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The International Skin Care Nursing Group at the launch of SADNA

Launch of the South African Dermatology Nursing Association

This last year has seen the formation by a group of dermatology nurses to establish the South African Dermatology Nurses Association (SADNA).

SADNA was formed as an independent professional association for dermatology nurses throughout South Africa. It was an historic event for dermatology nurses who had dedicated so much to the care of people with skin disorders.

The aims of the SADNA are to:

- Promote the development of the highest standard of care for the patient receiving dermatological care
- Promote the development and recognition of the nurse's role in dermatology for the benefit of the patient
- Promote and support education and research
- Provide a source of expertise for nurses facing clinical and managerial challenges in the field of dermatology
- Provide a platform for the dissemination of developments and knowledge in the field of dermatology nursing

Our first activity as a group was to hold a pre congress dermatology nursing workshop at the 66th Annual Congress of the Dermatology Society of South Africa in Cape Town. Support from my fellow board members of the International Skin Care Nursing group added an international flavour to the workshop. Delegates were exposed to

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Contents

Pat Kelly (Dermatology Nurse Tutor) with dermatology nursing students on a field trip

a broader outlook on dermatology patient education and activities taking place outside of South Africa.

South Africa is a developing country, one of 54 Countries in Africa. The challenge in many of these countries is to find effective ways to provide dermatological care to the majority of people living in communities and rural areas. In South Africa with a population of 52.4 million there is 1 dermatologist to 300,000 of the population. A solution to the shortage of available dermatologists was to train professional nurses to diagnose and treat common skin conditions and to be able to recognise those patients needing specialist attention. On return to their workplace they would then establish a community day care centre.

The only dermatology nursing course on the African continent is offered by the University of Cape Town together with the Division of Dermatology at Groote Schuur Hospital. To date we have trained 157 professional nurses not only from South Africa but from several other countries in Africa. Many of these nurses have established community dermatology services in their areas. SADNA keeps in touch with many of these graduates not only replying to queries but also getting feedback and statistics which influence changes in the course curriculum.

Activities planned

- National conferences- promoting educational and development opportunities for nurses country wide
- Publications- to highlight the contributions of nurses to skin care and stimulating development
- Designing a course outlining core dermatology nursing skin care and wound care topics primarily for nurses in various settings
- Working with other groups to develop, evaluate and promote evidence - based practice in skin care nursing e.g. Best practice in Emollient therapy
- Developing and maintaining a website which will give access to skin care resources for nurses and other health care professionals
- Promote cross- professional collaboration through our role with other key organisations.

South African Dermatology Nurse Speakers at ISD Congress in Durban SA
L – R Judy Wallace (Vice President SADNA) Lynne Kennedy(Secretary SADNA) Johanna Stevens (Speaker) Pat Kelly (President SADNA) Delena Cloete (Speaker)

Mycetoma. A Review

The Mycetoma Research Centre was established in 1991 at Soba University Hospital, Khartoum, to provide integrated medical care for mycetoma patients and to foster research. The following review is a practical account, based on the author's unparalleled experience, and supported by relevant literature.

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Background

Mycetoma is a progressive and disfiguring tropical disease which has many serious medical and socio-economic impacts on patients, community and health authorities. There are two main types;

Eumycetoma is caused by true fungi, most commonly *Madurella mycetomatis;* **Actinomycetoma** is caused by bacteria, chiefly *Streptomyces somaliensis* and *Nocardia brasiliensis*[1,2]

Clinical features include the classical triad of a painless subcutaneous mass, sinus formation and purulent or seropurulent discharge containing grains. [3] It usually spreads to involve the skin and the deep structures, resulting in destruction, deformity and loss of function. It is occasionally fatal. 82 % of cases affect the hand or foot. In endemic areas other parts of the body may be involved such as the knee, arm, leg, back, head and neck, thigh and perineum. (Figs. 1, 2) No age is exempt, however, mycetoma occurs more frequently in young adult men aged 20–40 years and in one series almost 30% were young students. [4]

Epidemiology

The true incidence and prevalence of mycetoma world-wide is not precisely known. (reviewed in ref 5) Most of the reported data relate to hospital patients with advanced disease [6]. This is attributed to the nature of mycetoma which is usually painless and slowly progressive. Also, most patients present at a late stage because of poor health education, lack of health facilities and financial constraints. [7]

Mycetoma is endemic in many tropical and subtropical regions and prevails in the "mycetoma belt", which stretches in a band from the latitudes of 15°S to 30°N.[1,2] It extends from Sudan, Somalia and Senegal to Yemen, India, Mexico, Venezuela, Columbia, Argentina, and a few other countries. Although the African continent seems to bear the highest burden and prevalence of the disease. [3] it has been extensively reported from India. [9] Mycetoma has been reported in many temperate regions as well [10], with a few reports from the USA, Sri Lanka, Germany, Egypt, Turkey, Philippines, Japan, Lebanon, Thailand, Saudi Arabia, Tunisia and Iran.[11]

Globally, only two large epidemiological studies have been performed to estimate the disease prevalence. In these studies underestimated prevalence was calculated. These studies were performed by Abbott in Sudan during the period 1952–1955 and by Lopez Martinez and colleagues in Mexico between 1956 and 1985. [12.13] In Abbott's study, 1231 mycetoma patients were admitted to hospitals throughout the country over a period of 2.5 years and the estimated prevalence was 4.6 per 100,000 inhabitants. Lopez Martinez reported on 2105 mycetoma cases from 14 dermatological centers throughout Mexico over a period of 30 years and estimated a prevalence of 0.6 per 100,000 inhabitants. This grossly underestimated incidence is comparable to those from other "neglected" tropical infections such as Buruli ulcer, African trypanosomiasis, dracunculiasis and leprosy. Surprisingly, Mycetoma is not on most of the Neglected Tropical Diseases lists.

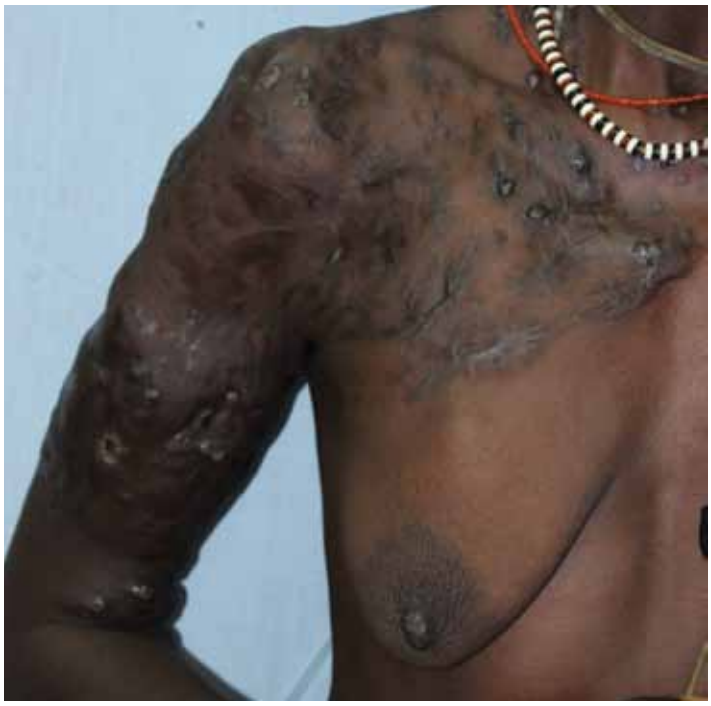

FIG. 1: Massive eumycetoma involving the right shoulder region, upper chest wall and right upper arm with multiple sinuses, discharge and black grains.

Currently there is no prevention or control programme in mycetoma because there is no well recognized prevention or treatment. The route of infection is unknown. The prevailing theory is that the causative organism is inoculated into the

Continued overleaf…

FIG. 2: Extensive back and gluteal actinomycetoma.

subcutaneous tissue by contaminated plant material, e.g. an acacia thorn, although the incubation period is unclear. Many believe there is an intermediate host, as yet unidentified. The disease is not contagious between humans or from animal to human.[2,3,4,]

Diagnosis

The diagnosis is clinical, based on history and examination, supported by histopathology. The causative organism can be identified by culture of discharged grains, supported by molecular techniques such as PCR, serodiagnosis using ELISA and counter immune-electrophoresis.

Current diagnostic tools for establishing the extent of disease include imaging techniques such as radiography, ultrasonography, CT and MRI,[14-21] (Figs 3,4,5)

Unfortunately, most of these techniques are not available in majority of mycetoma endemic regions. There is an urgent need for simple, affordable and field-friendly diagnostic tests, particularly in resource-poor endemic areas.

The **differential diagnosis** of mycetoma includes many soft tissue masses such as Kaposi's sarcoma, malignant melanoma and foreign body granuloma. Radiologically, osteogenic sarcoma, chronic osteomyelitis, osteoclastoma, bone cysts, and tuberculous osteitis look very similar to mycetoma.[1,4,5]

Management

The management of this distressing and devastating disease is disappointing. Specific treatment depends on the aetiological agent, the site and extent of the disease. Until recently, the only available cure for mycetoma was amputation or multiple mutilating disfiguring surgical excisions. However, a combination of medical treatment in the form of antifungals for the eumycetoma and antibiotics and antimicrobial agents for actinomycetoma and surgical treatment is now considered the best approach.

Reports on successful medical treatment of **eumycetoma** are scarce and inadequate. Even now, treatment is based on personal clinical experience and on the results of sporadic case reports, rather than randomized controlled clinical trials.

Various antifungal agents, including itraconazole, ketoconazole and ravuconazole, have been tried with little success. This is perhaps surprising, as the causative agents are low-grade infective organisms which are sensitive to several azole drugs *in vitro* and their eradication would be predicted by the administration of safe systemic antifungal drugs [22,23]. The grains, melanin and cement substance are postulated to protect the organisms against these antifungal agents. [24,25]. Currently, itraconazole in a dose of 400 mg/day combined with surgical excisions is the preferred treatment.[27]

In contrast, **actinomycetoma** is much more amenable to medical treatment with antibiotics and other chemotherapeutic agents. Combined drug therapy is always preferred to a single drug to avoid drug resistance and for disease eradication. Cure is possible, although a prolonged period of treatment is needed. Although several patients respond to medical treatment alone, combined medical and surgical treatment is still advocated. This accelerates healing and reduces the chance of relapse.[27] Currently the treatment of choice is 15 mg/kg/day amikacin sulphate and cotrimoxazole 980 mg twice a day However, amikacin sulphate is ototoxic and nephrotoxic.[27] The treatment is of a variable duration and may continue for 2-3 years with a mean of 18 months.

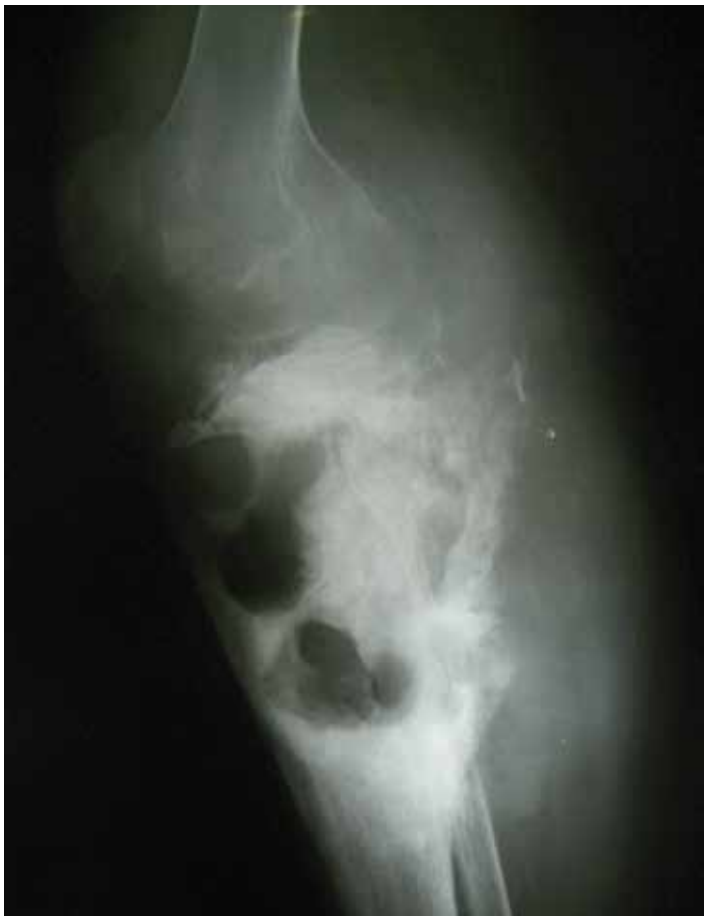

FIG. 3: X-Ray foot showing soft tissue mass, periosteal reaction and multiple cavities typical of eumycetoma.

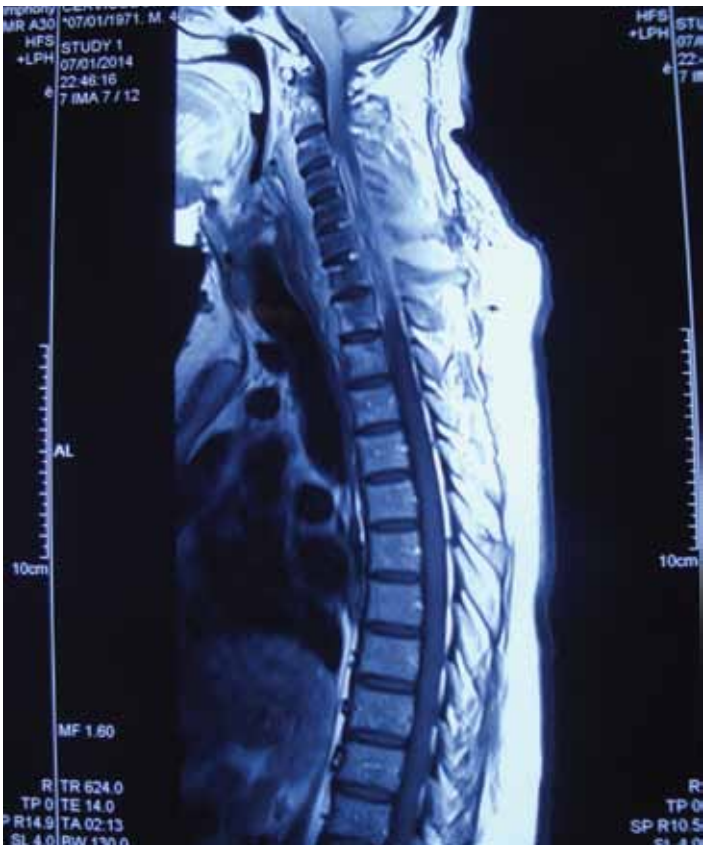

FIG. 4: MRI vertebral column showing soft tissue involvement, cavities and dot in sign typical appearance of mycetoma.

Medical treatment for both types of mycetoma must continue until the patient is clinically, radiologically, ultrasonically and cytologically cured. We find that recurrence is more common after an incomplete or irregular course of medical treatment. Poor compliance increases the risk of drug resistance. In our hands, the recurrence rate from surgery as monotherapy varies from 25 to 50%.[28]

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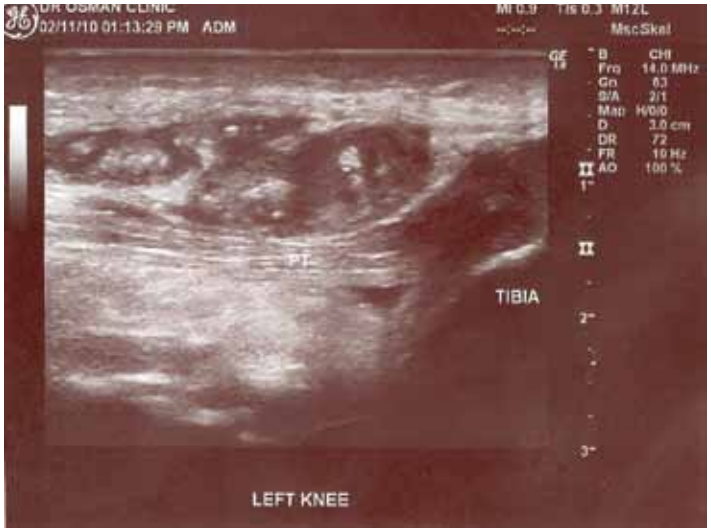

FIG. 5: Typical ultrasonic appearance of mycetoma.

A case of Madura Foot (Mycetoma) from Chaaria, N.Kenya

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The occurrence of Madura foot is sporadic in Chaaria, although not very rare; and normally it only affects pastoralist patients coming from the north of the country.

The clinical presentation is normally of painless swellings of the lower limbs (more rarely of hands, back and head). The swelling is chronic and after several years nodules form in the skin and break down to form discharging sinuses from which pus and fungal grains emerge. Normally there are no systemic symptoms unless secondary infection occurs.

The progress is slow but relentless. It may be a life-long condition.

The diagnosis is normally clinical.

For confirmation purposes we usually do a biopsy with collection of the grains, and sending material for cytology and histopathology. Normally we also check X-Rays to rule out bone erosions and osteomyelitis.

Most of the cases we have diagnosed in Chaaria are caused by the Eumycete Madurella mycetomatis. The treatment has generally been disappointing, although, following some international guidelines, we always try long term medication: we use ketoconazole 200 mg twice daily for up to 12 months.

In the frequent case of treatment failure we attempt surgical excision of the mycetoma, taking care of being very wide and not rupturing the capsule that often surrounds the infection. If there are few nodules sometimes we are successful in this way, but in larger infections like the one shown in today's pictures, relapse is almost the rule. In bigger and infected lesions, above all when the limb has been rendered useless, we have done amputations, but sadly even after this destructive procedure, relapse is likely.

There is evidence that the organisms are spread from the environment via a penetrating injury such as a thorn prick. The fungal causes of mycetoma have been isolated from plants and plant debris, while actinomycetes have been isolated from soil.

The condition is more common in males but the photos we present refer to a young woman of Borana tribe who is at the moment on medical treatment.

Further Reading

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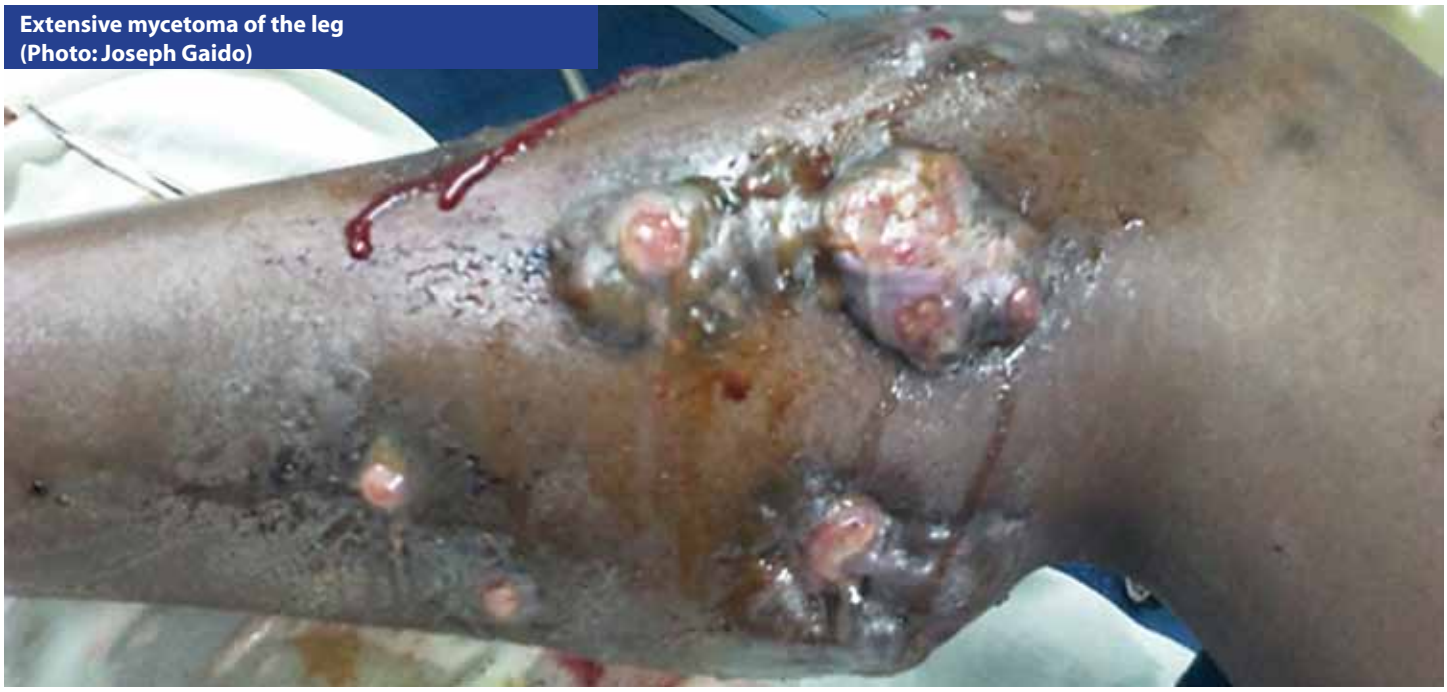
Extensive mycetoma of the leg (Photo: Joseph Gaido)



JOURNAL CLUB

The Elimination of Leprosy

Lockwood DNJ, Shetty V , Gerson OP Hazards of setting targets to eliminate disease: lessons from the leprosy elimination campaign. *Br Med J* 2014;348:g1136

The idea of controlling or eliminating any disease figures large in international public health measures for tropical and dermatological conditions. Yet it is a concept which, however desirable, needs to be thought through carefully and with clarity as the process involves a detailed understanding of what is, and what is not , known about the target disease. Nowhere has this issue become so important as in the current debate over international policy for leprosy control. This has been analysed recently in this paper from Professor Diana Lockwood and colleagues at the London School of Hygiene and Tropical Medicine .

In this study the authors point out some of the pitfalls in defining an elimination target, in this case that proposed by the World Health Organisation, as it can have unintended consequences such as an increased use of voluntary and, in many cases, incomplete reporting systems or the exclusion of cases for non-essential reasons in order to meet a notional target. The core implication discussed in her paper is that by striving for fixed and difficult to reach time lines and targets declared prematurely , research and teaching in leprosy has declined in all countries and it has left a gap in our ability to recognise and treat the new cases which still exist. For instance the International Journal of Leprosy ceased publication in 2005 even though it was still expressing doubts as to the likely efficacy of a policy for elimination. The initial target to eliminate leprosy by the year 2000 used a goal proposed by WHO to achieve a prevalence of less than one case per 10 000 population at a global level but this has now been modified, although countries and WHO still report leprosy rates. This level of infection was always difficult to achieve given the long incubation period which meant that elimination, if achievable at all, was would take longer.

The authors argue that the reduction in case numbers of leprosy, which all aspire to, is not aided by setting a fixed goal. In their words ."A target to eliminate should be set only if it is realistic. The following conditions are needed: straightforward diagnosis, effective treatment, low transmissibility, and ability to differentiate between current and past infection. Of the diseases listed for elimination on the WHO roadmap only rabies in Latin America fulfils these conditions. When it was clear that leprosy transmission continued in many countries the appropriate response should have been to redefine the campaign rather than cling on to it. It is important to learn the lessons from earlier elimination programmes. Targets need to be evidence based. Like a battle strategy, they need to be reviewed regularly and amended when inappropriate"

One should consider the old adage –" out of sight, out of mind" - as this indeed appears to have been a consequence of premature declaration of the elimination of an ancient disease which was showing slow but sure steps in case reduction. It is a pity that the aspiration to create similar elimination targets continues as other diseases which those interested in the care of skin patients may well encounter, such as onchocerciasis, Buruli ulcer and yaws may well meet a similar fate.

The message for us is to continue to teach the recognition and management of these important diseases.

PROF R J HAY

Bust of Dr Armauer Hansen, discoverer of the leprosy bacillus, Leprosy Research Laboratory, ALERT Hospital, Ethiopia (Photo: Chris Lovell).

Podoconiosis
what is it and what can we do about it?

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What is podoconiosis?

Podoconiosis is a type of lower leg swelling (lymphoedema, which in advanced cases progresses to elephantiasis) found in highland areas of the tropics. It has often been confused with lymphatic filariasis (LF), but there are several ways of distinguishing the two conditions (see Table 1). Podoconiosis develops in people (predominantly farmers) exposed over many years to irritant red clay soils because they do not wear shoes[1]. These soils are found in highland tropical areas, where ancient volcanic deposits have weathered at high altitude (over 1000m) under conditions of heavy rainfall (over 1000mm/year). Podoconiosis is therefore associated with high elevation, whereas LF is found at lower altitudes at which transmission of the parasite by mosquitoes can occur. Countries with a high burden of podoconiosis include Ethiopia, Uganda, DR Congo, Rwanda, and Cameroon. Further distinction between podoconiosis and LF can be achieved through the patient's account of early symptoms. In podoconiosis, symptoms (aching, burning) are almost always reported from the foot first, and the swelling progresses from the foot slowly up the lower leg, only rarely reaching above the knee (Figure 1). This is in contrast to patients' reports in LF, where symptoms frequently originate in the groin, and swelling may be noticed anywhere in the leg, often above the knee. Examination of midnight blood for microfilaria, or use of a rapid antigen test (Binax™) will help exclude LF if there is doubt. Other diagnoses that should be considered include leprosy lymphoedema, onchocerciasis lymphoedema, granuloma inguinale, lymphogranuloma venereum and endemic Kaposi's sarcoma.

Studies have ruled out bacteria, viruses and parasites as a cause of podoconiosis. It is thought to be a geochemical disease on the basis of the identification of mineral crystals within the lymph tissues of patients, but the precise trigger of the disease is unknown. There is now strong evidence of underlying genetic susceptibility[2], and endemic communities are usually aware that podoconiosis clusters in families.

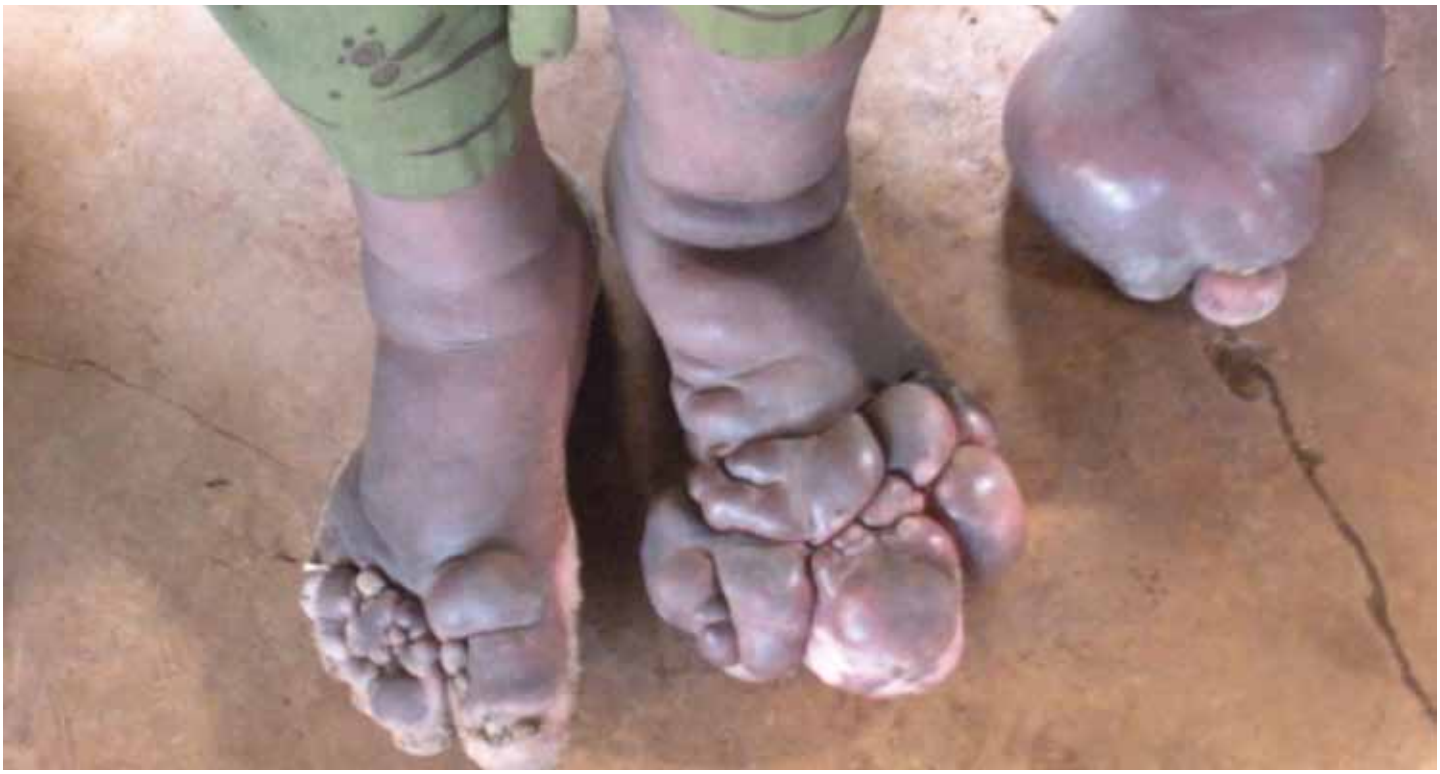

FIG 1: Bilateral, asymmetric, below-knee swelling with mossy changes and nodules.

FIG 2. Foot hygiene is a key element of podoconiosis management

CHARACTERISTIC	PODOCONIOSIS	LYMPHATIC FILARIASIS
Area of residence	>1500 m above sea level	<1000 m above sea level
Mean age of onset	10–20 years	25–30 years
Relation to natural history	Initial symptom	Late complication
Site of first symptom	Toes and foot	Any part of limb except foot
Local lymphadenitis	Follows swelling of limb	Precedes swelling of limb
Typical site of swelling	Distal, below knee	Above and below knee

TABLE 1: Characteristics used to distinguish podoconiosis and lymphatic filariasis

Recent work has detailed the enormous economic burden of podoconiosis on affected communities. In a southern Ethiopian zone of 1.5 million inhabitants, where the prevalence of podoconiosis is known to be 5.4%, the overall cost of podoconiosis was estimated to be in excess of US$16 million per year. In this zone, where the average income is less than US$100 per year, the direct costs to a patient are US$143 per year[3]. Individuals with podoconiosis are highly stigmatized. They may be excluded from school, rejected by their family, barred from social and religious gatherings, and banned from marriage to any unaffected individual[4]. Siblings of affected individuals are also frequently barred from marriage into unaffected families

What can we do about it?

Prevention of podoconiosis includes measures to avoid contact with irritant soil through regular washing and use of protective shoes. Programs are in place in several podoconiosis-endemic areas to distribute shoes to children with the aim of preventing disease in the future. Recent evidence suggests that the risk of disease is lower among people who live in houses with covered rather than bare earth floors, so this may be another prevention strategy.

Treatment follows the general principles of lower limb lymphoedema management, requiring daily foot washing, use of a simple emollient, bandaging, socks and shoes, and exercises to improve lymph flow. These measures can result in clinical and quality of life improvements[5]. A podoconiosis follow-up clinic should aim to provide:

Assessment. Record the following patient details: name, contact address, sex, age, occupation, number of family members, affected family members and age of onset of condition. For each leg separately, record clinical stage (see Box 1 overleaf), presence of moss, wounds or infection, greatest circumference of leg below knee, and presence/absence of acute attack (see Box 2 overleaf).

Training in foot hygiene. Demonstration of soaking both feet in a plastic basin with water and locally-available disinfectant, washing with soap, rinsing with clean water, drying and rubbing in oil or Whitfield ointment to improve the barrier function of the skin (Figure 2). Arrangements for water supply and disposal at the clinic site may have to be made.

Training in bandaging. Patients with more swollen legs (often with softer skin, hence called 'water bag' type) will benefit from careful use of short stretch bandages. Demonstrate how to apply the bandage while the leg is elevated, from the toes to 10cm above the upper limit of swelling, overlapping by half the width of the bandage each turn (Figure 3). Each patient will need at least 2 bandages for each affected leg, so he or she can wash one set of

Continued overleaf…

Box 1. Podoconiosis Staging Sheet: Description For Health Professionals

The stages represent severity of disease, and do not necessarily represent the disease process: it is possible, for example, for an individual to have stage 5 disease but never to have had above-knee swelling. The following terms are used in the descriptions:

- Dermal nodules: elevated, non-translucent lesions >0.5cm diameter, with width approximately equal to length.
- Dermal ridges: elevated lesions >0.5cm width, with length greater than width
- Dermal bands: palpable, but non-elevated ridges

Mossy changes: round or fusiform, *either* fluid filled (and hence translucent) lesions, *or* papillomatous hyperkeratotic horny lesions giving the skin surface a rough velvet-like appearance.

STAGE 1

Swelling reversible overnight.

The swelling is not present when the patient first gets up in the morning.

Changes such as hyperpigmentation and nail dystrophy are unusual, but may be seen. The swelling is usually confined beneath the ankle.

STAGE 2

Below-knee swelling that is not completely reversible overnight; if present, knobs/bumps are below the ankle ONLY.

Persistent swelling that does not reach above the knee. If present, knobs or bumps do not extend beyond the ankle.

The 'knobs or bumps' may take the form of dermal nodules, ridges or bands. Tourniquet-like effects may be observed at this stage or any subsequent stage, depending on the position of dermal ridges and nodules in relation to joints.

Mossy changes may be apparent, but their presence depends on a range of factors including the use of plastic footwear. Interdigital maceration and hyperpigmentation are often present at this stage, and nail dystrophy almost always present.

STAGE 3

Below-knee swelling that is not completely reversible overnight; knobs/bumps present above the ankle.

Persistent swelling that does not reach above the knee. *Dermal nodules, ridges or bands are seen or felt above the ankle. Tourniquet-like effects are frequently observed at this stage. Any of the other changes mentioned for Stage 2 may also be present.*

STAGE 4

Above-knee swelling that is not completely reversible overnight; knobs/bumps present at any location.

Persistent swelling that is present above the knee.

Any of the other changes mentioned for Stage 2 may also be present. In addition, signs of lymphectasia may be apparent, particularly on the thigh.

STAGE 5

Joint fixation; swelling at any place in the foot or leg.

The ankle or interphalangeal joints becomes fixed and difficult to flex or dorsiflex. This may be accompanied by adhesion and fusion of the toe web spaces, making the toes appear short or indistinct. Sensation is preserved. *X-rays show tuft resorption and loss of bone density.*

bandages while using the other.

Explanation of the need for socks and shoes. Clean socks and closed shoes are vital in preventing further exposure to irritant soil and for protecting the swollen leg. Some patients will be able to afford these themselves. If the patient cannot afford to buy shoes, but demonstrates commitment to self-treatment, they may be considered for subsidized footwear. Shoes large enough for patients with extensive swelling may not be available locally, so establishing a shoe workshop (staffed by treated patients) may be the best way to provide shoes and jobs for patients.

Demonstration of elevation and movement. Encourage the patient to perform toe points, ankle circles and calf raises 2-3 times per day. Elevate the leg whenever possible by raising the foot end of the bed or resting the foot on a stool while sitting. Both these will assist lymph return.

Nodulectomy. Discrete nodules that are preventing the patient from wearing shoes may be removed under local anaesthetic. More extensive surgery is not recommended.

Exploration of avenues of other forms of support. This may include individual counselling, support through patient-led groups or patient associations, vocational training (including shoe-making) and micro-credit schemes and spiritual support.

Patient-led, community-based treatment. Podoconiosis is relatively simple to diagnose in endemic areas[6], and to manage. Much of the management of podoconiosis can therefore be decentralised into the community. Experience from northern Ethiopia suggests that engagement of the community through 'Community Conversations' is essential groundwork for decentralised management. Community Conversations are voluntary discussions held bi-monthly for up to six months to facilitate the process of change. Typically, 'master trainers' train local trainers (often podoconiosis project personnel), who in turn train facilitators to lead Community Conversations with other community members. Once a Community Conversation group has met for approximately six months, they become responsible for

Box 2. Acute attacks.

Acute attacks (acute dermatolymphangioadenitis) are highly disabling episodes of fever, pain and increased warmth and swelling of the affected legs. These attacks often last 4-5 days, and patients are severely incapacitated by them. They are managed using anti-pyretics, analgesics (paracetamol or ibuprofen, or equivalents) and rest.

forming a Community Action Group to coordinate and lead the implementation of the Community Conversation Action Plan.

The Action Plan commonly includes formation of a group of motivated self-treated patients, to assist other patients with treatment and to act as ambassadors in their communities, explaining the cause of the disease and breaking down stigma by demonstrating that it can be both prevented and treated. Expert patients can enable 'task-shifting', promoting good practice among patients so that the burden on health professionals is diminished. Monthly monitoring of Patient Led Groups and quarterly evaluation is recommended. Another common activity of the Community Action Group is the development of a school health program focussing on the disease awareness and prevention among students. School health programs may evolve into sites of shoe distribution for disease prevention.

Summary

Although podoconiosis has been under-recognised for many decades, the experience of a number of projects within Ethiopia suggests that the disease is one that can be relatively easily controlled given dedicated community mobilisation. A national forum, the National Podoconiosis Action Network (NaPAN - http://www.napanethiopia.org/) has been developed to coordinate efforts against podoconiosis in Ethiopia, and a global initiative (Footwork – http://www.podo.org) to raise awareness of and coordinate partnerships against podoconiosis at international level.

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FIG 3: Demonstrating use of short stretch bandages

FURTHER READING

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An international dermatopathology meeting is to be held at the Regional Dermatology training Centre, Moshi, Tanzania, on 12-13 January. For further information about this meeting and the RDTC meeting following it (14-16 January), please contact; **Dr Helmut Beltraminelli, MD Department of Dermatology, Bern University Hospital, 3010 Bern, Switzerland, Email: helmut.beltraminelli@insel.ch**

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