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Building a Network: The International Skin Care Nursing Group (ISNG)

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ISNG Advisory Board

The worldwide burden of skin disease and therefore the demand for skilled skin care is high; especially within community dermatology settings. Invariably, specialist nurses and also many primary care practitioners represent one of the largest resources of skin care expertise within national health systems. For over a decade ISNG has successfully brought together nurses from across the globe to provide support for nurses working in the dermatology field, particularly in resource poor countries, to debate and exchange knowledge and enable education and development in the care of people with skin disease or a compromised skin barrier.

The central role of the ISNG is to promote the optimal involvement of nurses in the delivery of quality skin care worldwide through education, development and research. ISNG facilitates networking and educational opportunities; these have always been central to its activities. A number of international meetings have been organised, including those in Africa, India as well as across Europe, involving nurses involved in the care for those with skin disease. ISNG has also maintained active communication with approximately 300 dermatology nursing activists (and other related healthcare professionals) from 32countries through newsletters, our website (www.isng.org) and conferences.

It has an Advisory Group comprised of seven senior dermatology nurses from five continents and a dermatologist, Dr Vineet Kaur, all of who serve as key nursing leaders in the dermatology field. Most have played leading roles in national dermatology nursing organisations worldwide.

ISNG has also actively contributed to the setting up of national dermatology nursing organisations throughout Europe, but supported networking amongst nurses and with dermatologists in countries such as South Africa.

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Building a Network: The International Skin Care Nursing Group (ISNG) *continued*

Skin care practices for lymphatic filariasis –teaching effective drying of the interdigital spaces

The work of the Group has been recognised by the *International Council for Nurses* (ICN), the international statutory body for nursing. The ICN has granted ISNG with affiliate status, with it being one of only five affiliate organisations. ISNG is also now the nursing sister society to the European Academy of Dermatology of Venereology and is an affiliate of the *International League of Dermatological Societies*. It also has good working links with the *International Foundation of Dermatology*, with whom it shares a common purpose.

Advisory Board members have participated in public health projects. For example, during its first three years ISNG employed a Project Coordinator (Rebecca Penzer) to help the Board develop and contribute evidence-based advice on skin care to the *WHO* Global Morbidity Control Programme for the Elimination of Lymphatic Filariasis.

Some specific key developments may be illustrated with the case of India over recent years. Dr Kaur, our Advisory Group lead for Asia, has been leading a Nursing task Force instigated within the *Indian Association of Dermatologists, Venereologists and Leprologists* to support the development of dermatology nursing in the sub-continent. This embraces a number of prominent dermatologists from India, including several from the military, and ISNG members. A survey has been undertaken recently and work is currently taking place to develop a curriculum to support nursing education in this field.

Another key area of development is in South Africa where Pat Kelly (ISNG Board Africa lead) and Senior Professional Nurse from the University of Cape Town Division of Dermatology, has been working with dermatology colleagues (Prof Todd and Dr Jessop) and Dr Duma from the Division of Nursing to develop a Postgraduate Diploma in Dermatology Nursing. ISNG members have been involved in commenting on the curriculum, with the first course taking place later this year. This builds on the dermatology team's work over a number of years in preparing nurses with dermatological expertise to span out across Africa to meet community dermatology nurses in many places where expertise is very limited. This cascade model, where specialist nurses can meet needs directly but also share their skills with general nurses is an important approach, being seen in many countries in the world; however, the education preparation required to support this model requires substantial development in many parts of the world. ISNG continues to try to support such developments, wherever help is required.

The Group has been invited to set up the nursing scientific meetings at the *World Congress of Dermatology*; the first being in France (2002), Argentina(2007) and in May 2011 South Korea. The next programme in Seoul this year incorporates a range of international speakers debating on issues and best practice related to skin care nursing, drawing on a developing evidence base. Papers from the meeting will be disseminated to help nurses worldwide gain access to key discussion on developments in dermatology nursing worldwide.

Further details of the work of ISNG can be found in the forthcoming special issue of the I*nternational Journal of Dermatology* on community dermatology[1], where together with dermatology colleagues we explore the capacity of dermatology health professionals to respond to skin care/ dermatological needs worldwide. This will complement the special session held at the World Congress of Dermatology this May, with Professor Terence Ryan leading on this initiative for the *WHO* and the *International Society of Dermatology*; this work is actively supported by ISNG.

ISNG welcomes contact from isolated nurses worldwide who may wish to link with ISNG for support, allied health professionals involved in dermatology care or groups of nurses in a country wishing to develop a national group.

South Africa meeting- Cape Town 2006

Reference:

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Infectious causes of leg ulceration

J Fleming, D Odunsi, R Morris-Jones

Introduction

Skin ulceration can result from trauma, impaired blood supply and a myriad of infections; bacterial, fungal and protozoal. Differentiating between the possible causes of cutaneous ulceration can be difficult ˆ this article focuses on the clinical appearance, diagnosis and management of cutaneous leishmaniasis, buruli ulcer, tropical ulcer, Yaws, dracunculiasis, ecythma and ulcers caused by streptococci.

1. Cutaneous Leishmaniasis

There are over 20 species of the protozoon *Leishmania*, with a wide range of animal reservoirs both wild and domestic. Disease is transmitted by the female sandfly. *Phlebotomus*.

Classification and presentation

Cutaneous disease can be subdivided into acute, diffuse and chronic forms. Acute CL is the most common and presents with a small erythematous papule on an exposed skin site usually less than 10cm in diameter and painless unless associated with secondary infection. Over 4 -12 weeks it enlarges into a "wet" lesion which releases a seropurulent discharge that later dries forming a crust. On removal of the crust a shallow crater-like ulcerated lesion is revealed. The differential diagnosis of this ulcer would include a diabetic, venous or mixed venous-arterial ulcer however if the patient lives in an endemic area for Leishmaniasis this should be considered.

Alternatively, a "dry" lesion can form in which there is no ulceration, remaining a smooth, hyperkeratotic nodule.[1]

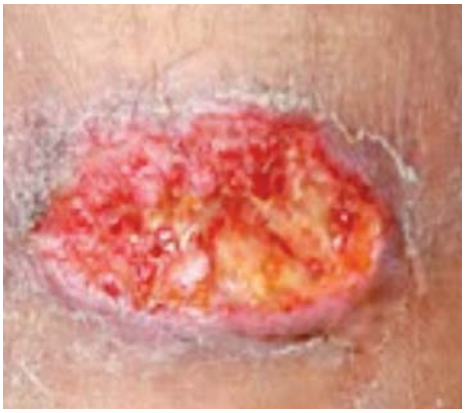
Treatment of cutaneous leishmaniasis

Systemic therapy includes sodium stibogluconate or meglumine antimoniate 20mg/kg/day once daily IV or IM for 20 days. Sodium stibogluconate can also be given by injection directly into and around the ulcer margins to good effect.

In rural settings where these drugs are unavailable leishmaniasis ulcers may be left to heal spontaneously which they usually do over 2-5 months. Antiseptic washes can be used to prevent secondary infection, Radio-frequency induced heat therapy has also been used to clear local cutaneous disease.[3] HIV testing should also be performed as co-infection may hamper efforts to treat the condition.

Preventive measures are central to disease control, and are aimed at avoiding contact with sand flies, these include:

- The use of bed nets impregnated with insecticide.
- Elimination of sand flies by environmental measures and insecticide.
- Protective clothing and use of insect repellent on exposed skin.
- "Reservoir control" – at risk or infected animal population control.



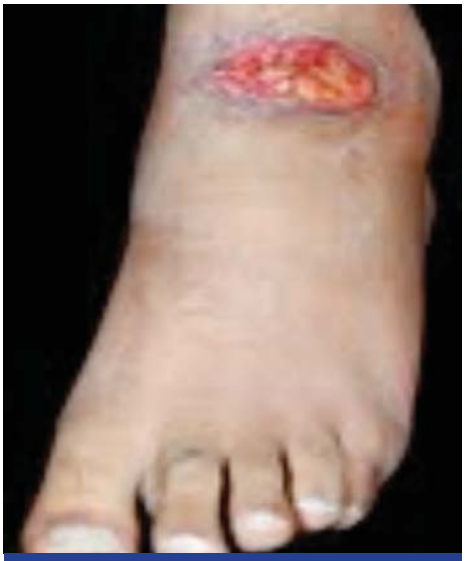


Fig 1. Ulcer from Leishmaniasis

2. Buruli ulcer

Buruli ulcer is a necrotizing disease of the skin and underlying tissue, caused by *Mycobacterium ulcerans*. Buruli ulcer is found in more than 30 tropical and subtropical countries. Over the last 2 decades the incidence of Buruli ulcer has increased, despite significant underreporting of cases. In 1999 there were 6000 new cases in Ghana.[4] Although the exact mode of transmission is unknown, *M. ulcerans* most likely causes cutaneous infection through a portal of entry such as a traumatic wound.

Fig 2 - 4. Buruli Ulcer

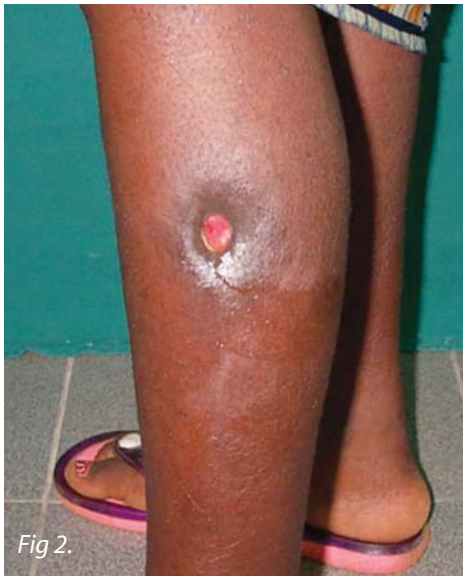


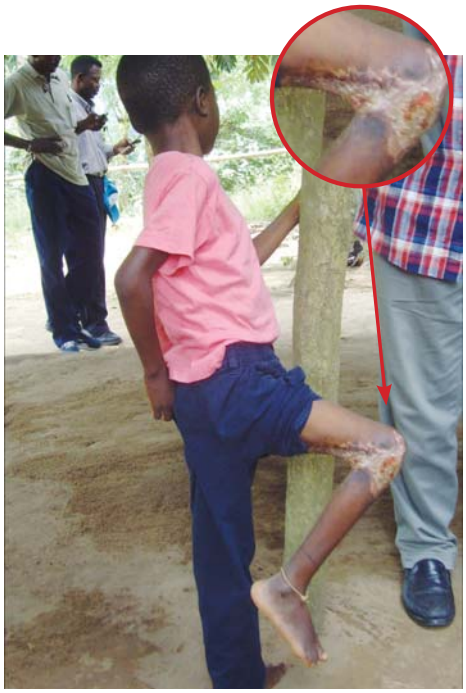
Fig 2.

Fig 3.

Fig 4.

Photos: Courtesy of Dr Y Lugor, Sudan, WHO website, accessed December 2010

Photo: Courtesy of Dr A. Tiendrebéogo, Nigeria



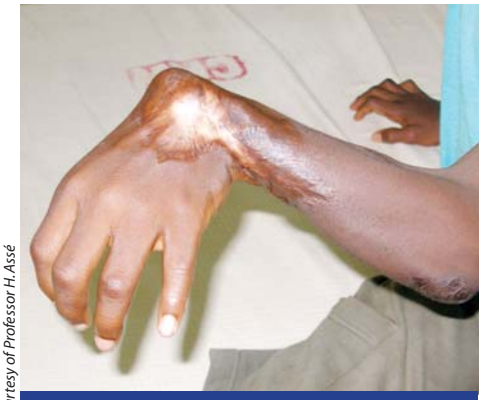


Photo: Courtesy of Professor H. Assé

Fig 6-7. Fixed flexion deformities resulting from scar adhesion

Clinical features

Buruli ulcer begins as a firm, painless nodule in the skin, which is around 1 to 2 cm in diameter. *M. ulcerans* produces a toxin, called mycolactone. This toxin causes extensive tissue destruction. Over the following weeks, the nodule breaks down to form a painless necrotic ulcer with undermined edges. The ulcer can extend down into deeper tissues destroying nerves, blood vessels, muscles, and occasionally bone. The legs are most commonly involved.

Making the Diagnosis

- In endemic areas Buruli ulcer is often diagnosed and treated based on clinical findings.
- A direct smear taken from the necrotic base of the ulcer can be stained with Ziehl-Neelsen stain and may reveal clumps of acid-fast bacilli on microscopy.
- *M. ulcerans* can also be cultured from swabs taken from the ulcer or fresh tissue biopsies, but this process can take 6 weeks or more.
- Polymerase chain reaction testing on swabs of ulcers or tissue biopsies can be performed where resources allow.

Infectious causes of leg ulceration *continued*

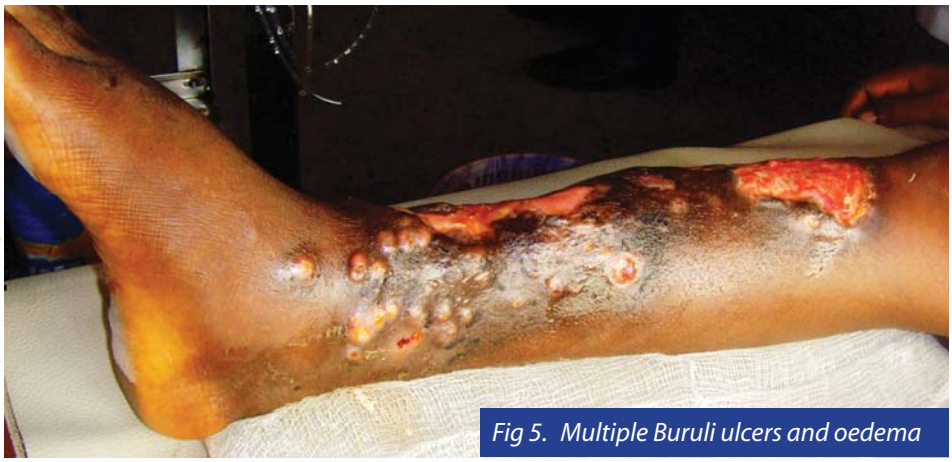


Photo: Courtesy of Dr K. Asiedu, Nigeria

Fig 5. Multiple Buruli ulcers and oedema

- A biopsy of the lesion can reveal characteristic microscopic changes and clumps of acid-fast bacilli seen on haematoxylin and eosin stained histopathology sections of lesional skin.

Treatment

Current WHO recommendations for treatment are as follows:

- A combination of rifampicin and streptomycin or amikacin for eight weeks as a first-line treatment.[4]
- In resource poor settings, rapid diagnosis is key to prevent complications and antiseptic and antimicrobial topical agents and dressings will help prevent secondary infection and potential osteomyelitis (Fig 8)
- Surgery to remove necrotic tissue, cover skin defects, and correct deformities is a crucial part of the management and helping to improve disability (see figs 6-7).[5]
- BCG vaccination appears to offer some short-term protection (less than one year) from the disease.

3. Tropical ulcer

Tropical ulcer, also known as tropical phagedenic or 'gnawed' ulcer, presents as a painful, rapidly enlarging sore, usually found on the lower limb of an individual living in a hot, humid tropical region.

What is the cause of a tropical ulcer?

Multiple factors play a role in causing tropical ulcers. There is initially some form of injury to the skin, often as minor as a scratch or an insect bite. Rural labourers who do not wear adequate protective clothing and footwear are at increased risk. Poor nutrition, poor hygiene, and chronic diseases such as malaria and intestinal parasites also increase the risk of a tropical ulcer developing. A variety of bacteria are found to be present; *Fusobacterium* species are almost always present in the early stages; *Bacillus fusiformis* and *Treponema vincenti* found in the late stages. Other bacteria such as *Escherichia* coli and *Enterococcus* species are implicated.

Clinical features

The ulcer is initially circular, superficial, painful, and has purple edges. It enlarges rapidly across the skin and down into deeper tissues such as the muscle or even the periosteum. The clinical presentation may initially resemble pyoderma gangrenosum. The ulcer may reach several centimetres in diameter after a couple of weeks. The edges become thickened and raised and the central crater may become necrotic.

Differential Diagnoses

It is important to rule out other causes of ulcers such as cutaneous leishmaniasis, atypical mycobacteria, pyoderma gangrenosum, and venous disease. Swabs can be taken from the base and edges of the ulcer to determine the type of bacteria present (see above).

Treatment

Antibiotics such as tetracycline, penicillin or erythromycin and metronidazole are preferred oral therapy. Surgical debridement and skin grafts may have a role but the area may need to heal by secondary intention after daily cleaning with a disinfectant such as Dettol and bacitracin, if available, or sterile dressings twice a day. Improving a patient's general health and nutrition will also aid healing.

4. Yaws

Introduction

Yaws remains one of the most neglected tropical diseases affecting the poorest, most vulnerable populations in the world leading to the expression, "Where the road ends, yaws begins". Attempts to eradicate Yaws have as yet been unsuccessful however the WHO has issued a new target of elimination by 2012. Yaws, also known as 'pian' or 'parangi', is a chronic, non-venereal infection caused by the spirochaete *Treponema*

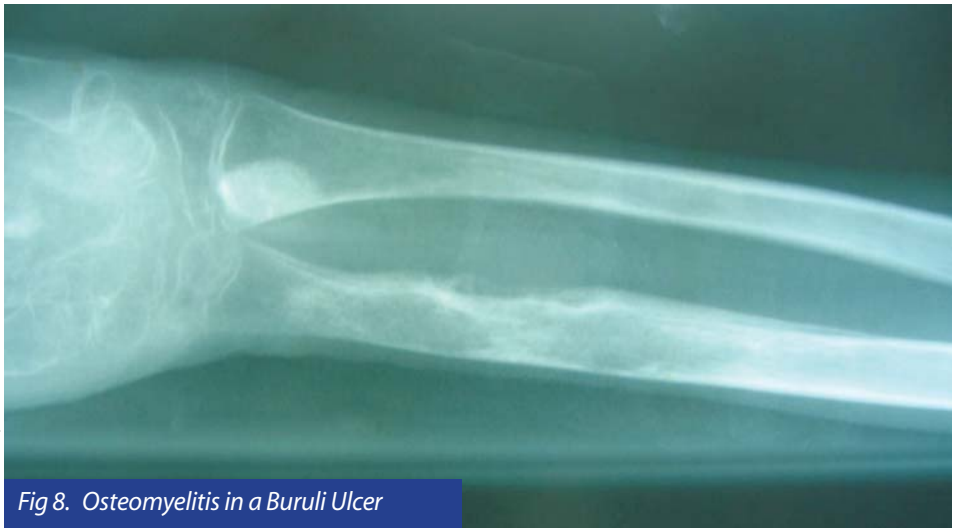


Photo: Courtesy of the WHO and Global Buruli Ulcer Initiative (GBUI)

Fig 8. Osteomyelitis in a Buruli Ulcer

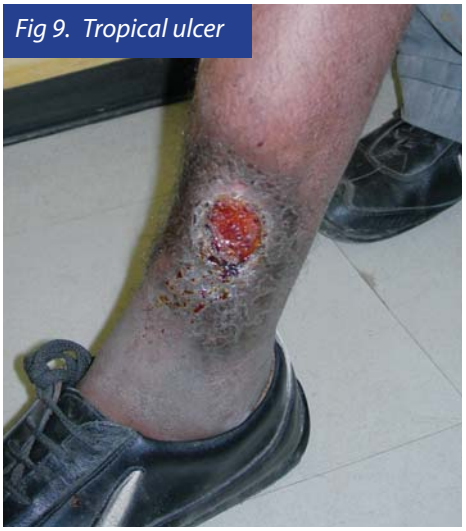


Fig 9. Tropical ulcer

Photo: Courtesy of Dr Stephen Walker and the Department of Dermatology, ALERT Hospital, Addis Ababa, Ethiopia

pallidum pertenue, that affects mainly skin, bone and cartilage. Yaws, from the word 'yaya' meaning sore, occurs mainly in developing world communities in warm, humid, tropical areas of Africa, Asia and Latin America and is spread by direct skin contact with an infected individual. Children are mainly affected (6-10 years).

Overcrowding, poor personal hygiene and sanitation facilitate disease spread.

Clinical course

After a 2-4 week incubation period a slowly enlarging warty nodule ('mother') lesion develops at the point of inoculation, occasionally nearby 'daughter yaws' appear simultaneously. This primary stage usually resolves completely within six months. Secondary Yaws occurs months to years later, and is characterised by disseminated cutaneous lesions of heterogeneous appearance, including 'crab yaws' on the palms/soles with desquamation. Painful bony lesions also occur and secondary skin lesions frequently ulcerate (becoming highly infectious), eventually healing after six to twelve months. 10% of untreated individuals develop 'late or tertiary yaws' with chronic disfigurement and disability through gross destruction of skin, cartilage and bone causing deformities of the legs, nose (rhinopharyngitis mutilans), palate, and upper jaw. Other skin signs include a painless ulcer with an overlying scab, papillomas and palmoplantar hyperkeratosis.[6]

Photo: Courtesy of the WHO[6]

Fig 10. 'Mother' and simultaneous 'daughter' Yaws warty nodules on the forearm

Diagnosis

Diagnosis is based on clinical and epidemiological findings and the following is helpful in making the diagnosis:

A young person, often a child (75% are children below 15 years) who lives in an endemic area and presents with one or more of the following signs:

- painless ulcer with scab
- papillomas
- palmar/plantar hyperkeratosis (thickening).

The clinical diagnosis can be confirmed by examining a sample from a skin lesion under darkfield microscopic examination. There is no specific blood test for yaws, but because it is closely related to the bacterium that causes syphilis, the blood tests for syphilis are diagnostic in yaws as well.[7]

Treatment

- A single intramuscular injection of Benzathine Penicillin is curative. Relapse is very rare. The dose for adults is 1.2 million units and for children 600 000 units.
- For those who are allergic to penicillin, tetracycline, erythromycin and doxycycline can be used.

Complications

Without treatment, about 10% of affected individuals would develop disfiguring and disabling complications after five years because the disease may cause gross destruction of the skin and bones. It can also cause deformities of the legs, nose, palate, and upper jaw.[6]

5. Dracunculiasis - guinea-worm disease

Introduction

Guinea worm is a large nematode (roundworm), *Dracunculus medinensis*, which is ingested through drinking contaminated water. The worm eventually causes a debilitating and painful infection, beginning with a leg blister. Around the time of its eruption through the skin, the person may experience itching, fever, swelling and burning sensations. Infected people often try to relieve the pain by immersing the infected limb in water. On immersion in water the worm emerges and releases thousands of larvae which may then contaminate open water sources such as ponds and wells. The larvae are ingested by the water flea (cyclops), in which they mature. When a person drinks water containing cyclops the flea is dissolved by gastric acid, and the activated larvae then penetrate the gut wall. After a year, a blister forms on the skin and the mature worm (one metre long), tries to emerge, repeating the life-cycle.

Guinea-worm disease does not kill, but infected people become immobile due to the pain, oedema and fever they experience rendering them non-functional for months. Since the

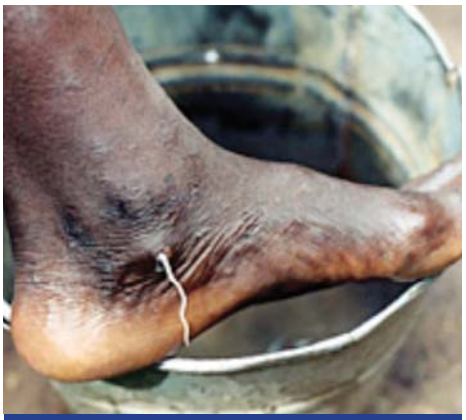


Photo: Courtesy of WHO/Ahmed Tayeh[8]

Fig 11. A mature guinea worm emerging from the skin.

Photo: Coutesy of WHO/Ahmed Tayeh

Fig 12. The worm is pulled from the skin a few inches every day.

Infectious causes of leg ulceration *continued*

Photo: Courtesy of WHO/Kazuyo Ichimori

Fig 13. During the transmission season, contaminated ponds can be treated with a chemical called Abate® (an organophosphorus insecticide), which kills the reservoir of the nematode larvae; water cyclops fleas.

peak transmission period often coincides with the agricultural season, fields are left untended and food production level goes down. In Mali, guinea-worm disease is called "the disease of the empty granary." As adults lie sick, older children must take on the household chores and miss months of schooling etc.

Prevention and Treatment[8]

Early case detection (when the patient feels the initial pain) is vital to contain the disease. Once a new case is identified, the volunteer must clean, disinfect and bandage the wound to protect it from secondary infections. The volunteer returns every other day to gradually pull the worm out, a few painful inches each day. Other important elements to control the spread of infection include:

- Installation of safe water supplies,
- Filtration of drinking water,
- Intensive surveillance and control through early case detection and containment,
- Treatment of ponds with a pesticide that kills the water fleas and health education on disease prevention.

6. Ecthyma

Ecthyma, often referred to as a 'deep impetigo' is an ulcerative pyoderma of the skin caused by group A beta-haemolytic streptococci. The lesion, often solitary, begins as a pustule or vesicle on an erythematous base and later erodes, ultimately forming shallow ulcers with overlying crust. Regional lymphadenopathy is common, even with solitary lesion.

Ecthyma most commonly affects the legs of the elderly, diabetics and children. The feet and ankles of those in tropical climates are particularly at risk.[9] The development of ecthyma can be precipitated by loss of barrier function from excoriations, eczema or insect bites. The overlying crust can often slow healing and predispose to scarring.

Some strains of *Streptococcus pyogenes* have a high affinity for both pharyngeal mucosa and skin, with pharyngeal colonization being documented in patients with ecthyma.[10]

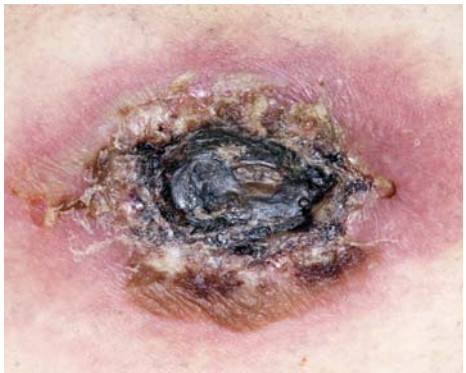


Fig 14. Ecthyma

Management

Maintaining skin hygiene using regular bactericidal soap and washing linen in contact with the skin is important to prevent spread or recurrence. To aid healing, the crusts should be removed by soaking or using wet compresses and applying antibiotic such as mupirocin[11] ointment daily to the eroded area.

More extensive ecthyma lesions require oral antibiotics such as penicillin, erythromycin or clindamycin; the duration of treatment varies because ecthyma may require several weeks of therapy to completely resolve. Widespread ecthyma may require intravenous antibiotics.

7. Ecthyma gangrenosum

This may occur in the context of pseudomonal bacteraemia in an unwell, immunosuppressed patient, although non-pseudomonal pathogens can be implicated.[12] It presents as haemorrhagic pustules with surrounding erythema that evolve into necrotic ulcers. Bacterially mediated blood vessel destruction induces dermal oedema and overlying tissue necrosis and subsequent ulceration.

Treatment will be guided by bacterial sensitivities but empirically treating with an antipseudomonal penicillin such as piperacillin, and an aminoglycoside such as gentamicin is first line. Fluoroquinolones, third-generation cephalosporins (cefuroxime, cephalexin), or aztreonam (if available) can also be used.

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Acknowledgement:

Images of Buruli ulcers sourced from the WHO website with thanks to Stéphanie Jourdan, Assistant to Dr Kingsley Asiedu Global Buruli Ulcer Initiative

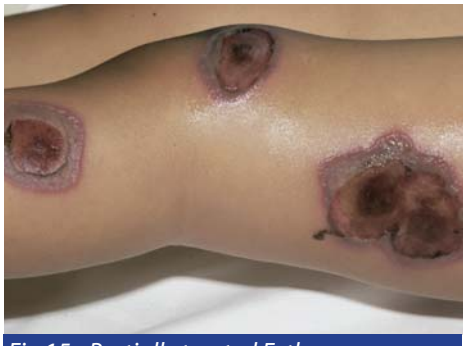

Photo: Courtesy of Dr K.Asiedu, Ghana

Fig 15. Partially treated Ecthyma Gangrenosum on the leg of a child

Community Based Management of Vitiligo

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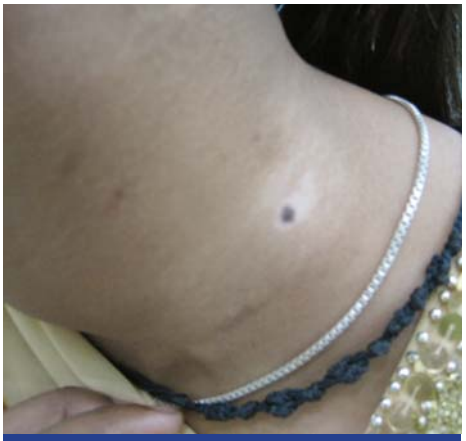

Fig 3. Halo Naevus

Introduction

Vitiligo is acquired or congenital complete, or partial, depigmentation of skin. In the hairy areas, about 25% of cases develop white hair. The depigmented macules (white patches) are of various sizes and shapes. The skin is otherwise mostly normal clinically. The patient usually does not report any physical symptoms, although there might occasionally be some itching. Some patients come for consultation after having used some home-made remedies or complementary medicines or improperly used modern medicines which result in inflammation. This may be acute with vesicles (fluid filled bumps) or chronic, where skin continues to remain thick and reddish (inflamed). This happens predominantly on sun exposed areas.

Vitiligo can develop at any age, but appears mostly in childhood and young adulthood, affecting both sexes equally. It has been classified into various types:

Broadly, vitiligo may be unilateral (i.e. affecting one side of the body) or bilateral (affecting both sides). Unilateral :

- Segmental - usually unilateral and involving a segment. (Fig 2)
- Halo Naevus - pigment loss occurs around naevi. Often associated with vitiligo elsewhere. (Fig 3)

Bilateral :

- Generalized - extensive and may involve any area (Fig 1)
- Acro-oro-facial - When only or mainly non hairy areas such as lips and hands, particularly finger /toes (often called 'lips and tips') are involved.

Chemical leukoderma is loss of pigment occurring in some susceptible individuals on contact with chemicals such as thiols, phenolic compounds, catechols and mercaptamines. Typically the areas in contact with the chemical lose pigment , but distant areas may also be affected. These chemicals produce depigmentation by a toxic effect on melanocytes or by inducing contact dermatitis. These chemicals are present in rubber gloves, adhesive glues (used in shoes, wrist bands, brassieres, and panties), electric cables etc. Indian Hindu women use "Bindi" on the forehead which is fixed with glue. It can occur around the mouth due to tooth powders/ pastes containing cinnamates. Flip flops used in rural communities as well as synthetic leather products like wallets can also cause chemical depigmentation. Rarely the nose rests of spectacles can lead to depigmentation. (Fig 5)

Pathogenesis:

It is necessary to understand the possible mechanisms leading to depigmentation in order to rationalize treatment.

1. Auto immunity[1]: Normally the immune system (defense system) of the new born learns to recognize between "self" and "non-self" while still in the mother's womb. In vitiligo, the 'memory' of the immune system is lost specifically towards melanocytes (the pigment forming cells). The system considers these cells as 'non-self' and therefore destroys them. Fortunately this 'loss of memory' is short lived and so the progress of disease stops. But this memory loss can recur. This is perhaps a very simplistic, yet scientific, way of explaining the genesis of vitiligo.
2. Increased susceptibility to oxidation stress (autocytotoxicity)
3. Direct destruction of melanocytes by chemicals.

Diagnosis:

There are macules which are partially or completely depigmented but otherwise the skin appears normal. The diagnosis in classic cases is fairly straight forward. However, in very fair skins, there can sometimes be some

Fig 1. Generalised Vitiligo

Fig 2. Segmental Vitiligo

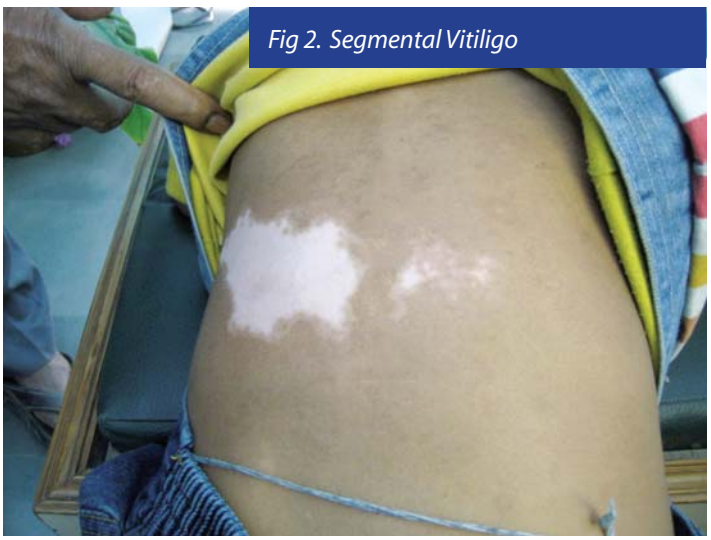

Fig 4. Inadvertent Excessive Sun Exposure

Fig 5. Chemical Depigmentation due to Spectacles

difficulty. If in doubt, a UVA lamp (Wood's lamp) can be used to enhance the contrast in the dark.

Other conditions that can most commonly be confused with vitiligo are pityriasis versicolor and leprosy.

Course of disease :

This is very variable. Very rarely some patients undergo spontaneous resolution. Typically it is a progressive disorder with halts, which may vary in duration or may be permanent. It may relapse after long periods of remission, with further areas of pigment loss. In general, progression rather than resolution is the norm[2].

Psycho-social impact:

It is a disease which has many myths attached to it especially in the developing world and in dark races. One has to fight and learn to live with vitiligo[3], although in practice this is often a challenge. The white blotches on dark background are immediately visible to others resulting in social isolation and emotional consequences. Society, particularly in South Asia, considers it infectious, a result of God's curse, a consequence of bad deeds in the previous birth etc. and so ostracizes the patient. Marriage becomes difficult, sometimes impossible. If hidden at the time of marriage, there is a higher rate of divorce or discordance in the couple. There is rejection by those around them[4]. Patients develop low self esteem, social anxiety and depression. Psychoeducation is important to make them and the family members understand that it is not primarily genetic, is non infectious and is of multifactorial etiology.

Patients and their families desperately seek treatment and are often mislead by quacks in the bargain. Most patients have gone through a variety of indigenous and unscientific treatment regimens resulting in irritation and blistering before consulting a specialist. In extreme cases, they may resort to tattooing and chemically burning the affected skin to hide the disease. (Fig 6)

Treatment:

The patient's detailed history must be elicited including the course of the disease and any previous treatments taken. The opportunity must also be taken to undress the patient and examine the entire body for extent of disease.

There are some guidelines available for the management of vitiligo[5], but these may not be practicable in community based management of vitiligo, where it is a highly stigmatized disease. The author suggests possible ways to improve this condition in resource- poor settings and in communities where there is a very high percentage of people living below poverty line.

Effective and affordable (to most patients) therapy includes topical and systemic psoralens (eg , 8-methoxypsoralen (MOP) -PUVASOL) with UVA from sunlight[6]. Conventional PUVA as used in dermatology centres use UVA emitting tubes which are neither available nor affordable in poor communities of the developing world.

This method has been in use for several millenia and has stood the test of time. It is the most effective option for community settings.

Topical 8-MOP lotion is available in 0.1% strength. It is diluted with spirit or water to 0.01% initially. The lotion is be applied carefully to the white patches (wiping it if it spreads on to normal skin). After half to one hour, it presumably reaches the basal cell layer. The area is then exposed to sunlight for 5 minutes and washed off. Washing off is important because if the lotion is left on the affected area, inadvertent sun exposure may lead to marked redness and blistering. If the palms and soles are affected, the exposure time can be increased up to 10 to 20 minutes. UVA levels in sunlight are very variable, although it is accepted that they are maximum from 10 am to 2 pm in most countries. The art of treatment is ensuring that adequate dose of UVA reaches the pigment forming cells. This can be roughly assessed by the degree of erythema persisting till next day. The best results are obtained if we can achieve and maintain light pink colour of the lesions. If it becomes red or raised/blistered, treatment should be discontinued till the redness disappears. On restarting treatment, the exposure time should be reduced and also the concentration of 8-MOP should be reduced by adding more alcohol/spirit. Treatment is generally repeated on alternate days.

This treatment is most suited for children or adults with a few small lesions. The patient needs detailed explanation to avoid damage from therapy. Topical psoralen should be avoided on the lips, except as a last resort and that too in greater dilutions in water.

Oral psoralens are useful in cases where the lesions are larger in size and/or more in number. The drug (8-MOP) is given in approximate doses of 0.6 to 0.8 mg /Kg body weight. The maximum concentration of the drug in the skin is attained after two hours and remains high for the next three hours. During this period the white patches must be exposed to sunlight, protecting the normal skin and eyes (specially the unaffected areas of the face) with a thick, tightly woven cloth. The period of exposure recommended is half an hour on each lesion. If the lesions are on both sides of the trunk, the patient may lie half an hour in the prone position and half an hour in supine position. The amount of sunlight exposure is to be adjusted according to the erythema that develops. A rough guide is to ask the patient to allow the lesions to become only upto light pink and look out for any more redness than this. During early morning or late evening, the sun exposure has to be for longer periods as the UVA is less. On the palms and soles, the superficial layers of epidermis are thick, so lesser amount of rays reach the melanin forming cells (melanocytes). The exposure has to be for more than double the time than on the other areas. Similarly on

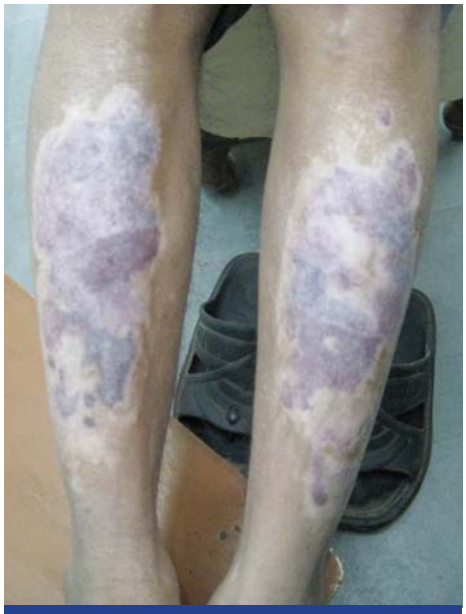

Fig 6. Damaged Vitiliginous Skin due to Tattooing and Chemical Burns

Community Based Management of Vitiligo *continued*

the lips, the exposure time should be less. It is important to emphasize that inadvertent exposure after the recommended time limit, often unavoidable in rural areas, must be prevented to avoid blistering. (Fig 4)

Sometimes one may use both oral and topical psoralens, each on alternate days. In combination therapy, the concentration of topical 8-MOP should be reduced still further. Such a combination is particularly useful for palms and soles.

Topical corticosteroids:

Mild to moderate topical steroids may be applied once or twice a day on the depigmented areas only. On the face and other areas with thin skin such as the axilla, groin, eyelids etc. only mild topical steroids are to be used. Even as monotherapy, it is sometimes effective to use topical steroids, especially if the lesions are two or three and of recent onset. Topical steroids can be safely and effectively used in combination with psoralens.

General measures:

Since most patients from poor communities have nutritional deficiencies, it is important to stress the role of a nutritious and balanced diet enriched with adequate vitamins and minerals.

Role of locally available herbs in management :

It is not unusual for doctors practicing in urban areas to not be able to realize the extent of economic deprivation in some rural communities. Food for subsistence can be a luxury and purchasing medication can be an impossibility. Another hindrance in some countries is that 8-MOP is not available without a doctor's prescription. Health workers in these situations therefore cannot help patients in the community. In these situations, we recommend the use of the original plants from which psoralens are extracted. These include *Psoralea corylifolia* (Bavachi) in South Asia and *Ammi majus* in North Africa.

The author and her colleagues have experience of using this herbal medicine. The seeds of Psoralea corylifolia are used in many rural communities in India. It is extremely cheap (approximately US$ 1.00 per Kg). One teaspoon of crushed seeds are boiled for 10 minutes and decanted. The paste is applied on the skin and the decanted extract is taken orally. After 2 to 3 hours the paste on the patch is removed and the areas exposed to sunlight as with 8-MOP. The results of this rather crude but sometimes only available methodology have been satisfactory in some deprived communities. The plant can be grown around the hut and in the fields.

It is important for doctors and other health care professions working in the field, to first realize the enormous impact this disease can have on a person's life. This should drive them to try and treat every patient with the available resources along with education of the community about the non infectiousness of this condition.

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Implementation of the 2010 WHO recommendations for ART initiation: will it change the prevalence and spectrum of HIV-related skin diseases?

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Introduction

HIV-infected patients commonly suffer with skin diseases which are common both before and after antiretroviral therapy (ART) initiation as HIV-related skin diseases occur because of both immunodeficiency as well as immune reconstitution. Furthermore, drug rashes are common in HIV-infected patients and antiretroviral (ARV) drugs are a common cause. Therefore, any change in recommendations for ART initiation and ART regimens will impact on the prevalence, spectrum and severity of HIV-related skin disorders.

Since the World Health Organization's (WHO) 2006 recommendations for ART initiation new evidence has emerged on when to initiate ART and optimal ART regimens in resource-limited settings. This led to the 2010 revised guidelines[1]. This article outlines its key recommendations and then discusses

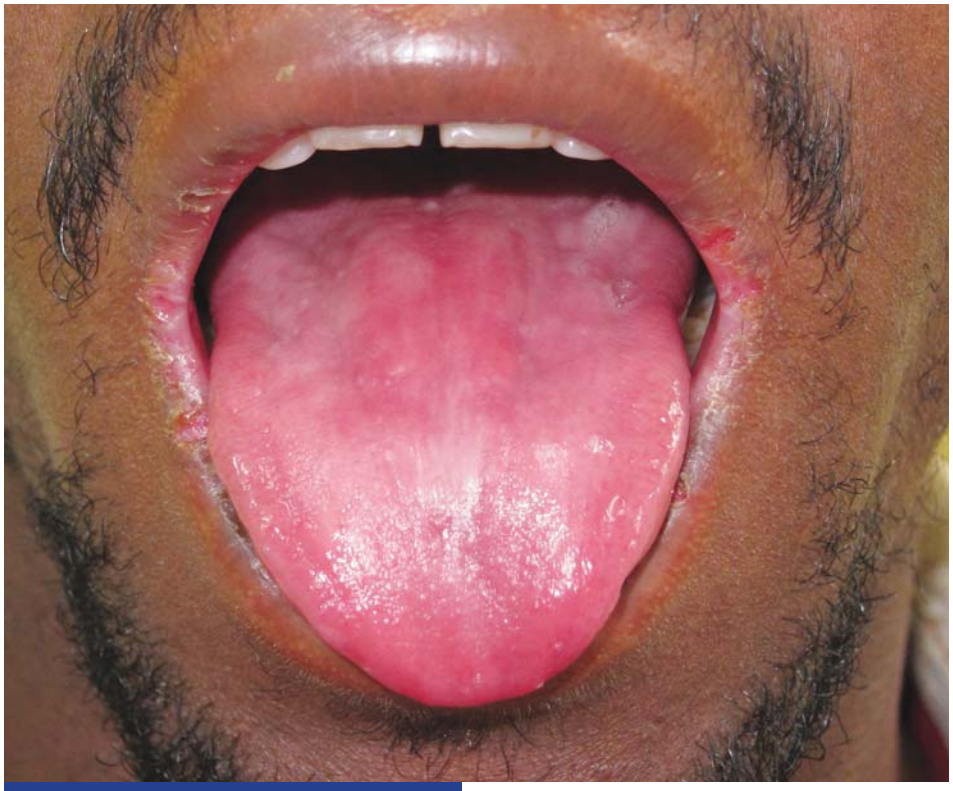

Fig 1. Candidal glossitis and cheilitis

how their implementation may change the burden of HIV-related skin diseases.

2010 WHO update of ART initiation in adults and adolescents

This recent update has introduced some significant recommendations which are summarized as follows:

- Earlier initiation of ART at a CD4 count of ≤ 350 cells/ mm^3 or for those with WHO clinical stage 3 or 4 (Table 1) irrespective of CD4 count and if CD4 testing is not available.
- Less toxic, simplified, fixed-dose first-line ART regimens have been recommended (Table 2) which avoids d4T (stavudine) as it is associated with toxicity as well as cosmetic disfigurement. WHO recommends that d4T should also be phased out of second-line ART regimens.
- ART initiation for all patients with TB co-infection irrespective of the CD4 count.
- ART initiation for all patients with active hepatitis B co-infection irrespective of the CD4 count.
- Strategic monitoring in order to determine the efficacy and toxicity of ART. In particular, the aim of monitoring is to be able to detect ART failure earlier than relying only on clinical monitoring.

Virological & immunological monitoring

After ART initiation the suppression of the viral load (VL) occurs sooner than a detectable increase in the CD4 count. Therefore, monitoring the VL (baseline pre-ART and post-ART) is an earlier indicator of ART efficacy than CD4 count monitoring. However, VLs are very expensive in most resource-poor regions of the world. If it is available the VL should be monitored every 6 months. However, if it not available or if it is too expensive WHO suggest that it is requested only when immunological failure with ART is suspected. An alternative to VL tests could be 'dried blood spot' (DBS) tests. These are not able to give an absolute VL count but are able to detect a decline in VL. They are a low cost option which has been successfully field-tested and the hope is that they will improve access to virological monitoring in resource-limited settings which cannot afford VLs[(2)].

WHO recommends regular CD4 count testing after HIV diagnosis in order to more effectively monitor HIV disease progression and therefore determine when a patient needs to start ART. After ART initiation the CD4 count should be done every 3-6 monthly in order to effectively monitor the patient's response to ART.

WHO recognises that in the absence of CD4 counts and VLs, ART can still be started although studies have shown that clinical monitoring alone is associated with AIDs-defining events and increased mortality.

Implementation of the 2010 WHO recommendations for ART initiation *continued*

The impact of the updated 2010 WHO recommendations for ART initiation

These new recommendations will mean that a significantly higher number of HIV-infected patients will be eligible for ART. This of course will lead to an increase in costs and manpower required to deliver ART for each country. WHO recognises that not all of these recommendations can be implemented in all countries because of limited resources. Therefore, WHO advises that each country should adapt these recommendations to suit its own circumstances and that national ART advisory committees should be responsible for this. Ethiopia, for example, has still not been able to implement the 2010 WHO recommendations. Its current national guidelines are as follows:

ART initiation in Ethiopia is recommended:

- For WHO clinical stage 4 disease
- CD4 < 200 cells/ mm^3 irrespective of clinical stage
- Stage 3 & CD4 ≤ 350 cells/ mm^3

Fig 2. Chronic genital herpes ulceration, condyloma acuminata and molluscum contagiosum

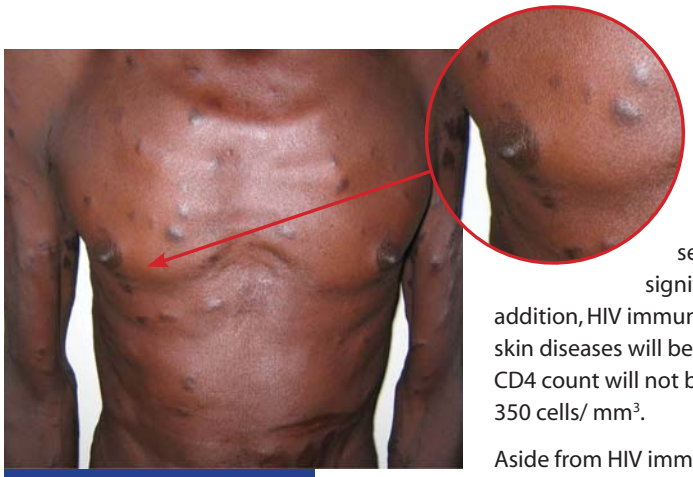

Fig 3. Kaposi's sarcoma

This means that many HIV-infected patients in Ethiopia will still not benefit from starting ART at the 2010 recommended higher CD4 count of ≤ 350 cells/ mm^3 but will have to wait until their CD4 count falls below 200 cells/ mm^3, which is in line with previous 2006 WHO guidelines. This is also true for many other resource-poor regions of the world where they are presently unable to offer ART to eligible HIV-infected patients under the current WHO recommended criteria of CD4 ≤ 350 cells/ mm^3.

Cotrimoxazole prophylaxis

WHO 2010 guidelines advocate cotrimoxazole prophylaxis recommending it for all HIV-infected patients with CD4 count ≤ 350 cells/ mm^3 in resource-limited settings where bacterial infection and malaria are common and for all stage 3 and 4 conditions irrespective of CD4. If CD4 counts are not available the guidelines recommend cotrimoxazole for all HIV-infected patients with symptomatic stage 2, 3 or 4 diseases.

What will be the impact on HIV-related skin disease if ART is initiated at a higher CD4 count?

HIV immunodeficiency-related skin diseases are common, particularly at lower CD4 counts. They are also more severe and atypical and they respond poorly to treatment at lower CD4 counts. Studies have demonstrated that the majority of HIV immunosuppression-associated skin diseases occur at low CD4 counts. For example, chronic ulcerative herpes simplex, papular pruritic eruption and eosinophilic folliculitis commonly occur at CD4 counts less than 100 cells/ mm^3. Extensive molluscum contagiosum and severe and widespread seborrhoeic dermatitis are also more common at CD4 counts less than 200 cells/ mm^3 [3]. Therefore, if the recommendations of these recent WHO guidelines are implemented and ART is started at higher CD4 counts in resource-poor settings, the prevalence of HIV immunosuppression-related skin diseases seen by clinicians should significantly decrease. In addition, HIV immunosuppression-related skin diseases will be easier to treat as the CD4 count will not be allowed to fall below 350 cells/ mm^3.

Aside from HIV immunodeficiency-related skin diseases declining with the implementation of ART at higher CD4 counts, the prevalence of HIV Immune Reconstitution Syndrome (IRS)-associated skin diseases is also expected to decline. This is because the major risk factor for IRS-associated diseases is a low CD4 count, usually less than 200 cells/ mm^3 [4]. IRS-associated skin diseases are rarely fatal but can be associated with non-adherence if patients feel dissatisfied that their ART has lead to a worsening of existing skin disease or the development of a new skin disease.

HIV-infected patients have a higher incidence of drug rashes compared to non-HIV-infected immunocompetent patients. Drug rashes are a significant problem in HIV-infected patients both before and after ART initiation. Before ART initiation the risk of drug rashes increases as the CD4 count falls. Studies have shown that the average CD4 count of HIV-infected patients getting drug reactions is approximately 300 cells/ mm^3 [3]. Therefore, the implementation of these recent recommendations is unlikely to change the prevalence of drug reactions in pre-ART patients. Studies have shown that the risk of drug reactions increases after ART initiation[5]. There are two main reasons for this: drug reactions usually require some degree of immunological capacity which improves after ART initiation; and ARVs themselves are commonly associated with drug rashes, particularly nevirapine and efavirenz which are both included in the first-line WHO-recommended ART regimens. In addition, cotrimoxazole prophylaxis often causes drug rashes as well. Therefore, the 2010 guidelines are unlikely to decrease the risk of drug reactions after ART is initiated.

Stavudine is associated with a very high risk of disfiguring facial lipoatrophy which is often irreversible even after drug withdrawal. Unfortunately treatment options for lipoatrophy such as autologous fat transplantation and facial fillers are not available in resource-poor settings. Because of the risks of stavudine-associated toxicity the 2010 guidelines have recommended replacing stavudine with tenofovir or zidovudine in the first-line ART regimen. However, tenofovir and zidovudine are both more expensive than stavudine and require more laboratory monitoring. Therefore, stavudine continues to be widely used in both first-line and second-line ART regimens in resource-poor settings. Therefore, in order to minimize the risks of toxicity the 2010 guidelines suggest that stavudine should only be given at the lower dose of 30mg twice daily irrespective of body weight.

In conclusion, the implementation of the recommendations proposed in the recently revised WHO guidelines for ART initiation will have an impact on the prevalence, spectrum and severity of HIV-related skin diseases. In general, there should be a significant decrease in the burden of HIV-related skin diseases. At the 2011 6th International AIDs Society Conference in Rome, Italy, the challenges in scaling up universal access to ART based on the 2010 WHO recommendations were discussed *[Lancet editorial, 2011]*[6].

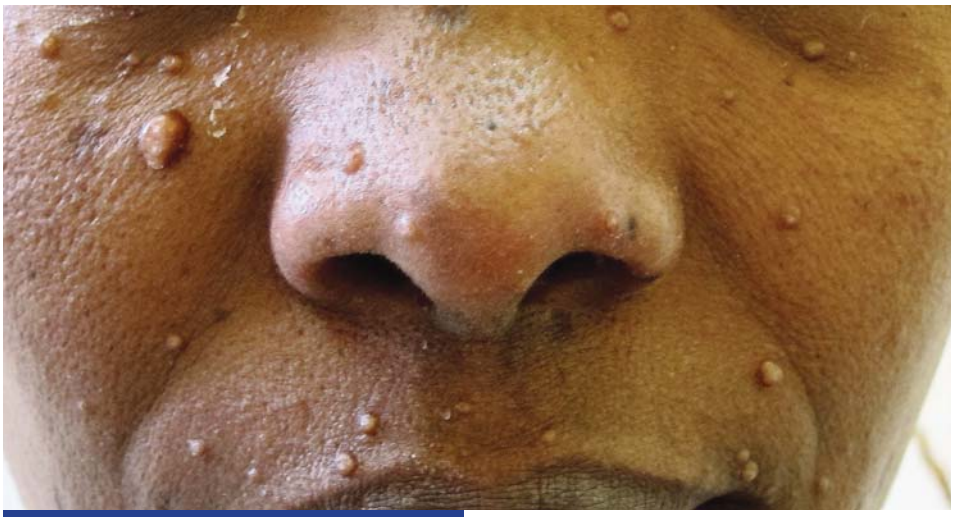

Fig 4. Disseminated cryptococcosis

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Table 1.
WHO clinical stage 3 and 4 mucocutaneous diseases

Stage 3
Oral candidiasis (Figure 1)
Oral hairy leukoplakia
Acute necrotizing stomatitis, gingivitis or periodontitis
Stage 4
Chronic orolabial, genital or anorectal herpes simplex infection > 1 month duration (Figure 2)
Kaposi's sarcoma (Figure 3)
Any disseminated endemic mycosis eg. extra-pulmonary cryptococcosis, histoplasmosis, coccidioidomycosis, penicilliosis (Figure 4)

Implementation of the 2010 WHO recommendations for ART initiation *continued*

Table 2.
WHO 2010 recommended ART regimens

2x NRTI: TDF (tenofovir) or AZT (zidovudine) + 3TC (lamivudine)
FTC (emtricitabine) + + 1x NNRTI: NVP (nevirapine) or efavirenz (EVF)