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DERMATOLOGY IN A TROPICAL ENVIRONMENT

RESEARCH, TREATMENT AND CURRENT ISSUES

CONTENT

DERMATOLOGY

Skin conditions are among the most commonly encountered clinical problems for every health worker in the tropics. We are therefore grateful to the editors of MT for dedicating this special issue to dermatology, with emphasis on conditions that occur in a tropical environment.

In addition, this issue will be in full colour which is essential for optimal reproduction of clinical photographs that obviously are so important in dermatology. The contributions were written by members of the Dutch Tropical Dermatology Society (Werkgroep Tropische Dermatologie) with one contribution by Ed Zijlstra. Given the limited space some of the contributions will be published on the NVTG website: 'Scarring alopecia' by C. van Hees, 'Bullous diseases' by J. Engelen and 'Tropical infectious ulcers' by J.E. Zeegelaar.

We would like to take the opportunity to draw attention to another website that provides access to information on skin diseases in Africa. It is based on the excellent chapter on skin diseases among children in Africa by Arjan Hogewoning in his PhD thesis on 'Skin diseases among school children in Africa'. The site also provides access to other publications such as 'Common Skin Diseases in Africa' by Colette van Hees and Ben Naafs and 'Sexually Transmitted Diseases' by Merja Koussa and Koos Sanders. A book on 'Dermatological Preparations for the Tropics' by Peter Bakker and Vincent Gooskens *et al.* that is reviewed in this issue, will also become available on the website: www.africanskindiseases.org

Lastly we would like to mention another publication by members of the Working Group: 'Imported Skin Diseases', published by Wiley-Blackwell Publishing, edited by William Faber, Rod Hay and Ben Naafs.

WE HOPE THAT THIS SPECIAL ISSUE CONTRIBUTES TO A BETTER UNDERSTANDING AND MANAGEMENT OF SKIN CONDITIONS IN A TROPICAL ENVIRONMENT

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Lupus erythematosus

Lupus erythematosus (LE) is an autoimmune disease with very diverse clinical manifestations, potentially affecting major internal organs such as kidney and heart, but more commonly the joints and the skin. The disease has a worldwide distribution but certain populations like Afro-Americans in the USA and people of Afro-Caribbean descent have a higher risk of developing systemic lupus erythematosus (SLE) compared with other groups. These populations may also have an earlier onset and more serious disease manifestations of SLE. This is probably due to the genetic predisposition for LE in these patients. SLE is more often seen in women; the male: female ratio is 1:9.

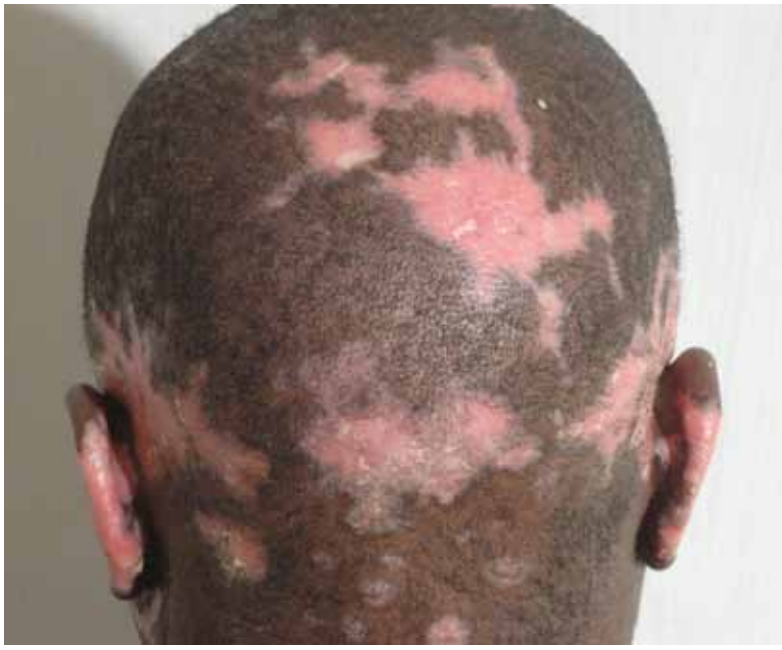
PATHOGENESIS OF LE

There is an aberrant immune response to cellular and nuclear self-antigens that may be triggered by environmental factors in a genetically predisposed individual. The various tissues involved show chronic inflammation that eventually may lead to organ damage and loss of function.

Apoptosis is a strictly regulated process of programmed cell death that follows characteristic biochemical and morphological features. This type of cell death is contrary to necrotic cell death. It is a process that is vital for tissue homeostasis and apoptotic cellular remains are continuously cleared in a non-inflammatory way by macrophages or dendritic cells. This removal of apoptotic debris is associated with the induction of tolerance to the constituents of the material. In patients with LE there are indications that there is an increased amount of apoptotic material and that tolerance for self-antigens is being lost. Apoptotic cellular antigens accumulate in the body and are processed in an inflammatory way with activation of T- and B cells, production of autoantibodies and the deposition of antigen-antibody complex in tissues.

An important environmental trigger is ultraviolet radiation (UVR), which can induce apoptosis of keratinocytes in the skin within a few hours after UV exposure.

Pro-inflammatory clearance of apoptotic keratinocytes may be an important reason for UVR-induced skin lesions in patients with LE. Studies have found that more than 90% of patients with LE show some degree of photosensitivity. Therefore, it is important that all patients with LE are advised on photoprotection. Sunprotection may lead to vitamin D deficiency. Studies show an inverse correlation between SLE disease activity and serum vitamin D concentration although the effect of increasing vitamin D levels on disease activity is in general rather small.



Discoid Lupus Erythematosus (DLE: courtesy Regional Dermatology Training Center, Moshi, Tanzania)

There are many drugs that may trigger or worsen manifestations of LE and it is striking that the offending drug may have been used for several months or sometimes years. (Table 1) Patients with drug-induced LE often have more extensive skin symptoms than those with the idiopathic form and these lesions may persist for weeks or months after stopping the offending drug. These patients also frequently complain of malaise, arthralgia or fever; however, the central nervous system or the kidneys are not involved.

Table 1
DRUGS THAT MAY INDUCE LUPUS ERYTHEMATOSUS

- Calcium-antagonists
- Thiazide diuretics
- ACE inhibitors
- Beta blockers
- Terbinafin
- TNF-alpha blockers
- Protonpump inhibitors
- Statines
- Antibiotics

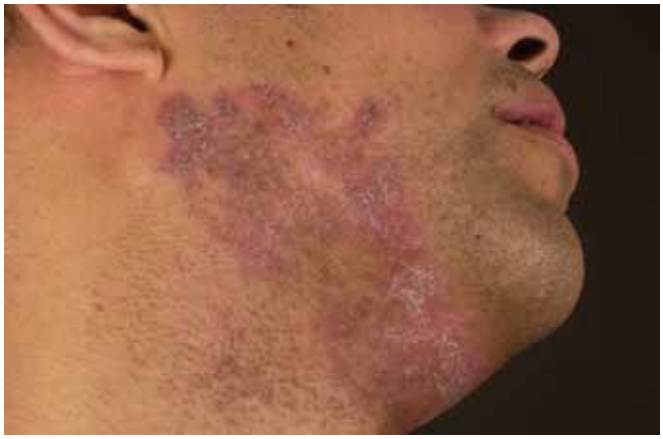
The diagnosis of cutaneous LE is confirmed by histopathological examination of a skin biopsy of a suspected skin lesion. This will often show an interface dermatitis with necrotic keratinocytes and a lymphocytic perivascular infiltrate and gradual thinning of the epidermis. It is not helpful to determine the ANA titre because also in the general population positive titres can be found.

Cutaneous LE is often classified as acute, subacute and chronic cutaneous LE and other subtypes such as chilblain lupus, tumid lupus and neonatal lupus. Non-specific manifestations of cutaneous LE include vasculitis, periungual telangiectasia, Raynaud's phenomenon, non-scarring alopecia and others.

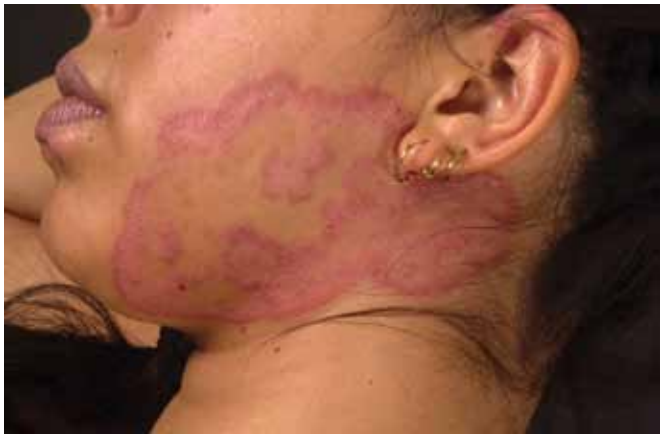
Acute cutaneous LE shows erythematous papules and plaques in face and neck area with erosions. The subacute form has noticeable erythematous scaling patches and plaques that are sometimes annular and are found especially on the trunk and the extensor surface of the upper extremities. Chronic cutaneous LE is manifested by circumscribed papules and plaques with hyperkeratosis and sometimes erosions, often in the face but sometimes more widespread over the body. These lesions heal with scar formation. It is not always feasible to establish one specific diagnosis and there may be several subtypes present simultaneously. In these cases it is better to simply use cutaneous LE as a diagnosis. The risk of progression of cutaneous LE to SLE is about 5-15%. It is important to note that 17% of the patients with subacute cutaneous lupus erythematosus (SCLE) already have four criteria as suggested by the American College of Rheumatology for SLE.

PREVENTION

Patients with SLE are at an increased risk of cardiovascular problems and thrombo-embolic events. Smoking again increases these risks and also causes increased disease activity. Therefore, advice and counselling on how to stop smoking is very important. All patients with LE should receive advice on sun protection, such as behaviour or lifestyle change, protective clothing, and the application of sunscreens. When assessing a patient it is paramount to scrutinize the medications that are being used and stop drugs that potentially induce or exacerbate LE symptoms.



Discoid Lupus Erythematosus (DLE)



Subacute Cutaneous Lupus Erythematosus (SCLE)

CONTINUE READING

TREATMENT OF LE

The goal of therapy in patients with LE in general is to reduce inflammation in all affected tissues or organs and furthermore to prevent damage that may evolve from these processes. The mainstays of treatment of cutaneous LE are topical corticosteroids and immune response modifiers, such as tacrolimus and pimecrolimus. Often potent corticosteroids such as betamethasone valerate are needed in order to reduce inflammation and if this alone is insufficient e.g. tacrolimus may be added. Hyperkeratotic forms of cutaneous LE may be treated with retinoids. If the inflammation is rather deep in the dermis or topical steroids are not effective systemic steroids may be necessary to control inflammation, preferably for short periods of time. Immunosuppressive drugs, such as methotrexate, azathioprine or mycophenolate mofetil, may be used if systemic steroids are contra-indicated or added to the therapeutic regimen as steroid-sparing agents. (Table 2)

Table 2
THERAPY OF LUPUS ERYTHEMATOSUS

- Topical and intralesional corticosteroids
- Topical tacrolimus or pimecrolimus
- Antimalarials (hydroxychloroquine, chloroquine)
- Systemic corticosteroids
- Retinoids (Neotigason, Roaccutane)
- Immunosuppressive agents (methotrexate, mycophenolate mofetil, azathioprine)
- Dapson, thalidomide (lenalidomide), clofazimine
- Pulsed dye laser therapy
- Fumaric acid
- UVA-1, UV hardening therapy
- Biologicals and anti-cytokine therapy (rituximab, belimumab)
- Cyclophosphamide
- Intravenous immunoglobulin
- Stemcell transplantation

ANTIMALARIALS AND LE

Antimalarial treatment is gaining more prominence as a therapeutic tool and also as a potential measure to prevent progression to SLE. There is also increasing evidence that they reduce disease activity and mortality in patients with SLE. Therefore, all patients with SLE should use an antimalarial, preferably hydroxychloroquine, for prolonged periods of time, irrespective of disease activity, other medication used, and also during pregnancy.

Hydroxychloroquine is often given as a daily dose - 6.5 mg/kg lean body weight and chloroquine 3 mg/kg lean body weight. They are probably equally effective but chloroquine has more side-effects. A good way to commence with hydroxychloroquine is to start with 200mg daily and, if no side-effects occur, to increase the daily dose to its maximum level, also taking the kidney function into account. Opthalmological examination is warranted with continued chronic use and high cumulative doses because of the risk of retinopathy.

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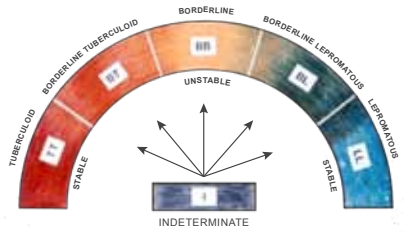
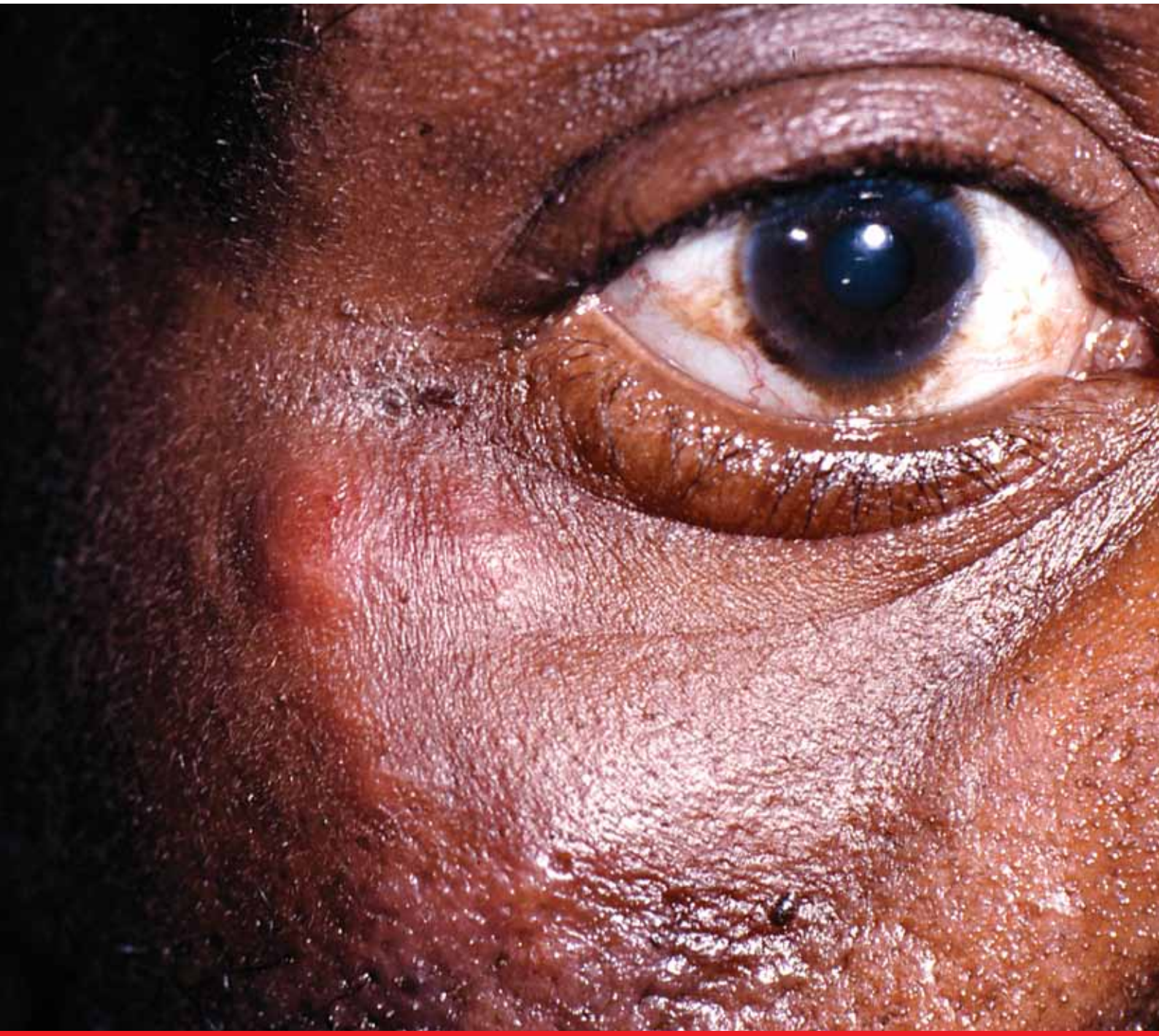
LEPROSY



THE SPECTRUM OF LEPROSY
(Ridley-Jopling Classification)



Type 11 reaction

The general health attendant in resource-poor countries is increasingly confronted with patients complaining of ants running under their skin, of aches and pains in face or limbs, or about loss of sensation in patches or in hand or foot. The area feels numb, dead, or has 'pins and needles'.

The patient may present with hypopigmented or erythematous macules or papules and nodules or plaques which are skin coloured or slightly red. The health worker may think of a diagnosis, but often has to guess and the patient may become worse. Usually leprosy is not suspected; since it is often not included in the curriculum and there is no supervision of a leprosy service, because this has been dismantled, as a result of the claim of WHO that leprosy since 2005 has been eliminated as a worldwide Public Health problem. The alternatives, dermatologists and neurologists, are hardly trained in leprosy, are often not interested and not available. It may have been better in the early 1990s, when knowledge was still available to make a clinical diagnosis and to detect and treat complications in time.

THIS PAPER IS MEANT TO MAKE THE READER AWARE THAT LEPROSY IS STILL PRESENT, TO HELP DIAGNOSE AND TREAT LEPROSY, AND TO RECOGNIZE AND TREAT COMPLICATIONS

DIAGNOSIS OF LEPROSY

Most important in the diagnosis is awareness. Clinically, leprosy is diagnosed when the patient shows two out of three signs. For endemic countries one is considered enough.

The three signs are:

1. Loss of sensation in a skin lesion
2. Enlarged nerve
3. Positive skin smears

Loss of sensation is tested with a wisp of cotton wool; not with a ballpoint. The area in the lesion is tested by touching. With closed eyes the patient points to where he is touched. To make sure, the area outside the lesion is tested as well.

Enlarged nerves can be cutaneous nerves, subcutaneous nerves in the vicinity of skin patches or nerve trunks. Palpate at least the posterior auricular nerves, the ulnar nerves, the radiocutaneus nerves, the median nerves, the lateral popliteal nerves and the tibial posterior nerves. You can expand to every palpable nerve. Feel for thickness, consistence and tenderness.

Smears are taken to detect acid fast bacilli from the ears and other colder areas and from the rim of the lesion in paucibacillary (PB) patients and central in the lesion in multibacillary (MB) patients. The smear is taken with a surgical blade, while squeezing the skin, to numb and to diminish the bleeding; only tissue fluid is required. The cut is smeared with the blade 90 degrees on the cut direction, allowed to dry and stained for acid fast bacilli. The number of bacilli is read and graded along a logarithmic scale (BI: bacillary index) and the percentage of solid bacteria, these are live (viable) bacilli, is estimated (MI: morphological index).

Laboratory investigations are of limited help in the diagnosis of leprosy. However, the skin smear certainly is. Of some help is the titre against phenolic glycolipid I *(PGL-1)*, a cell wall species specific glycolipid. However, this can be positive in contacts, and negative in paucibacillary leprosy. It is used by some to replace the smear. It helps to classify leprosy in PB and MB, and it can be used to follow the effect of treatment in MB patients and to detect relapses. Lymphocyte transformation tests against different antigenic determinants have been a disappointment up to now. PCR and NASBA are often negative in paucibacillary leprosy. They can be useful in the follow-up and the detection of relapses. Biopsy for histopathology can be very helpful, as can immunopathology, but the latter is only experimental. A problem can be that even within lesions, the histopathology of one spot may differ from the other. However it may give an idea of which immunological process goes on.

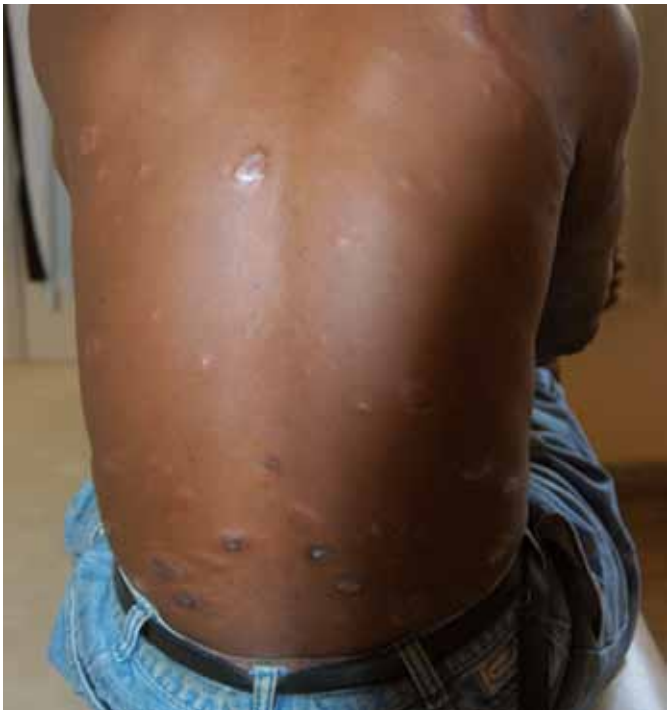
INFECTION AND CLASSIFICATION

Leprosy is highly infectious, but the attack rate is low. The major reason of this low attack rate is that most people genetically are unable to supply the mycobacteria in their cells with what they need to survive, because they lack the genes the bacterium needs.

In order to predict complications and to stratify along cell mediated immunity (CMI), the Ridley-Jopling scale is important, with on one side of the spectrum TT (polar tuberculoid) leprosy with a single well described lesion or an enlarged nerve, with no bacilli detectible and a high CMI against *M. leprae* antigenic determinants. On the other side there is LL (polar lepromatous) leprosy with nodules and or plaques, symmetrical enlarged nerves or even only an infiltrated skin all over (Lepra bonita),with an absence of CMI against *M.leprae* antigenic determinants and many bacilli. In between is the borderline group, which comprises the majority of the patients. Borderline tuberculoid (BT) with predominantly tuberculoid features or borderline lepromatous (BL) with predominantly lepromatous features. Between those two is an unstable group

of mid-borderline (BB) patients with typical, punched out or dome shaped lesions.

For fieldwork purposes, to make it simpler, one just counts the number of lesions: 5 or less is paucibacillary leprosy, more than 5 multibacillary leprosy.



Type I reaction

TREATMENT

Multi Drug Therapy (MDT): Paucibacillary leprosy: 600 mg rifampicin once monthly under supervision and daily 100 mg dapsone for 6 monthly doses in 9 months' time. The dose is for a 60 kg patient.

Multi bacillary leprosy: 600 mg rifampicin and 300 mg Lampren (clofazimine) once monthly under supervision and 100 mg dapsone and 50 mg Lampren daily. Twelve monthly doses should be given within 18 months for low BI patients, 24 monthly doses in 36 months for patients with a BI of 4 or more. The doses are for 60 kg patients.

Be careful with dapsone in Nordic Caucasians who easily develop haemolysis.

The treatment is effective, hardly any relapses are seen.

REACTIONS

Reactions belong to the normal course of a leprosy infection. However, treatment can prevent or precipitate them. There are 3 types of reactions; Type I leprosy reaction, also called Reversal Reaction (RR), type II leprosy reaction, also called Erythema Nodosum Leprosum (ENL) and Lucio's phenomenon.

Type I reaction is a CMI reaction, a type IV Gell and Coombs reaction against *M. leprae* antigenic determinants. Clinically, there is increased inflammation of lesions, which become visible and erythematous, are raised or enlarged; they may even ulcerate. Nerves may be inflamed, enlarged and tender, with diminishing strength, sweating and sensitivity. There may be acro-edema.

Type II leprosy reaction is an antigen-antibody immune complex reaction in the tissues, particularly in skin and nerve. The skin shows the characteristic red, painful, tender nodules. However, all organs can be involved and all tissues can be inflamed. There may be fever and leucocytosis.

The treatment of Type I reaction is primarily corticosteroids, 30-40 mg starting dose, tapering down, guided by, for instance, graded sensory testing, in 6-12 months, in which the dose needs to be 20 mg at least to be effective. In some cases dapsone helps to prevent a reaction.

Type II reaction treatment is difficult. The reaction is episodic, 95% of ENL episodes last less than one month. Mild reactions can be treated with NSAIDs; arthritis with antimalarials, but severe reactions need high dose steroids (60-120 mg) for a short period, diminishing to zero in a month or less. A new attack should be treated the same way. Lampren may prevent a Type II reaction or can be used as treatment. Thalidomide as treatment is superior above anything and can be used as prevention.

When nerves continue to deteriorate despite proper medical treatment, a nerve release operation needs to be considered. This can also be done for nerves without a reaction but which remain tender after treatment.

The Lucio phenomenon presents as an infarction of the skin, due to bacilli blocking the venous return. This is only seen in untreated leprosy. The treatment is MDT.

The results of nerve damage, loss of sensation and muscle strength are the sequelae or the stigmata of leprosy. These should be countered with supplying special, padded tools, utensils and shoes. Sometimes in order to increase grip or to improve foot movement, a tendon transfer may be considered, but always with an experienced physiotherapist present.

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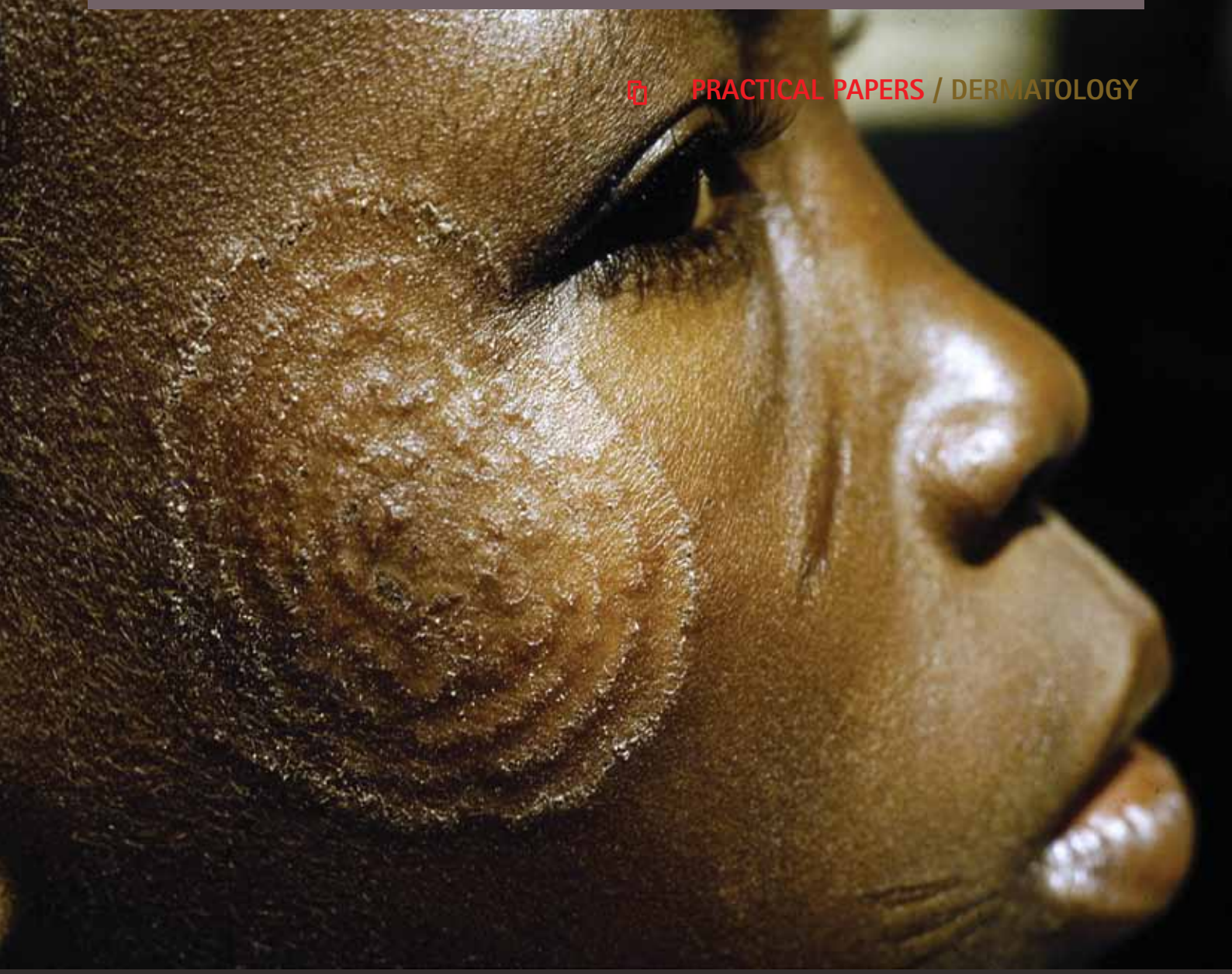
LITERATURE

Leprosy, A practical guide. Editors: Enrico Nunzi and Cesare Massone. Springer Verlag 2012.



Tinea imbricata

FUNGAL INFECTIONS

FUNGI ARE A NORMAL PART OF SKIN FLORA, BUT ALSO A COMMON CAUSE OF DISEASE. FUNGAL SKIN INFECTIONS CAN CLINICALLY BE DIVIDED INTO SUPERFICIAL AND DEEP MYCOSES.

I SUPERFICIAL MYCOSES

Superficial fungi live on the dead keratin layer of the skin. Symptoms are scaling, erythema and vesicles, usually more prominent at the borders of a lesion. Fungi can also elicit an allergic or 'id reaction', an itchy eruption of small blisters at a site distant from the fungal infection, often hands/fingers. Fungi on the skin can be divided into dermatophytes (thread-like, branching filaments) and non-dermatophytes, for example yeasts.

Dermatophytes causing superficial mycoses are members of the groups Trichophyton, Microsporum and Epidermophyton. Infections with these dermatophytes are called 'tinea' and are classified based upon site of infection.

DERMATOPHYTE SUPERFICIAL INFECTIONS

Tinea capitis

This infection is seen primarily in children and the elderly. Finding the source of infection (human or animal) and limiting the spread are important. Attention should be given to sharing combs, etc. Complaints depend on the fungus and the host response and vary from mild erythema and scaling to extensive infection with hair loss, redness, pustules and exudate, leading to destruction of follicles (scarring alopecia). Causes are *M. canis*, *T. verrucosum* (often from animals), *T. tonsurans* and *T. schoenleinii*. Culture helps in diagnosis, but should not delay treatment. Treatment is necessary to prevent permanent hair loss, and should be started immediately. Oral antifungal agents such as griseofulvin (treatment of choice), and an imidazole or terbinafin are given to ensure that the fungus in hair and follicles is eradicated.

Tinea barbae

This dermatomycosis is limited to postpubertal males. Severe inflammation with multiple pustules and sometimes abscesses and sinuses is seen. The causative organisms are often zoophilic, for example *T. mentagrophytes*. As in tinea capitis, oral treatment should be given.

Tinea corporis

Tinea on the trunk and extremities classically presents as 'ringworm', with redness and scaling at the periphery and clearing in the centre. *T. rubrum* is the major cause. Variants are tinea faciei (face) and tinea cruris (groin). The latter is often associated with tinea pedis (see below) and can be confused with candidiasis, eczema and erythrasma. Diagnosis is based on clinical findings and KOH examination.

Tinea manuum

Redness and scaling ('eczema') on only one hand should always raise the suspicion of a tinea. Eczema is usully located on both hands. A KOH examination can be used to differentiate. Treatment is usually topical.

Tinea pedis / athletes foot

This is one of the commonest infections. It is often diagnosed but in more than 50% the cause may be bacterial or a mixed infection. Its increasing prevalence is particularly related to an increase in wearing closed shoes. It can be divided into three categories:

1. Interdigital, especially between the 4th and 5th toe. Symptoms include redness, peeling, maceration and fissuring. Bacterial colonization can lead to 'maceration' and odour. Maceration is often caused by bacteria alone, KOH can differentiate.
2. Moccasin type, a more chronic variant with hyperkeratotic scaling of the sole, heel and sides of the foot, with sometimes fissures.
3. Vesiculobullous, a highly inflammatory variant.

T. rubrum is the commonest cause. The vesiculobullous variant is often caused by *T. mentagrophytes*, a zoonotic pathogen, explaining the more inflammatory reaction. In general patients have few subjective complaints. Treatment is given to limit progression and to avoid the risk of secondary bacterial infection. Usually topical therapy is sufficient.

Tinea unguium / onychomycosis

Though a common infection in temperate climates, fungal infection of the nail is less prevalent in (sub) tropical countries. Yeasts and spores are the main causes. Except for the (finger) nail, the nail fold can also become infected, causing paronychia. Fungi cause only a minority of nail disease. KOH examination or culture should therefore be performed before (systemic) treatment is started.

NON-DERMATOPHYTES SUPERFICIAL INFECTIONS

Malassezia furfur and *Candida albicans* are both part of the normal skin flora. They can cause several skin diseases due to overgrowth and environmental factors such as heat and humidity, and host factors such as individual susceptibility, immunodeficiency, antibiotic use, nutritional status, etc.

Malassezia furfur

Pityriasis versicolor

This is a chronic, asymptomatic infection usually located on the trunk, neck and upper arms. Characteristically, it presents with hypopigmented (darker skin types) or hyperpigmented (lighter skin types), round to oval, sometimes confluenting, mildly scaly patches or plaques. To demonstrate the scaling, stretching the lesion is helpful. It is especially common in the tropics and subtropics and in HIV, sometimes occurring in up to 60% of the population. KOH examination shows the typical 'spaghetti and meatballs'. Therapy can be given locally or systemically, depending on extent and cause. Relapses are common and must be differentiated from post inflammatory pigment changes. Scaling is a sign of active lesions.

***M. furfur* (pityrosporum) folliculitis**

This is usually located on the upper half of the trunk. Itchy, monomorphic papules and pustules are seen. Microscopic evaluation with methylene blue or Gram stain confirms the diagnosis.Treatment is the same as for pityriasis versicolor.

Seborrhoic dermatitis
This is not a true fungal infection, but possibly an immunological reaction to *M. furfur*. Opinions about the role of *M. furfur* in seborrhoic dermatitis differ. It will not be discussed here.

Tinea barbae

Candida albicans

Candida intertrigo
Red patches with scaly, sometimes pustular borders. Satellite lesions are common. Predilection sites are the axillae, submammary, umbilicus, genital area, groin, peri-anal and the finger- and toe webs.

Differential diagnosis: intertriginous eczema, seborrhoic dermatitis and erythrasma. Bacterial superinfection is common.

Mucocutaneous candidiasis
Oral candidiasis: creamy, white flakes on a red, inflamed surface. The papillae may be atrophic or hypertrophic. Usually patients are immunosuppressed or use corticosteroid inhalers In candidiasis of the corners of the mouth, erythema and fissures can be seen. It can occur without underlying condition.

Vulvovaginal candidiasis
Usually itchy or burning erythema and 'buttermilk-like' vaginal discharge. Risk factors should be given attention to avoid relapses.

Onychomycosis and paronychia (see above).

II DEEP MYCOSES

Deep or subcutaneous mycoses are caused by a variety of fungi, that infect the skin including the subcutaneous tissue and in some instances underlying tissues and organs. See also 'Tropical infectious ulcers' and 'Mycetoma' in this special issue or the NVTG website.

EVALUATION

Evaluation consists of scrapings of the 'active', red and scaly borders for KOH investigation. In onychomycosis the border of the affected nail can be used.

Culture is useful in the diagnosis of tinea capitis and onychomycosis. Treatment can be optimized, as some fungi, especially the zoonotic pathogens, are less sensitive to imidazoles.

Sometimes histopathology with PAS-D stain or Grocott silver is necessary. This is especially useful in deep mycoses.

One always has to bear in mind that due to, for example, inadequate treatment or host factors like immunosuppression, mycoses can lose their typical features. This is called 'tinea incognito'. KOH examination or histopathology can be used for diagnosis.

TREATMENT

Most superficial dermatomycosis can be treated topically with an imidazole cream or Whitfield's ointment. Usually the therapy should be given for at least 4 weeks or even better until one week after redness and scaling has disappeared.

Oral candidiasis can be treated with nystatin suspension, vaginal candidiasis with imidazole pessaries and cream.

If hair or nails are involved, or if the superficial infection is too extensive, systemic treatment should be given, for example griseofulvin (not for Candida!), terbinafin or one of the imidazoles. Treatment should only be stopped after complete clearance. Re-evaluating the patient at the end of treatment is therefore important.

Deep mycoses are very difficult to treat and usually require a combination of surgical debridement and systemic imidazoles.

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Mycetoma

Mycetoma (Madura foot) is a thoroughly neglected condition. It occurs worldwide, but Sudan seems to be the homeland. It affects mainly poor people in remote areas which may explain why it does not appear on any list of neglected tropical diseases and is not considered a priority by health authorities. However, it causes considerable chronic morbidity and often it leads to amputation of limbs after years of suffering; in severe cases, there is also mortality. As, in principle, it can be treated by simple medical treatment with limited surgery if needed, it deserves far more attention.

Mycetoma is a chronic infection of subcutaneous tissues that leads to disfigurement and disability in many parts of the world. It is caused by fungi or bacteria, hence it is called eumycetoma or actinomycetoma, respectively.

Eumycetoma is mostly caused by *Madurella mycetomatis*, while Nocardia *spp.* and *Streptomyces somaliensis* are the most common pathogens in Actinomycetoma.

The disease is prevalent in the so-called mycetoma belt which stretches between latitudes of 15° South and 30° North, including Sudan, Somalia, India, Mexico, Venezuela and Argentina. These areas are relatively dry with a short rainy season of 4-6 months duration. In the dry season temperatures are 45-60°C during the day, 15-18°C at night with a relative humidity of 12-18%.

All ages can be affected but in endemic areas most patients present between 20-40 years of age thus affecting the most productive age group. Most patients are farmers or herdsmen; in a recent study in Sudan, however, 30% of patients were students and schoolchildren. The true incidence of mycetoma is not known; in many endemic areas mycetoma occurs in remote areas and many patients lack education or financial means to report to a hospital for treatment; others may fear amputation of the affected limb.

Worldwide most cases occur in Sudan; these are mainly eumycetoma and in 70% of these *M. mycetomatis* is the causative agent. The Mycetoma Research Centre in Khartoum has more than 6000 patients under treatment.

In a recent field study in a village in the endemic area in central Sudan the prevalence was 8.3/1000 inhabitants; eumycetoma was the third commonest health problem after malaria and schistosomiasis. The disease presentation included the whole spectrum from small nodules to extensive lesions and amputated limbs. Few patients had access to medical treatment; surgery was the rule.

The exact source of the infection is unknown; it is thought to be transmitted through a thorn-prick in areas where people tend to walk barefoot; the disease may, however, also affect other parts of the body. To date, the fungus has not been isolated from the soil or other sources. In the endemic villages most cases seemed to occur in the most densely populated part of the village where people live in the same compound as their animals (sheep, goats, dogs, chicken, donkeys) and the ground is covered with animal dung.

CLINICAL PRESENTATION

The typical clinical triad consists of a painless subcutaneous mass, sinus formation and purulent discharge that contains fungal grains. The lesion is usually on the lower limb, involving the foot. From there local spread occurs to lymph nodes and bone; haematogenous metastatic spread may also occur to other parts of the body. Mycetoma may also occur on the hands, and to a lesser extent on the chest, knee, head and neck and perineum. Pain is not a feature unless there is bone invasion or secondary bacterial infection; in one study the latter occurred in 65% of eumycetoma patients. The most common bacteria causing superinfection are *S. aureus* (56%), *S. pyogenes* (34%) and *P. mirabilis* (10%).

DIAGNOSIS

The causative organism may be suspected from the colour of the grains; for example the grains are black in *M. mycetomatis*, red in *Actinomadura pelletieri*, white in *Acremonium* and *Fusarium spp.* and yellow in *Streptomyces somaliensis* and some *Nocardia spp.*

Definite confirmation is essential as this affects treatment; the organism may be identified by fine needle aspirate and examined by cytology, culture and PCR.

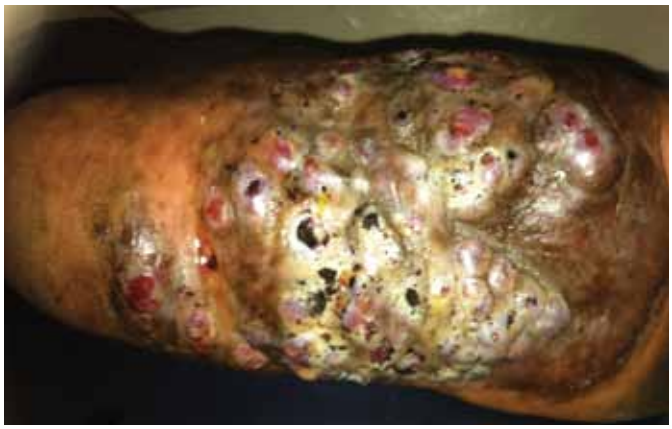
Imaging is useful to assess the extent of the lesion; this is done by X-ray, ultrasound, or MRI scan; clearly these techniques are only available to a certain extent in (referral) hospitals.

There is no rapid and reliable diagnostic test for use under field conditions and diagnosis will be mainly clinical; this may be problematic, particularly in early lesions.

TREATMENT

Treatment is basically with antifungals (eumycetoma) or antimicrobials (actinomycetoma), and surgery as appropriate. Actinomycetoma is treated with antibacterial agents such as

cotrimoxazole; in extensive lesions aminoglycosides are added such as amikacin; the duration may be several months. While medical treatment in actinomycosis is usually satisfactory, in eumycetoma this is not the case. Current antifungals used include ketoconazole and itraconazole; however, these need to be given for 12 months. After that the lesion will have reduced in size and will be more accessible for non-mutilating surgery. However, often the fungus may still be cultured from the lesion after treatment and the recurrence rate is high. In addition, the cost of treatment is high and many patients drop out from follow-up early during treatment. All too often over time this leads to amputation of the limb after years of suffering and disability. In remote areas surgery is the primary treatment.



Mycetoma caused by M. mycetomatis; note the sinuses discharging black grains (courtesy Prof A. Fahal)

Recurrent mycetoma in the thigh; the longitudinal scars and yellow debris are the result of traditional healing (courtesy Prof A. Fahal)

Newer antifungal agents such as voriconazole, posaconazole and isavuconazole are promising; *in vitro* studies show excellent activity. In contrast to ketoconazole and itraconazole, activity against voriconazole is not inhibited in its action by melanin produced by the fungus, suggesting better clinical activity. *In vivo* experience is so far limited to case reports but shows favourable results. Echinocandins are not effective against *M. mycetomatis*.

ISSUES IN MYCETOMA CONTROL INCLUDE

- Assessment of disease burden
- Advocacy for acknowledgement as a neglected tropical condition by WHO
- Health education
- Rapid and reliable diagnosis
- Affordable and safe medical treatment followed by surgery if necessary; explore concomitant antibacterial treatment in eumycetoma
- Research as to the source of the organism and mode of
- transmission

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Case report from Mexico: a sister and brother with painless ulcers

SETTING

The rural San Carlos Hospital is located in Altamirano, a small town in the south of Mexico bordering the jungle of Chiapas. This nonprofit mission hospital provides health care primarily to the poor indigenous population of the region. The staff consists of Mexican 'primary care doctors', a surgeon and an internist. It has basic laboratory facilities, an X-ray machine and an ultrasound apparatus. The nearest regional referral hospital is three hours away by car.

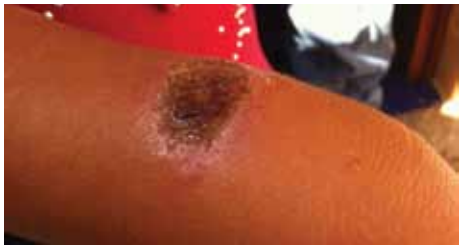

Figure 1. A lesion covered with scab; note the satellite papules at the edge of the lesion

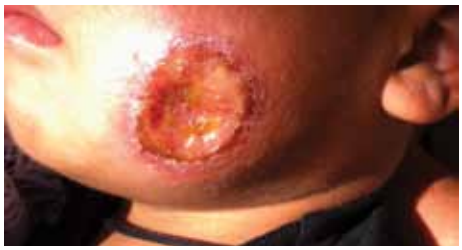

Figure 2. A large wet ulcerating lesion on the cheek

CASE REPORT

A four-year-old girl and her three-year-old brother, both previously healthy, presented at the outpatient department with painless, non-itching ulcers. The girl had one oval dry ulcer of 2 by 3 cm on the lower right arm (Figures 1, 2), the boy had one round wet ulcer with a diameter of 3 cm on the left cheek and another wet, partly ulcerating nodule of 2 cm on the forehead. Both children also had painless enlarged lymph nodes in the neck, axillae and inguinal regions.

The lesions appeared two months earlier as small nodules that gradually increased in size and finally ulcerated. Treatment of the ulcers with an (unknown) ointment and later with oral penicillin had no effect. Apart from minor discomfort of the lesions, no other complaints were reported. A ten-year-old nephew had had a similar lesion on the ear that healed spontaneously. Besides a slightly raised percentage of eosinophils, the haematology results showed no abnormalities. Scrapings were taken from the floor of the ulcers; no parasites were found. Cutaneous leishmaniasis (CL) was suspected. Because of the negative microscopic findings, **CONSULT ONLINE** was asked for advice.

REPORT FROM THE SPECIALISTS AND CLINICAL COURSE

Two dermatologists and one internist-infectious disease physician responded the same day. All were of the opinion that the clinical features fitted in best with CL, whereas deep fungal or bacterial cutaneous infection was considered less likely.

To confirm the diagnosis it was advised to take a deep scraping, an aspirate or biopsy from the edge of the ulcer where the *Leishmania* parasites reside and to examine a Giemsa stained specimen for Leishmanial amastigotes (also called Leishman Donovan bodies). Haematology laboratory results are not of any help. The slightly raised eosinophil count was thought to indicate possible concomitant helminth infection, as protozoan infections usually have no eosinophilia.

It was further suggested that, depending on the *Leishmania* species, most cases of uncomplicated local CL are self-limiting. However, in our patients prompt treatment was necessary to accelerate healing and to prevent disfiguring scar formation, in particular in the face. First choice are pentavalent antimonials, like Pentostam® (sodium stibogluconate) and Glucantime® (meglumine antimoniate), injected in the edge of the lesion. A second option, pentamidine isethionate is less appropriate, as it can only be administered parenterally and will probably be less effective for the species of this region. Miltefosine, a new oral drug, is costly and not available in this setting.

Because of the lack of a diagnosis and unavailability of antileishmanial drugs, the patients were referred to a regional hospital, where infectious diseases should be reported. So far, no feedback has been received.

DISCUSSION

Leishmaniasis is a disease caused by protozoan parasites of the genus *Leishmania* and transmitted by the bite of female phlebotomine sand flies. About twenty *Leishmania* species cause human disease. Most species have a reservoir in animals (zoonotic transmission), but some are transmitted from man to

man (anthroponotic transmission). The clinical manifestations depend on the species of the parasite and the immune response of the host, and can be divided into three major syndromes: cutaneous (CL), mucocutaneous (MCL) and visceral leishmaniasis (VL), also called kala-azar. [1-4]

The leishmaniases are endemic in 98 countries, mainly in subtropical and tropical areas of all continents except Australia and Oceania. The annual incidence of VL is 0.2-0.4 million and that of CL 0.7-1.2 million cases. [5,6] In Mexico over the period 2004 to 2008 annually 811 cases of CL and 7 of VL were described. [5]

As a result of increasing international travelling in the last decennia cutaneous leishmaniasis especially from the New World, has regularly been encountered in Dutch tourists and military personnel. [7,8]

After infection, papules appear within a few days to weeks. They may develop into nodules that ulcerate and often heal spontaneously within months to years, leaving an atrophic scar. The lesions do not itch and are painless unless secondary bacterial infection develops. Multiple lesions and spread along the lymphatics may occur, resembling sporotrichosis.4 Infection results in lifelong immunity which is not sterile; in case of immunosupression recurrence may occur.

Less than 5 percent of CL cases caused by *L. braziliensis* progress over time to MCL, with destructive lesions of the nose, oropharynx and larynx. [1,4]

The differential diagnosis of CL includes many diseases, which list can be shortened on the basis of epidemiology, clinical setting and morphology of the lesion. [1,9]

Diagnosis is made by demonstration of parasites in a Giemsa stained aspirate, a scraping from nodules or the edges of the ulcers, or an impression smear from a biopsy. PCR is the most sensitive diagnostic method, but expensive and not available in the resource-poor setting. Culture is laborious, often not available and takes too much time, whereas histopathology may only be of help in experienced hands. [3,4]

As few cases of New World CL may be expected to be self-healing, treatment should be offered particularly in multiple lesions, single lesions on the face or other areas where they cause functional impairment. Another consideration is the risk of MCL; if *L. braziliensis* is a possibility all patients should be treated. Treatment options include application of heat, cryotherapy, intralesional antimony, ointments with paromomycin or imiquinod, or a combination, and systemic therapy (oral azoles, iv or im antimony, iv or im aminosidine, iv or im amphotericin B [preferably as liposomal formulation], iv or im pentamidine, or oral miltefosine. [2-4]

CL in Mexico and in most other parts of Central America is caused by *Leishmania mexicana* or *Leishmania braziliensis*. Uncomplicated lesions with unknown Leishmania species are effectively treated with short course intralesional pentavalent antimony. It is cheap, has no systemic side effects and therefore warrants good compliance. [5,6] However, treatment in the face in young children is not attractive and other options include systemic antimonials and (liposomal) amphotericine B. If the parasite is identified as *L. mexicana infection*, ketoconazole and miltefosine may also be considered. [6]

At present, no vaccine or chemoprophylactic drug is available. Prevention is aimed at reducing infectious bites of the vector and control measures of the reservoir. [3,4]

CONCLUSION

In this case report we presented two Mexican children with painless ulcers. Although the diagnosis has not been confirmed, the clinical presentation is very suggestive of cutaneous leishmaniasis and the children were referred to a regional hospital for further management.

The case report gives an example of a common dermatologic disorder with which tropical doctors can be confronted in their daily work. It also gives an example of the burden of neglected tropical diseases in the world's poorest populations, and shows the importance and need of systematic prevention and control programmes.

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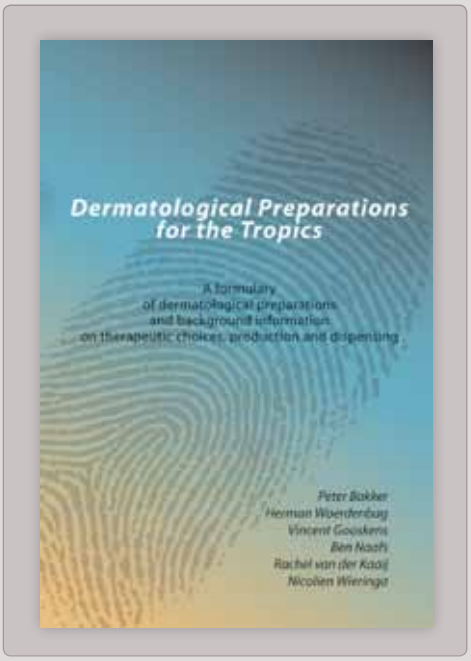
DERMATOLOGICAL PREPARATIONS FOR THE TROPICS



Dermatological Preparations for the Tropics >

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Skin diseases in developing countries are often considered less of a health priority than diseases like HIV/AIDS which have a higher mortality. However, the many adults and children that suffer from common skin disorders like pyoderma, scabies, tinea capitis, pediculosis capitis and acne have significant morbidity. In many parts in Africa skin diseases account for up to 20 % of the visits to primary health care facilities. In addition because the majority of skin diseases can easily be treated with cheap and simple medication and dermatological preparations it is important to pay attention to this.

Dermatological Preparations for the Tropics is a practical guide that can assist health care workers in finding and producing an effective treatment for most skin diseases. Special attention is paid to different dermatological disorders and which therapies could be used, how they work and which therapies are suitable for use in a tropical environment.

Most importantly this book describes the Basic Standards of Good Manufacturing Practice including which equipment to use, hygiene and the procedures to follow for manufacturing dermatological preparations on a larger scale. The book also explains in a very clear and understandable way how to produce these preparations and it discusses the stability of the preparations in tropical environments. Domestic production of skin preparations can be a good start for building self-reliance in medicine manufacturing.

Dermatological Preparations for the Tropics is the result of cooperation between dermatologists, pharmacists and public health specialists. It is a very practical guide that will help pharmacists and pharmacy technicians to prepare simple dermatological preparations at very low cost. It is highly recommended for all health care workers who want to treat many patients with skin diseases with cheap preparations which are still of good quality.

The book will also become available online, at www.africanskindiseases.org.

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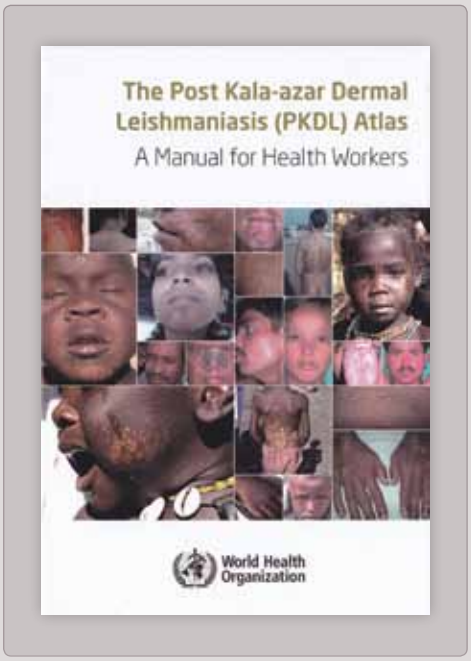
THE POST KALA-AZAR DERMAL LEISHMANIASIS (PKDL) ATLAS: A MANUAL FOR HEALTH WORKERS



The Post Kala-azar Dermal Leishmaniasis (PKDL) Atlas: A Manual for Health Workers

Authors
E.E. Zijlstra and J. Alvar, World Health Organization, 2012

This manual – or rather a photo atlas – is a very practical clinical aid in the diagnosis of the neglected and complex dermatological condition of Post Kala-azar Dermal Leishmaniasis (PKDL). PKDL is a common complication of visceral leishmaniasis (VL, or kala-azar), and is seen mostly in countries in East Africa, particularly Sudan, and in southern Asia.

The neglect of PKDL, both from a clinical and an epidemiological point of view, is partly caused by the difficulty in recognizing PKDL and making a definitive diagnosis. Microscopy of skin smear or biopsy generally has a low sensitivity. Therefore, diagnosis is usually made clinically by the combination of the typical rash, its distribution, and a previous episode of VL. Especially for health workers in remote endemic settings without access to dermatological expertise this remains a major challenge, because the clinical presentation varies and the list of differential diagnoses is extensive.

This atlas can help health workers make a reasonable diagnosis of PKDL based solely on clinical grounds. The authors have made a call to experts and people in the field to contribute photos, and after reviewing many thousands, they are presenting a selection of over 400 high-quality photos, covering the wide spectrum of presentations of PKDL, as well as the common differential diagnoses per endemic area. The PKDL atlas has become an attractive reference work, which will not only serve as a valuable guide to better and earlier recognition of PKDL by health workers in remote areas, but will also be a useful tool in clinical teaching.

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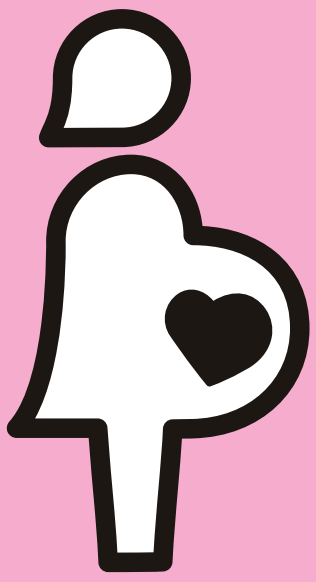
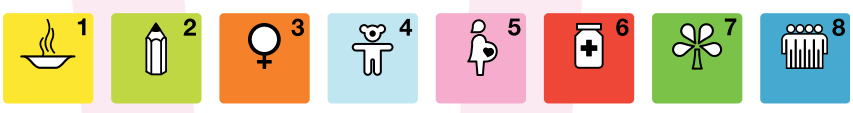


5 IMPROVE MATERNAL HEALTH

HOW *COMMUNITY-DRIVEN MATERNAL DEATH AUDITS* TRIGGER CHANGES

During her first pregnancy, Sabita D, 18 years old, received 3 antenatal check ups. Her family decided that she would deliver at home with a traditional birth attendant; delivering in the hospital would be too expensive. During labour, she was in severe pain and the TBA referred her to the hospital. Arriving at the Deoghar district hospital, she was refused because of unavailability of blood. Then they took her to a private hospital, but there were no beds available. Finally another private hospital agreed to admit her, the doctor mentioned that she needed an operation and asked the family for money. It took a few hours to arrange the money, and when the family wanted to deposit it, they found that Sabita had already died while waiting for her treatment.



INTRODUCTION

The fifth Millennium Development Goal (MDG 5) aims to reduce the maternal mortality ratio (MMR) by three-quarters between 1990 and 2015. Maternal deaths are defined as the number of women who die during pregnancy or within 42 days of the termination of pregnancy. However, accurate information on MMR is missing; even in countries with adequate civil registration systems, special studies have revealed that about 50 per cent of maternal deaths go unreported due to misclassifications. Simavi is a Dutch NGO that supports community health care initiatives. The organization promotes community based maternal death audit, also called verbal autopsies, as a tool to prevent similar future deaths. In India, one of their local partners NEEDS piloted the tool in Deoghar district in Jharkhand, one of the Northern states in India with a high MMR.

BACKGROUND COMMUNITY BASED MATERNAL DEATH AUDIT IN INDIA

In absolute numbers, India is the number one leading country contributing to maternal deaths. In this country, every ten minutes, one mother dies due to pregnancy related problems. The Indian government is committed to reduce the MMR, as reflected in the Indian Health Policy, through the establishment of Maternal Health Committees in every district, and the implementation of Maternal death audits (also called verbal autopsies). Simavi's partner NEEDS, is a member of the Maternal Health Committee in Deoghar District. In 2011, it started to organize community based maternal deaths audits at block level to (1) identify maternal deaths and their reasons, (2) understand institutional mechanisms and practices, leading to maternal problems, (3) understand community practices contributing to this problem, (4) finding solutions to improve maternal health.

PREPARATION AND DATA COLLECTION

NEEDS trained 18 local volunteers during 2 days' training on the use of verbal autopsy forms, conducting in-depth interviews and focus group discussions (FGDs). The first research took place in Devipur block, with a population of 84,000 covering around 50% of the block area. The first step was to contact traditional birth attendants (TBAs) and female frontline workers (ASHAs), who are focal persons for pregnant women. They could report death cases of women in reproductive age (WRA) in the past 12 months. The team identified in total 40 death cases.

The second step was reviewing all death causes through planned interviews with family members of the women. Volunteers used a verbal autopsy form and 11 cases were identified to be maternal deaths. Forms were reviewed by the President of the Indian Medical Association Jharkhand and all 11 cases were verified as maternal deaths. It was alarming that none of them were officially recorded as maternal deaths. The last step was the collection of information on the causes of death by history taking through in-depth interviews with family members.

LOOKING FOR SOLUTIONS

A report was prepared on the process, describing the individual deaths and presenting the different causes related to the maternal mortality. One case was excluded since insufficient details were available. Common causes related to (1) delays in seeking appropriate assistance at community level (e.g. lack of information, not attending antenatal care), (2) delays in reaching the appropriate facility (transport/ costs, opening hours, availability of staff) and (3) delays in receiving adequate and timely care on arrival.

With the information on the causes, FGDs were conducted in the community with women of Self-Help groups, the Village Health Committee and front-line health workers to collect information on community based solutions. This included discussing pressuring mechanisms related to requesting emergency transport facilities and voicing concerns of lack of services and referral possibilities. At community level, mechanisms to improve access to information and pregnancy-related services were discussed as well as saving and loan systems through Self-Help groups for emergency cases.

NEEDS also presented the report to the clinicians and health staff to start the discussion on ways to prevent repetition. Then the organization was invited to present their findings before a national advocacy platform. Consequently, NEEDS took the initiative to present the findings to the state government of Jharkand but it did not result in any action. After a second national advocacy presentation, the findings were published in the Hindu, an important national newspaper. Thereafter, the national health authorities instructed state authorities to organize ambulance services within a week. This was organized instantly and existing ambulance services and their telephone numbers were mapped, so that community members, frontline workers and health workers instantly could contact them in case of emergencies.

PEOPLE REALIZE THEIR RIGHT TO SERVICES AND THE FACT THAT HEALTH WORKERS AND THE GOVERNMENT CAN BE HELD ACCOUNTABLE

DISCUSSION

In spite of the long recall period for the family members, the maternal mortality audit provided evidence with a very detailed level of information. The information led to the identification of mechanisms to prevent delays at different levels and practical solutions to prevent maternal deaths. Simavi and NEEDS emphasize the involvement of the community members in the process, thereby recognizing and underlining their own role in improving safe motherhood. Moreover, people realize their right to services and the fact that health workers and the government can be held accountable and requested to take action. In this case, the advocacy activities of NEEDS were very successful in triggering political interest at national level.

CONTINUE READING

Also the findings reached a wider audience through newspaper coverage. The support from the Indian government – promoting safe motherhood and encouraging the implementation of maternal death audits – contributed to the fact that the findings of NEEDS, relating to 11 maternal deaths only, were taken seriously. An important finding was that none of the deaths were reported as maternal deaths in the health statistics. This clearly shows the advantage of a community based maternal death audit compared with a facility based audit. It is known that maternal deaths are frequently unreported resulting in an inaccurate MMR. The 11 deaths in half of the block of Devipur would correspond with a MMR of 1140, which is 4x higher than the State MMR (Census 2011). This finding is a direct result of the community based maternal mortality audit system, thereby underlining the importance of such interventions. Since NEEDS is planning to conduct audits within one month after the death of the mother, the system can provide almost real-time maternal mortality information, which encourages timely action, not only from community members, but also health workers and responsible health authorities.

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This is the first edition of MT *without* International Health Alerts

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