rickettsia africae** is one of the most common of these conditions due to tick bites and results in African tick bite fever with inoculation eschars, regional lymphadenitis, myalgia and severe headaches. Half will show a maculopapular rash without itching and some may even show multiple eschars (not seen in other tick bites).

## Fungal skin conditions

Here the dominant conditions are Tinea capitis, Tinea corporis, Tinea cruris, Tinea pedis and other dermatophyte infections. Most are easily treated with topical clotrimazole or miconazole but Tinea capitis often needs shaving first and if extensive may need oral anti fungal therapy (if available give terbinafine 250 mg daily. Likewise nails need oral therapy as well as topical treatment with terbinafine.) If itching or inflammation is intensive can use topical steroids once effective antifungals are in place.

**Chromomycosis:** A special problem is when the foot and lower leg is attacked by **mainly fungi and even some bacteria** often introduced deep into the tissues by thorns. Prolonged anti fungal treatment + antibiotics may stop the process used early but most late cases end up in amputations.

**Madura foot:** A special problem is when the foot and lower leg is attacked by a **combination of fungi and bacteria** often introduced deep into the tissues by thorns. The grains where the organisms grow very slowly result in an area of hard swelling, with the infection even spreading to, and destroying, bone. There is often multiple sinus tract formation. Prolonged anti fungal treatment and long-term antibiotics such as streptomycin and rifampicin may stop the process if used early but most late cases end up in amputations.

## Parasitic skin conditions

**Cutaneous larva migrans “creeping eruption”.** This is when a non-human parasitic larva, especially *Ancylostoma braziliensis* or *A. caninum* after contact with the larvae e.g. on a beach where there is no control of the faeces of dogs or cats. Their hookworm larvae penetrate the skin and do not develop into the next stage but wander around under the skin. This causes an intensely itchy skin eruption, a red, excoriated track which shows the aimless wandering of the parasites. Diagnosis is by the typical clinical picture. Most lesions are on the feet but small children often have lesions on the buttocks and genitalia. Treatment is with albendazole or ivermectin or local freezing.

### Leishmaniasis

This is a group of diseases caused by parasites related to the trypanosomes but completely different in their clinical presentations. All of the leishmania parasites are spread by sandflies of the genus *Phlebotomus* or the sub-genus *Lutzomyia*. Most of them are zoonoses but some have spread

mainly from man to man. All of the parasites are in the amastigote phase of the parasite when they invade man and are intracellular parasites within the reticuloendothelial cells. Many are very localized in their geographic distribution and can sometimes come in almost epidemic form. WHO estimates that there are 1.5 - 2 million cases of cutaneous leishmaniasis annually. 200 million live in settings where they are exposed to the risk of leishmaniasis.

Sandflies require a precise microclimate that is provided in each endemic focus at particular seasons of the year.

**Transmission:** amastigotes of the parasite are ingested from blood or tissues of the mammalian host by the female fly and transform into promastigotes in the gut, rendering the fly infective after about 10 days. The main reservoir of the parasite apart from man is the gerbil or the domestic dog. In India cattle may be a reservoir.

The diseases are divided into :

**Visceral leishmaniasis**

**Cutaneous Leishmaniasis**

**Muco-cutaneous Leishmania or espundia**

Another sub-division is into:

Old World Leishmaniasis

New World Leishmaniasis

**Cutaneous Leishmaniasis**

This is caused by *L. tropica* or *L. major* (old world) or *L. mexicana* (new world).

This may present in a variety of ways depending on the organisms and the immune response of the individual.

The range of the skin manifestations is wide from a single dry cutaneous lesion which may be ignored unless on the face to a destructive mucocutaneous lesion which must be treated.

There may be, initially in all, a small papule and this may be as far as it develops but often goes on to an ulcer which may show satellites and diffuse cutaneous leishmaniasis DCL (especially in Ethiopia). In the New World there is a localized form showing as a dry lesion on the helix of one ear, the Chiclero ulcer.

The parasite is searched for in skin smears and aspiration from the edge of the lesion or adding culture on NNN medium. The parasite is difficult to see on biopsy material.

When the lesion is dry and localized it is often self-healing but can be stimulated to heal by exposing it to 40-42<sup>o</sup> heat over several days which kills the parasites.

When the lesion is moist and widespread pentamidine or pentostam can be used as treatment.

**Mucocutaneous Leishmaniasis (espundia)**

This is caused by *L. brasiliensis* and is similar to cutaneous leishmaniasis but is more aggressive and invades the external nares and the adjacent mucocutaneous areas of the nasopharynx, lips and even larynx. It destroys both mucosa and cartilage. It can be very disfiguring. There is often apparent healing and reactivation.

Treatment is with pentostam or amphotericin B in very long courses of at least 4 weeks.

Pentamidine can be used in relapses. It has been a major problem with people moving into rain-forest areas such as when the trans-Amazon highway was being built.

There are skin signs after visceral leishmaniasis with pigmented and hyperkeratotic lesions in patches and plaques often called post-kala-azar dermal leishmaniasis.

**Control**

DDT is still very effective in sandflies and if their breeding grounds such as rodent burrows, cracks in termite mounds or cattle sheds are identified these can be sprayed. Gerbil colonies can be

destroyed and affected domestic dogs can be treated or destroyed.

Humans can protect themselves by using impregnated bed.nets or permethrin impregnation of clothes or drapes and the use of repellent creams.

### **Onchocerciasis**

This is a disease caused by a filaria parasite *Onchocerca volvulus* where all the clinical manifestations are due to the microfilariae and not to the adults. It is found in West, East and Central Africa where the biting fly *Simulium* lives and breeds in "white water" rivers and thereby transmits the filaria to man. The microfilariae cause problems when they die with itching, a papular rash and secondary infections because of scratching. The most severe skin effects are usually in the lower body where the majority of bites occur. There is gradually skin thickening with intradermal oedema and scarring and finally in a hypertrophic stage the skin looks like that of a lizard. Then the skin starts to atrophy and looks old and loses elasticity and becomes paper thin and wrinkles when you touch it. The microfilariae that migrate to the face go to the eyes and cause snowflake opacities in the cornea and sclerosing keratitis with opaqueness causing blindness. There can be optic nerve destruction and optic atrophy as well as choroïdo-retinitis.

A huge multinational campaign has been very successful in West and Central Africa to reduce the prevalence of the disease by the widespread use of intermittent ivermectin donated by the manufacturer. This does not get rid of the parasite but effectively frees the body of microfilariae and thereby relieving the symptoms. It has to be repeated every 6-12 months. Long-term doxycycline acts on the *Wolbachia rickettsiae* within the filariae and finally reducing microfilariae.

## **Infestations causing skin conditions**

**Lice** Here the main culprits are the head louse: *Pediculus capitis*, the body louse: *P. humanus* and the pubic or crab louse: *Phthirus pubis*. Bed bugs - vägg löss: *Cimex lectularius* (cause severe itching and the damage from scratching).

**The body louse** is an important vector of epidemic typhus (*Rickettsia prowazeki*), relapsing fever (*Borrelia recurrentis*) and trench fever (*Bartonella quintana*). Transmission is by close personal contact, increased by poverty, overcrowding and poor hygiene. The lice pierce the skin to take a blood meal, injecting saliva and defecating at the same time. A rash appears due to a hypersensitivity reaction to the saliva. These lice live in the clothing

**The pubic louse** leaves behind a blue macule after injection of an anticoagulant. It is a sexually transmitted infection and affects the pubic hairs. The eyebrows, eyelashes and axillae may also be involved. Treat the patients and all their sexual partners with malathion applied to the whole affected area of the body. Leave on for 24 hours before washing off.

**The head louse** spreads within families or classes in school through close contact. Itch and a papular eruption on the nape of the head are common. Treat all in the family, class or school. Resistance to treatment is now a major problem and some health areas recommend a rotation of treatments because of this trend. Sometimes the only answer is meticulous combing of hair with a special comb after applying a conditioner to the hair.

**Scabies:** is caused by *Sarcoptes scabie* and particularly affects children and young adults. It usually spreads within families through close contact. It usually shows up as itchy papular eruptions in the finger webs, wrist flexures, axillae, abdomen especially around the umbilicus and waistband area, buttocks, groins and if seen on the penis or scrotum it is almost diagnostic. Treat all members of the family (even those who are asymptomatic) with malathion or permethrin with the preparation applied to all skin areas of the body from the neck downwards.



**Treatment instructions:** Take a warm bath and soap the skin all over. Scrub the fingers and nails with a firm brush. Dry your body. Apply malathion 0.5% liquid from the neck down including the soles of the feet. Wash off after 24 hours. If you have to wash your hands before 24 hrs. reapply the malathion afterwards. For the particularly itchy areas after this treatment (it takes 2 weeks before the itching starts to fade and during this time the itching is usually worse) use calamine lotion to give some relief.

**Ticks** These are divided into soft ticks Argasidae and hard ticks Ixodidae. The former live in houses, caves and burrows and in a resting phase can survive for 7 years without food or water. They need blood meals and can spread African relapsing fever (*Borrelia duttoni*). Hard ticks are ticks are creatures of open fields and ambush passing hosts. They also suck blood and one of the commonest diseases they spread is **African tick-bite fever** showing usually with an eschar (sometimes multiple) with symptoms of headache, fever, myalgia etc.

**Fleas** Their bites are often very irritating and itchy. They are usually spread from animals, dogs and cats who may not even itch or scratch themselves. Aqueous malathion lotion is usually the best treatment of the animals. especially in the tropics they can spread plague, typhus, Q-fever, tape worm, lyme disease and listeria.

**Tungiasis** This is caused by a special burrowing flea: *Tunga penetrans* resulting in a localized skin eruption near to the big toe on one side of the foot. This is seen especially in those not wearing shoes. It is also known as Chigoe or jigger or sandflea ("Amaundu" in Lamba, Zambia). Careful surgical excision with the flea and eggs is effective treatment. Secondary infection can be very disabling if the whole foot becomes involved.

### **Myiasis**

Various flies can lay their eggs on clothing which, when worn without thorough ironing, may lead to the larva hatching under the warmth of the skin. This larva then penetrates the skin and forms a small lesion like a small furuncle where the person may be aware especially at night of the larva eating into the nearby tissues for nutrients. A common cause in Africa is *Cordylobia anthropophaga* or Tumbu fly, or Mango fly, or Putzi fly Treatment is by applying thick vaseline over the breathing hole of the larva and then squeezing out the larva when it moves towards the surface. Prevention is by careful ironing of all clothing and the drying of underclothing and any clothes that cannot be ironed under an impregnated mosquito net.

## **Cancerous skin conditions**

Here the main conditions seen are **Kaposi sarcoma** nowadays almost always HIV-related (previously seen in a few older men with no link to HIV as in central Europe where it was first described).

**Basal cell carcinomas** are not seen in Africans because of their skin colour and its melanin protection. (this protection is absent for albinos and those with depigmentation)

**Squamous cell carcinoma** is not uncommon and can sometimes be linked to poorly healed burns, other chronic sores due to accidents or chronic diseases such as tropical ulcers. Previously this had a strong link to exposure to arsenic when this was used in cattle dipping baths to reduce tick infestations. This starts as a fleshy dry nodule that breaks down to form an ulcerating lesion with hard raised edges. They are locally very invasive and need a wide excision to avoid recrudescence. Albinos are also at risk of this cancer.

**Melanoma.** In Africans this is **often seen under the foot** when the skin has been subjected to minor trauma regularly over many years. It has often spread such that wide excision is too late to

cure the condition. If these develop from a mole they can be suspected using the **ABCDE** help:

**A Asymmetry** The shape of the mole shows asymmetry

**B Border.** The border show irregularity

**C Colour.** The colour of the mole shows variety of shades and colours

**D Diameter** If the diameter is greater than the eraser on a pencil suspect melanoma

**E Evolving** If the mole shows changes over a period suspect melanoma

## Allergic skin conditions

**Atopic eczema** is unusual in Africans living in traditional settings. This is almost certainly related to their rich bacterial flora and their intestinal helminth load.

**Psoriasis** is much less common in Africans (except those who are HIV-positive). When present it is often difficult to find treatment. An unusual measure is to mix half bitumen and half vaseline and smear this on the affected areas. Of course there will have to be special clothes used so that this mixture does not spoil the usual clothes and all bedding needs protection from defilement. It has an alleviating effect on the lesions.

A special problem not least in expatriates in the tropics is **Pompholyx** because of heat, sweating and humidity. affecting the hands and feet. It shows as rows of small vesicles on finger and toe edges with marked itching. This condition often needs a stronger topical steroid used under a plastic glove to ensure reversal of the inflammation. Sometimes secondary fungal infections may need anti fungal therapy. Use emollients instead of soap to prevent recurrence.

**Contact allergic dermatitis** is often linked to exposure to chemicals, oils or other irritant industrial substances such as cement. Likewise there are lotions and creams used to lighten the skin due to the modern longing for a lighter colour ( or other cosmetics) which may cause irritation and a dermatitis. Contact with nickel and other metals may have the same effect.

**Drug eruptions** are not uncommon (10-20% of those treated may show these). The commonest drugs in question are sulphonamides, allopurinol, carbamazepine, phenytoin, one of the Anti-TB drugs (especially thiacetazone in the presence of HIV - nowadays replaced by other drugs). ACE inhibitors, frusemide, barbiturates. Drugs may lead to photosensitivity: sulphonamides, thiazides, phenothiazines, tetracyclines (ca. 3%) especially in higher doses.

## Malnourishment and the skin

Two important conditions are worth describing

**Pellagra** is due to deficiency or chronic lack of niacin (nicotinamide) in the diet. This is especially common in areas with maize as the staple main diet with too little supplementation with other foods. It was originally classified as vitamin B<sub>3</sub>

Pellagra is classically described by "the three Ds": **D**iarrhoea, **D**ermatitis and **D**ementia. A more comprehensive list of symptoms includes: High sensitivity to sunlight with dark dermatitis where the skin is exposed to sunshine: hands and wrists, Aggression, alopecia (hair loss), edema (swelling), Smooth, beefy red glossitis (tongue inflammation), Red skin lesions, Insomnia, Weakness, Mental confusion, Ataxia (lack of coordination), paralysis of extremities, peripheral neuritis (nerve damage), Diarrhea, Dilated cardiomyopathy (enlarged, weakened heart), Eventually dementia. Treatment with nicotinic acid 50-150 mg daily for two weeks is usually quickly effective. In severe cases this can be given iv. in a dose of 100 mg.

**Kwashiorkor** always means severe malnutrition + infection or a toxin such as aflatoxin or extreme lack of antioxidants or micronutrients. It is not primarily a protein deficiency, rather severe deficient calorie intake plus an infection or toxin.

There is a major imbalance between the potential for **damage by free radicals** and the **protective antioxidant systems**. Infection, oxidative bursts and free iron all contribute to the risk for damage. Children with oedematous malnutrition have **severely reduced levels of glutathione** in blood and mortality is greatest in those with impaired activity of glutathione peroxidase. Marked malnutrition reduces the activity of the sodium/potassium pump with an imbalance in the distribution of electrolytes and fluids. The cell membranes become more "leaky" as ATP runs out and leads inevitably to **low intracellular potassium and high intracellular sodium**. Protein synthesis begins to shut down and all cell processes are damaged. Immune systems collapse and the microvilli of the intestine become more porous allowing bowel organisms into the blood stream.

**Clinical picture:** The skin is often pale, thin with peeling skin where dark "flaky paint" areas contrast against pale "milk chocolate" coloured skin. Often there are sores when these flaking areas peel. The hair is often sparse and thin with a lighter colour and with poor roots so that it can easily be pulled out. It often has a reddish tinge. There is usually an enlarged liver. This child is usually 1-3 years. There is oedema of the face, legs and arms but never ascites. There is often a moon face and the child has usually a moderately low weight for age around 60-80% of the standard. There are wasted muscles especially over the shoulders, upper arms, and the scapulae. There is often a pot belly and the muscles are usually flabby. The child is usually miserable and apathetic with poor or no appetite.

These children are extremely fragile and need very careful resuscitation **treating infection** with broad antibiotic cover (e.g. Chloramphenicol + metronidazole) since 70% have bacteraemia from intestinal leakage due to damage to the intestinal mucosa, **hypothermia** with warmth, **hypoglycaemia** with iv. glucose and **hourly feeding** often by naso-gastric tube. In the early stages feeding will be needed day and night. The fluid should have low protein, low sodium but enough calories, potassium, zinc, magnesium and vitamin A to replace their severe deficiency. They may be dehydrated despite oedema and will need rehydration with e.g. Resomal with **low sodium** but with the above minerals. Even if anaemic they **should not be given iron** at this stage as this stimulates bacterial proliferation. In severe anaemia some blood may be given. Diuretics are banned, despite their oedema, since this will send them into even deeper shock. Skin lesions are treated with zinc ointment which stimulates healing.

**Feeding is at three stages:**

**Resuscitation** - usually with a milk mixture aiming at 400kJ/kg body weight per day for children 6-24 months. The amount of protein should be kept low at this stage with 0.6 g/kg/day. While the anorexia is severe this might have to be by tube feeding. If there is serious hypoglycaemia and hypothermia feeding may need to be hourly day and night. A voracious appetite usually signals the end of the resuscitation phase.

**Early Catch-up phase** - build up the calorie intake slowly and thereafter the protein intake. The number of feeds can now be reduced. If suddenly high energy refeeding is introduced too early the child may develop cardiac failure, profuse diarrhoea and circulatory collapse.

**Late Catch-up phase** - high energy feeds

Return to good mixed meals using family foods

Vitamin and mineral supplements including iron if they are anaemic.

**Social and emotional.** These aspects are at least as important as the first two

Comfort, affection, and mental stimulation for the child  
Support and sympathy for the family  
Help with the family's social problems.