TRADITIONAL WOUND DRESSINGS

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Introduction

This article will assess the published evidence for some traditional remedies; evidence for use of honey was covered in a previous article (Cox N H, *J Comm Dermatol* 2006; **3**: 20-22). The present discussion will include comments on saline, as well as fruit and vegetable agents, such as banana leaf and potato peel.

Many plants contain chemicals that are also used as wound healing agents, but most of these are in the form of alcoholic (or sometimes aqueous) extracts, rather than being applied as intact plant material. Some, especially those that are applied directly (e.g., as an intact leaf, or as a crushed pulp) are discussed briefly here. One issue that may apply generally to plants is that several botanical polysaccharides activate macrophage immune responses leading to several effects, including enhanced wound healing.[1] Another non-specific issue that may explain benefits on wounds from freshly pulped plant materials is that 'wounding' plants causes them to generate hydrogen peroxide.[2] This chemical is used medically in

A banana leaf prepared for use as a natural non-stick dressing
Photo: Dr S Walton, Hull, UK

various proprietary topical creams for its antimicrobial activity, and may be a further non-specific way in which plants work in wound healing. As in humans, plants also produce cationic peptides that have antimicrobial properties and improve wound healing, but whether application of plant materials has additional effect beyond the natural production that occurs in man is unclear.

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Traditional Wound Dressings

Fig. 1: Banana leaf dressing in a patient with pemphigus. The patient lies on the leaves which provide a non-sticking surface, avoiding damage when removing absorbent dressings from raw skin.

Photo: Dr S Walton, Hull, UK

There is documented benefit of wound healing for numerous plants and extracts from plants. The emphasis of this article is on those that are applied as dressings, rather than those in which some form of therapeutic extract is used.

Saline

Publications that use the word 'saline' run into tens of thousands, but it is difficult to find articles specific to therapeutic trials in wound treatment. Where these are available, saline is usually viewed as a control that keeps wounds moist, but that has no other activity.

Briefly, picking out some relevant studies, saline soaked-gauze was as effective as silver sulphadiazine cream or an alginate emulsion in treating fresh partial thickness burns (in pigs) in one randomised controlled experimental study,[3] but less effective than silver sulphadiazine in another study. However, a more recent study suggests that saline-treated wounds heal slowly (saline was one of two control substances), and that silver sulphadiazine and other antibacterials may inhibit wound contraction by comparison to various other agents (such as aqueous cream, which was the other control substance).[4] An antimicrobial (0.057% sodium hypochlorite) was compared with saline as a wound cleanser and was associated with lower bacterial counts and faster healing.[5] Aloe vera compared with saline appears to have an anti-inflammatory effect, with faster healing times than saline alone, and that this or nystatin can reverse the impaired healing associated with antimicrobials.[4] Overall, it would appear that a dilute antiseptic is likely to be more effective than saline alone, that the latter is better than nothing, but that other agents have better wound-healing properties. Saline should be viewed mainly as a wound cleanser, rather than as a therapeutic dressing.

Banana Leaf

My personal experience of banana leaves is that I have eaten fish cooked in them, presumably to keep the fish moist. This may sound irrelevant, but this property is probably also important in their use as a wound dressing.

Banana leaf is non-adherent, protects wounds from trauma and prevents them becoming too dry. It is readily available and widely used, not only for wounds but also for eroded areas in dermatological conditions such as pemphigus (Figure 1) and toxic epidermal necrolysis. One study compared banana leaves with a traditional boiled potato peel bandage, and showed that the banana leaf dressings were equivalent in healing rates, but had the advantage of much lower cost and were easy to use.[6] The same authors have also shown that pain related to dressing changes is much less with banana leaf than with Vaseline (yellow soft paraffin impregnated) gauze.[7] However, a recent article raised concerns about the risk of infection from use of banana leaves, showing that only autoclaving and aseptic handling prevented culture of organisms from banana leaves (flame heating, boiling, and scorching with burning methylated spirits were not effective).[8]

Infection is a serious risk when there are large areas of skin erosion. Historically, in the UK, infection in pemphigus was a major cause of deaths. Otherwise, much of the literature on therapeutic uses of banana, or various extracts from bananas, relates to an anti-gastric ulcer property – this appears to be both prophylactic and also related to increased mucosal healing, but it is not clear that it has any role in wound healing.

Potato Peel and Other Potato Constituents

There are many articles that discuss use of boiled potato peel as a wound dressing.[9-12] Most reported experience relates to burns, and the best results, as might be expected, are in superficial partial thickness burns. However, deeper wound granulation may also be improved and full thickness wounds have been specifically investigated in rats. Other wounds that have been studied include pemphigus, pemphigoid and leg ulcers.[12] The active constituents are unclear but homogenated extracts are less effective than intact boiled potato peel. Potato peel fixed to gauze bandages is more effective than gauze bandage alone, suggesting that the outer cork layer (periderm) has a physical protective effect and probably prevents the wound from getting too dry. To be most effective, therefore, the dressing needs to be manufactured so that the pulp side is applied to the wound (usually with wheat starch paste on top and then a cotton gauze). Other potato constituents that have been suggested as useful are steroidal glycosides (steroidal glycolalkaloids), present in the boiled peel, and the fibre from sweet potato (this seems to be effective for absorbing exudates).[13]

In practical use, potato peel does not seem to be antibacterial. However, some studies have used it applied in preference to silver sulphadiazine or other antiseptics.[12] Boiled potato peel can be prepared as bandage rolls to make it convenient for dressing burn wounds. As with banana leaf, autoclaved dressings are preferable. One systematic review documented potato peel as being less effective than honey as a wound dressing (although numbers in all studies were small) but it may be more easily available.[14] Another concern is that contact urticaria to potato peel is reasonably common, although this may affect those preparing dressings, rather than the patient, as the causative protein is probably altered and non-allergenic after the boiling process.

Papaya (Paw paw; *Carica papaya*)

Papaya (paw paw) contains papain, chymopapain and other similar cysteine proteinases. In cooking, these have a tenderising effect on meat. In human medicine, paw paw (papaya) has been used on burns and other wounds – it is cheap, readily available, and easily applied by mashing the pulp for daily application.[15] Its proteinase activity probably gives it a desloughing activity, and it may also have an antibacterial effect. An extract of papaya has been shown to inhibit bacterial catalase (allowing bacteria to be more easily killed by macrophages) and has an antioxidant effect that reduces tissue injury.[16] Seed extracts have also been shown to have antibacterial effects against various organisms.[17]

Papaya has also been used in shampoo form to remove head lice[18] but, because it was combined with thymol and tea tree oil, it is uncertain which component was effective. It can, however, cause an immediate hypersensitivity reaction, and studies of systemic use in rats have shown that it damages testes and sperm to cause reduced fertility.[19] Data on absorption from wounds is not available but the threat of testicular damage suggests that it should only be used with caution.

Sphagnum Moss (*Sphagnum junghuhnianum* and others)

Sphagnum and related mosses (bryophytes) are found worldwide. They absorb water and have antiseptic properties. Use as a wound dressing has been documented as far back as the Bronze Age, and huge amounts were used during World War I to make wound dressings that were apparently less irritant and more absorbent than cotton dressings.[20] In India, recent research has compared 15 types of moss and documented sphagnum to be one of the more effective antimicrobial types (using ethanol extracts).[21]

Other Natural Dressings

Amniotic membrane has been used for many years as a natural dressing for burns and leg ulcers. Infection would now be considered a significant problem.

Collagen has been used in various forms of dressing, and is a component of manufactured (and expensive) 'skin equivalent' dressings. A novel method to produce a less expensive form of collagen dressings was reported in India recently. Fish scales, usually viewed as a waste product, were cleaned and processed to make them into a 'fish scale collagen sheet'. This is fibrous, allows transfer of water, and is strong enough to be used as a wound dressing.[22]

The Messages

Honey and banana leaf both appear to be superior in promoting wound healing compared with saline-soaked gauze. Honey is superior to some other traditional agents (e.g., potato peel) or frequently used antibacterials (e.g., silver sulphadiazine) as a wound dressing. However, there is evidence and at least some scientific rationale to suggest that boiled potato peel and mashed papaya have benefits in wound healing, and these plants are both cheap and readily available in many countries. It is possible that 'high technology' collagen sheet dressings might be available from fish scales in the future.

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PRESENTATIONS AND LOCAL REMEDIES OF WHITE JELLYFISH (RHIZOSTOMA SP.) STINGS IN IRAQI FISHERMEN

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Introduction

Jellyfish stings are common worldwide with an estimated 150 million cases annually.[1] More than 100 species are toxic to humans and their stings cause a wide range of clinical manifestations - from skin inflammation to cardiovascular and respiratory collapse.[2-4] Fatalities and hospitalisations may occur, mainly in the Indo-Pacific regions.[5]

Jellyfish are marine invertebrates belonging to the class Scyphozoa of the phylum Cnidaria. They can be found in every ocean in the world and in some fresh waters. The use of the term 'jellyfish' is actually a misnomer since scyphozoans are not fish, which are vertebrates.[4] The body of an adult jellyfish consists of a bell shape producing jelly and enclosing its internal structure, from which tentacles are suspended (Figures 1 and 2). Each tentacle is covered with cells called cnidocytes that can sting or kill other animals. Most jellyfish use these cells to secure prey or for defence. Others, such as the Rhizostomae, do not have tentacles at all.[6,7]

Persons who are involved in water activities such as swimming, sailing, saltwater fishing are more likely to get affected.[8,9] The toxin affects the sodium and calcium ion transport across the cellular membranes. It may affect skin, the myocardium, nervous tissue, hepatic tissue, and kidneys.[3] Skin lesions from jellyfish stings may present as immediate skin reaction such as local erythema, pain,

Fig. 1: Rhizostoma species

Photo: Kathem K Al-Rubiay et al

Fig. 2: Rhizostoma species

Photo: Kathem K Al-Rubiay et al

pruritus, paresthesiae, blistering, and swelling.[10-12] A delayed skin reaction occurs within a few days and mainly presents as pruritic papules[13,14] with histological findings similar to that of allergic contact dermatitis.[16] Systemic reactions are usually associated with exposure to a large amount of toxin. They are usually limited to nausea, headache, and chills, but may lead to major anaphylactic reaction with a drop in blood pressure, acute heart failure, and acute renal failure.[16-19]

Jellyfish stings are commonly treated by the application of normal saline or 5% acetic acid (house vinegar), ice pieces, seawater or tap water. Medical treatment can be achieved by antihistamine drugs and topical corticosteroids. Recently, a topical sting inhibitor has been used for prevention of jellyfish stings.[20-23] Several studies on jellyfish stings were carried out. However, none of these have been carried out in Basra which lies on the Arab Gulf in the south of Iraq. Our study is the first of its kind to study the clinical presentation and management of jellyfish stings in Basra.

Materials and Methods

One hundred fifty-five fishermen were selected randomly in this cross -sectional study at three Marine stations of fishermen in Basra. They were in Al-Fao (70 fishermen), Khour Abid Allah (45 fishermen) and Um-Kasser (35 fishermen). All were males and their ages ranged between 24-45 years (mean 33.6 ± 6.5 years). The study was conducted over a six month period. We used a questionnaire specifically developed for the purpose of this study. This questionnaire included the type of skin reactions, any systemic involvement, time of onset, the progression of the reaction, types and methods of treatments by fishermen, and any hospitalisation and fatality caused by jellyfish. The data were collected through face to face interviews and were statistically analysed.

Results

The study showed that the type of jellyfish was white jellyfish (*Rhizostoma sp*), with a white translucent umbrella and eight oral arms. Locally, the jellyfish is called Thaklol, Zaklol or white jellyfish (Figure 1).

Overall, 78.7% of fishermen gave a history of stings during the previous three months with no significant difference between

Presentations and Local Remedies of White Jellyfish Stings

Table 1: Local name and percentage of stings among fishermen in three stations (Overall percentage 78.7 in three stations)*

Stations	Local name	No. of fishermen	No. of stings	% of stings*
Al-Fao	Thaklol	70	60	85.7
Khour Abid Allah	Zaklol	45	30	66.6
Um-Kasser	Zaklol	35	28	80

*Table 2: Distribution of cases according to sites of stings among fishermen in three stations during the previous three months (*Overall % - hand 67.8%; legs 32.2%; abdomen 29.7%; eyes 11%)*

Signs and symptoms	Immediate reactions	Delayed reactions
Onset	Less than 5 minutes	After 72 hours
Number	In all cases	In 62% of cases
Pain	Very painful (89 %)	Painless (92 %)
Itching	Severe (68 %)	Mild (61%)
Burning sensation	Severe (45 %)	+/- Mild
Rashes	Fingerprint in 90.5%	Monomorphic papular (89.3%)
Fainting	Some cases (3%)	Not recorded
Course	Disappear during 48 hours	Cure within few weeks

Table 3: Clinical features of immediate and delayed skin reactions

Stations	No. of stings	Types of treatments (%) *			
		Seawater	Tap water	Ice pieces	No treatment
Al-Fao	60	66.7	48.3	41.7	25
Khour Abid Allah	30	60	36.7	40	20
Um-Kasser	28	67.9	42.9	35.7	28.6

*Table 4: Distribution of cases according to type of treatment (*Overall % - seawater 65.3%; tap water 44.1%; ice pieces 39.8%)*

Stations	No. of stings	Sites (%)*				
		Hands	Legs	Abdomen	Eyes	Others
Al-Fao	60	68.3	28.3	16.7	10	21,7
Khour Abid Allah	30	76.7	36.7	20	1 3	26.7
Um-Kasser	28	57.1	35.7	17.1	10.7	25

them in three stations (X^2 =0.74, P=0.6907) (Table 1). The common sites were the hands (67.8 %), followed by the legs (32.2 %). Other parts of the body were attacked, such as the abdomen, eyes and back, with no significant difference according to sites among fishermen in the stations (Table 2). The majority of fishermen (95.2%) claimed that stinging led to skin reactions immediately after exposure and within 5 minutes. The presenting complaints were pain (89 %), itching (68%), burning sensation (45%), and erythematous weals (Figure 3) in 90.5%. Others reported fainting (3%) and inability to move the part, especially when the attack was on the lower extremities. After a few days (3 days) of expo-

Fig. 3: Twelve hours after the exposure to jellyfish stings

Photo: Kathem K Al-Rubiay et al

sure, a new group of painless and itchy erythematous, monomorphic, papular rashes (Figures 4 and 5) occurred at the site of contact in 62% of cases, as delayed-type of reaction which resolved spontaneously in the majority of the cases (Table 3).

The most common type of local treatment used among fishermen was seawater in 65.3 % of cases, followed by tap water (44.1%) and ice pieces (39.8 %). About a quarter of fishermen (24.6%) considered stings were not significant and did not believe there was any need for medical help (Table 4).

Discussion

Fishermen are commonly exposed to jellyfish stings, especially in summer, because of fishing. From the information we gathered, the commonest type of jellyfish in the three stations was *Rhizostoma sp.,* class Scyphozoa, which belong to phylum Cnidaria and called locally Thaklol, Zaklol or white jellyfish.[6] During fishing, most white jellyfish die but remain attached to fishing nets. Fishermen may try to remove them. This may explain the high percentage (78.7%) of a positive history of stinging in this study among fishermen.

The study showed that white jellyfish envenomation can cause an immediate local skin reaction, presenting as weals in 90.5%.[6] Also, the study showed that delayed cutaneous hypersensitivity reactions were also common (62%) after three days of exposure and appeared clinically as itchy, erythematous, painless, monomorphic papular rashes. However, other reporters claimed that it occurs after five days or more, and is pruritic and painless.[13,14] Some authors described fatal or near-fatal stings and mortality rates up to 20%.[2,18,19] Our study did not support this and showed that no fatality and no admission to hospital was reported among workers in three stations during the study. The hand was the commonest site stung by this jellyfish, more than the legs, because during their work the fishermen used to pull the fishing net over the side of a boat. This finding is similar to that found in other studies.[5,16,17] Most of the fishermen reported that stings by jellyfish were simple and not life threatening and so there was no need to visit the doctor or the hospital. They were satisfied with many local remedies and some of them used sea or tap water or ice pieces; the pain disappeared and the rash improved rapidly. This approach of treatment was similar to that found by many studies, except the habit of washing with vinegar that was mentioned in some reports.[20,23] In conclusion, white jellyfish *(Rhizostoma sp.)* caused high numbers of the stings among fishermen in Basra city - that led to immediate and delayed reaction to the skin. Self treatment is common practice. Although life-threatening envenomation was
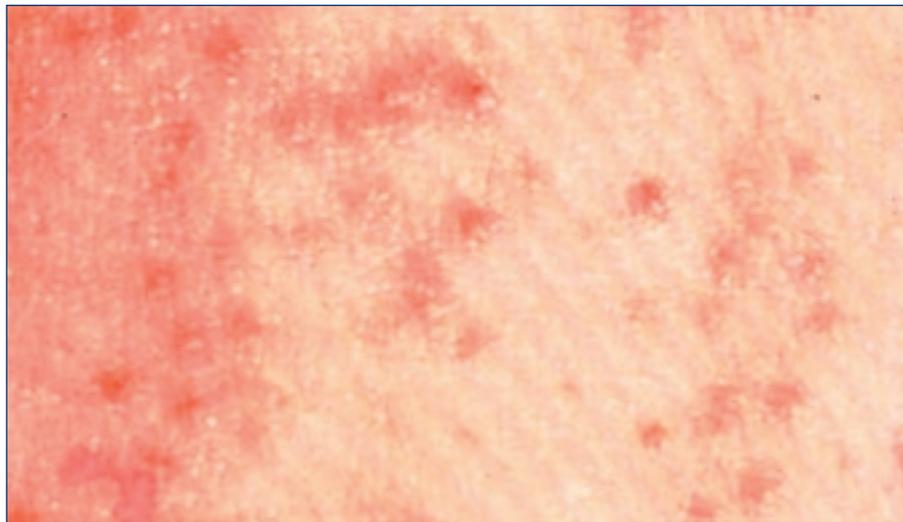
Fig. 4: Four days after the exposure to jellyfish stings

Photo: Kathem K Al-Rubiay et al

not recorded, we recommend stressing the importance of public education, prevention policies and protective clothing, especially for fishermen. Physicians practising in these areas must be aware of the problem and advise the patients about the hazards of jellyfish.

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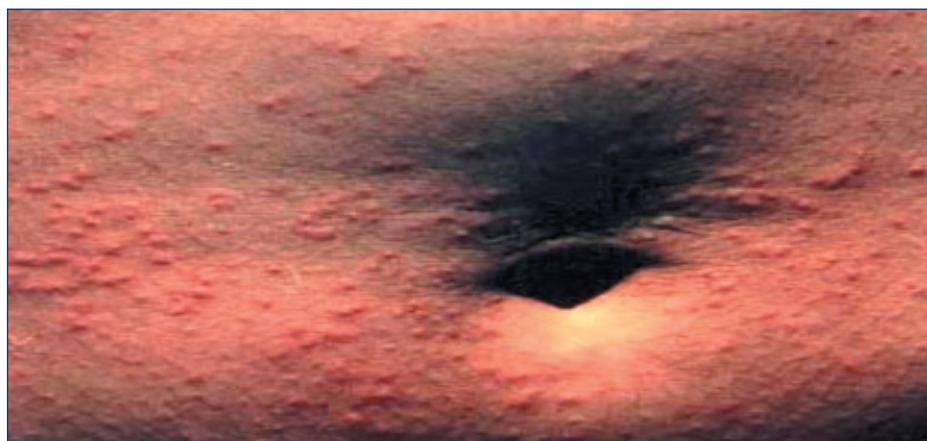
Fig. 5 : Seven days after the exposure to the stings

Photo: Kathem K Al-Rubiay et al

DIAGNOSING AND MANAGING LEISHMANIASIS IN THE COMMUNITY

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Introduction

The term leishmaniasis is given to infection with the protozoan parasite *Leishmania*, of which more than 20 species can cause disease in humans. Although the different species of parasites are microscopically indistinguishable, and all share the same basic life cycle pattern, with transmission to humans by the bite of a female sandfly, there are very fundamental differences in the diseases that they induce. For clinical purposes, leishmaniasis must be categorised as one of three forms, and not as a single disease.

Disease is localised to the skin in cutaneous leishmaniasis (CL). In muco-cutaneous infection (MCL) there is a predilection for late involvement and destruction of nasal and oropharyngeal mucosae, whilst in visceral disease (VL), infection becomes systemic. Although presentation of each of the three forms of this infection may be modulated to a certain extent by changes in the human host immunity, the manifestations are mainly determined by the species of *Leishmania*.

A little geography and epidemiology, therefore, goes a long way towards understanding the distribution of the clinical presentations of leishmaniasis.

The geography of human leishmaniasis is dictated by the distribution of appropriate sandfly vectors, intermediate hosts and *Leishmania* species. A knowledge of which species are found where, will often not only allow an accurate diagnosis of leishmaniasis to be made, but also dictate the most suitable therapies (Table 1). An informed approach allows appropriate treatment in the absence of laboratory diagnostics. Unfortunately, there are regions where different parasites overlap - for example, *L. major* and *L. tropica* in parts of the Middle East, or in South America where the *L. Viannia* complex that causes MCL is present alongside the *L. mexicana* complex that does not. Here, laboratory diagnostics can guide case management.

These maps of the world shows the distribution of leishmaniasis disease (Figures 1 and 2) across 88 countries with very different ecological habitats – from rainforest to arid desert. Reservoirs of infection for transmission of the different *Leishmania* species via the sandfly vector may be human (anthroponotic) or animal (zoonotic), and these differences have fundamental implications for disease control strategies.

The highest prevalence of zoonotic CL is seen in Iraq, Afghanistan, Iran, Saudi Arabia, Brazil and Peru, whereas over 90% of global anthroponotic VL is found in India, Nepal, Bangladesh, Brazil and the Sudan. Historically, CL was mainly confined to rural areas where reservoir host animals live, but more recently the spread to peri-urban populations is increasing.

It is estimated that 350 million people are living at risk in leishmania-endemic regions and that 2 million new cases of leishmaniasis occur annually (3:1 CL:VL). This may, however, be a gross underestimate as only one third of the affected countries have compulsory reporting.[1] The disease has been somewhat neglected as a major global health problem until recently when several factors raised its profile. These include large epidemics in refugee populations, a gradual shift from rural to more populated peri-urban areas, an increasing prevalence of co-infection of leishmaniasis with HIV, producing more severe forms of visceral disease with higher mortality rates.[2] Also, significant drug resistance has emerged to pose major treatment problems in some regions.

The female sandfly (Figure 3) typically bites animal or human hosts in the evening or night. Bites are usually not recalled as being painful. Exposed skin sites are the most commonly bitten, especially the face and hands. Children and men working outdoors are most frequently affected, although a single sandfly may bite and infect several members of the same household. Bed-nets and curtains impregnated with insecticide help to prevent transmission.[3]

In endemic areas, communities have long recognised the local disease manifestations with descriptions such as 'Balkan sore' (CL), 'Delhi boil' (CL), and 'black fever' (VL). Young children have been deliberately inoculated at covered skin sites in order that they may develop immunity and, thus, prevent scarring facial lesions in later life.

Clinical Presentation

Cutaneous Leishmaniasis

At the site of the sandfly bite a small erythematous papule develops within a few weeks. The painless papule enlarges, develops a dark red or purple colour and after a few months ulcerates (Figure 4). Traditionally the ulcers have been termed 'dry' or 'wet', depending on ulcer
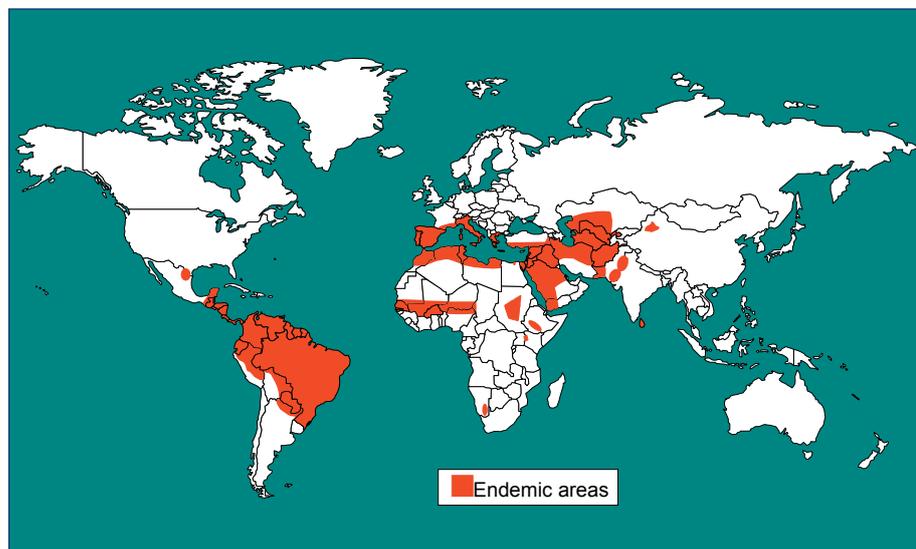

Fig 1: *World map showing regions endemic for cutaneous and muco-cutaneous leishmaniasis. Reproduced with kind permission of World Health Organization*

Endemic areas

Leishmaniasis in the Community

exudates or a dry scab which is produced. Regional lymphadeno-pathy is common. The natural history of CL lesions is that ulcers will heal spontaneously after several months; *L. major* lesions usually heal faster than those of *L. tropica* (2 - 6 months compared with 6 - 15 months respectively), but there are no hard and fast rules. Natural healing will leave a scar that may be disfiguring and, if overlying a joint, cause contracture. Patients do not usually have any systemic symptoms. Rarely, diffuse CL occurs, usually in those with impaired cell-mediated immunity, with cutaneous lesions spreading out from the initial site to event-ually involve the entire skin.

Table 1: Species of Leishmaniasis and their Typical Clinical Presentations

Clinical Presentation	New World (Americas)	Old World (Asia/Europe)
Cutaneous Leishmaniasis	***L. mexicana complex*** · *L. mexicana* · *L. amazonensis* · *L. venezuelensis*	· *L. major* · *L. tropica* · *L. infantum* · *L. aethiopica*
Visceral Leishmaniasis	***L. donovani complex*** · *L. chagasi = L. donovani*	***L. donovani complex*** · *L. donovani* · *L. infantum*
Mucocutaneous Leishmaniasis	***L. Viannia complex*** · *L. (V.) braziliensis* · *L. (V.) guyanensis* · *L. (V.) panamensis* · *L. (V.) peruviana*	

Mucocutaneous Leishmaniasis

In mucocutaneous disease, there is a spread of the *Leishmania* parasites to mucosal tissue, most commonly the mouth, palate and/or nose. Typically, the initial lesion will already have healed by the time mucocutaneous involvement becomes apparent sev-eral months, or even years, later. Mucosal involvement may mani-fest initially with non-specific nasal stuffiness or nose bleeds. Later, inflammation of the gums, painful ulceration and destruction of oro- and naso-pharyngeal/laryngeal cartilage may result. The resulting deformity can lead to severe morbidity, and secondary infection and sepsis may cause fatalities.

Visceral Leishmaniasis

This disease is usually indolent in nature but its presentation is variable and may be acute. Patients typically present with fever, malaise, weight loss, lymphadenopathy, hepatomegaly, spleno-megaly (hard, non-tender), and hyperpigmentation of the skin (kala azar). Visceral disease is more common in the immunosup-pressed or malnourished. Patients may die from anaemia, haemor-rhage, sepsis or secondary illnesses in their vulnerable state. Post VL skin lesions have been reported in approximately 2-10% of patients. The lesions may be hypopigmented macules (usually in the trunk), erythematous patches (in a photo-sensitive 'butterfly' distribution over the cheeks) or pink nodules (usually on the face).

Risk factors associated with severe or recurrent leishmaniasis
- Very young age
- Malnutrition
- HIV co-infection
- Partially treated disease.

Clinical Differential Diagnosis

(adapted from Magill, 2000[4])

CL (Cutaneous Leishmaniasis)
- Tuberculosis - lupus vulgaris or cuta-neous *Mycobacterium tuberculosis*
- Leprosy (*M. leprae*)
- Other mycobacterial infections including Buruli ulcer (*M. ulcerans*)
- Syphilis or endemic treponematosis (yaws)
- Cutaneous anthrax

- Sarcoidosis
- Myiasis – at papule stage of CL
- Implantation mycosis – sporotrichosis, lobomyco-sis, blastomycosis or chromoblastomycosis
- Neoplasm (lymphoma/leukaemia).

MCL (Mucocutaneous Leishmaniasis)
Cutaneous stage as for CL
Mucocutaneous:
- Wegener's granulomatosis
- Behçet's disease
- Systemic mycosis: histoplasmosis, paracoccidioidomycosis
- Nasopharyngeal carcinoma.

VL (Visceral Leishmaniasis)
- Malaria
- Typhoid
- Schistosomiasis
- Tuberculosis
- Brucellosis
- Syphilis
- Sarcoid
- Systematic lupus erythematosis.
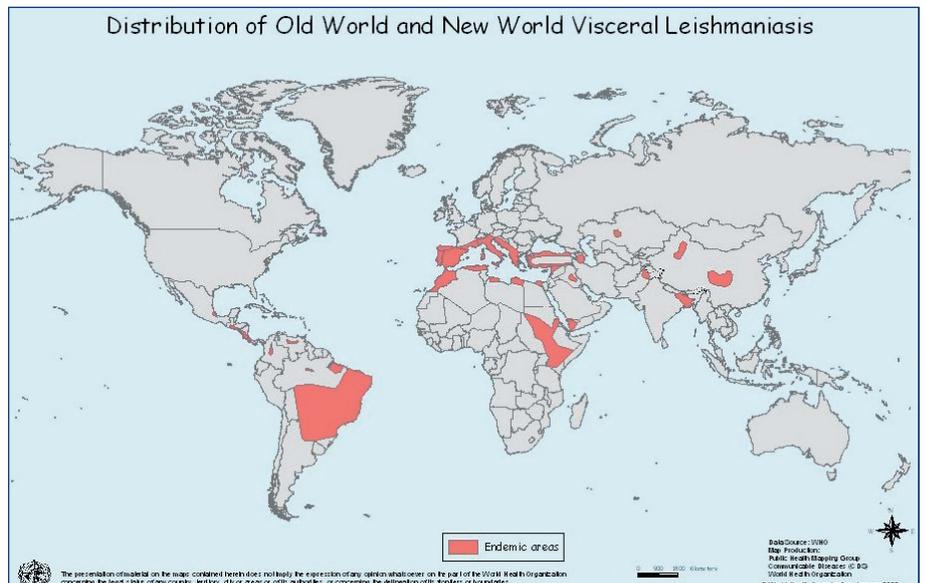
Fig 2: World map showing regions endemic for visceral leishmaniasis.
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Leishmaniasis in the Community

Fig 3: Female phlebotomine sandfly
First published in Morris-Jones S & Weber M. New Engl J Med 2004: 350; 2313-4. Copyright © [2004] Massachusetts Medical Society.

Investigations

Experienced health care workers in endemic areas often diagnose leishmaniasis on the clinical appearances alone with a high degree of accuracy.[5] Moreover, in many endemic regions laboratory investigations are not routinely available.

1. Microscopy

Where light microscopy can be performed, training to recognise parasites on tissue smears will allow a definitive diagnosis of leishmaniasis. Smears are spread on a glass slide and stained before viewing.

- **CL (MCL)**

  For CL or the cutaneous stage of MCL, smears can be prepared from a slit skin scalpel scraping or a biopsy touch preparation of an ulcer. Scalpel scrapings are best taken from the indurated margin of the most recent lesions, as these are most likely to harbour more parasites. The skin should be cleaned before sampling, and then the ulcer edge stretched tight. A small incision should be made radially across the indurated edge to a depth of a few millimetres. While the skin is still stretched to produce a bloodless field, the scalpel blade is turned 90° and scraped along the indurated cut edge. A small amount of serous tissue fluid can then be gathered on the scalpel blade and smeared immediately onto a glass microscope slide. This material is menthol-fixed (70%) and then stained with Giemsa/Leishman or Wright stains and examined (usually x 500-1000, under oil immersion). *Leishmania* parasite amastigotes may be seen inside macrophages and mononuclear cells, but not uncommonly are squeezed out and lie 'free' between cells. The amastigotes are typically ovoid, measuring 2-5 μm by 1-2 μm and are readily recognised by their large nucleus and distinct pathognomonic kinetoplast (giving the so-called 'dot and dash' appearance; see Figure 5).

- **VL**

  Smears for diagnostic microscopy may also be made in cases of possible VL, using peripheral venous blood, fine-needle splenic aspiration, or rarely liver or bone marrow biopsy. When using venous blood, samples should either be centrifuged for buffy coat examination or thick blood films made to improve the detection rate, as parasite density in peripheral blood may be very low.

2. Culture

Leishmania parasites can be cultured over 1-4 weeks from skin, blood, bone marrow or splenic aspirates using Novy-McNeal-Nicolle (NMN) medium. The technique is time consuming and requires a laboratory with facilities for microscopy, refrigerated storage of medium, and parasite incubation at 22°C.

3. Leishmanin skin test (Montenegro test)

This intradermal injection of killed promastigotes is used to assess cell mediated immunity to *Leishmania* parasites in the form of a delayed type hypersensitivity reaction. In areas endemic for CL and MCL, a positive result is frequently present in a large proportion of the population, reflecting previous exposure, and may, therefore, have limited diagnostic use in individual cases. False positive results have been reported with diseases such as sporotrichosis.[6] In VL, the sensitivity of the test is initially poor, due to the global immunosuppression caused by the disease, although this improves by the time of treatment completion.

The use of the test is further complicated by the variability in batch production, and it is, therefore, perhaps best reserved for epidemiological studies rather than individual case diagnosis.

4. Serology

A more specific test for diagnosing visceral disease is the direct agglutination test (DAT). This uses stable freeze-dried antigen, the test is inexpensive, reliable, easy to use and highly sensitive and specific.[7] This test can be used in the community setting. The test is also reasonably reliable at diagnosing VL in the context of HIV co-infection.

5. Molecular methods

There are a huge range of molecular techniques that have been described to diagnose leishmaniasis. Many use the multiple copies of kinetoplast DNA to detect infection. However, given the enormous variety of *Leishmania* species and sub-species, there is no single molecular test that will reliably detect and distinguish all human *Leishmania* parasites. Such tests are most appropriate for diagnosis in areas where there is little genetic variation in the parasites being detected.

Currently, molecular tests remain too expensive for routine use in resource-stretched settings. Nevertheless, in laboratories where there are sufficient resources, molecular tests have been applied with great success to detect, speciate* and, therefore, direct treatment. However, a negative PCR result does not rule out leishmaniasis.[8]

> *Speciation – The evolutionary formation of new biological species, usually by the division of a single species into two or more genetically distinct ones

6. Indirect methods

VL produces a profound hypergammaglobulinaemia. Historically, in field conditions, a crude and non-specific but rapid method for diagnosing leishmaniasis was to add formalin (40%) to patient serum and look for a white precipitant indicating high protein levels.

Non-specific haematological parameters that may support the diagnosis of leishmaniasis include low haemoglobin, low lymphocyte and neutrophil counts as well as low platelets, all reflecting the suppression of the bone marrow by parasite infiltration.

Management

Immunisation

Although there has been much speculation about the potential for successful immunisation against leishmaniasis, the unfortunate

Leishmaniasis in the Community

reality is that no effective vaccine is available. Traditional inoculation of 'live' leishmaniasis into the skin at a covered skin site has long been recognised as a way of producing long-lasting immunity. However vaccination with killed parasites or recombinant proteins induces only temporary protection.[9]

CL

Cutaneous leishmaniasis is a disfiguring disease associated with social stigma but otherwise does not cause significant morbidity or mortality. Lesions will resolve spontaneously (usually within 5-15 months) and many of those affected never seek medical attention. The rationale for therapy is that more rapid healing will result in less scarring. Both cryotherapy[10] and heat treatment[11] have been used with some success for isolated lesions. When cutaneous lesions are single or few in number, they may be treated simply with a series of intralesional sodium stibogluconate (pentavalent antimony) injections. The entire lesion/ulcer margin should be infiltrated at each injection, which may be painful. The lesions usually heal with some post-inflammatory pigment change and mild scarring.

However, in many countries those affected do not usually attend a medial practitioner unless they have exhausted traditional remedies first. A study in Ecuador shows that locals were very familiar with *sarna brava* ('angry sore') which they would treat themselves using a variety of therapies including heat, cold, cauterization, strong chemicals and plant preparations. Extracts from 45 different plants were used – including banana pulp, papaya juice, lemon peel/juice, and tobacco leaves.[12] In Afghanistan, electro-thermocauterisation under local anaesthetic has been used successfully to treat CL for many years. Honey has been shown to keep cutaneous lesions clean, reduce secondary bacterial infection and even reduce the size of some lesions in an animal study.[13]

In Sudan, there are studies showing the effectiveness of garlic methanol extracts which were as effective as antimonial therapy; 66% of cutaneous lesions healed completely and 24% partially at around 20 days.[14] Neem paste was less effective than garlic, although 33% of patients had complete healing and 50% partial healing at 20 days. Garad paste was not shown to be effective.[14]

Several studies undertaken in Iran have shown some encouraging results with the use of Z-HE paste made from a herbal extract and applied directly to the skin lesions. After 6 weeks of treatment, 74% were completely cured, 12% had a partial response and 14% no response.[15]

15% paromomycin (aminoglycoside antibiotic) in an ointment used twice daily for around 15 days has been shown to be effective with cure rates of 74% in old world disease and 85% in new world cutaneous leishmaniasis.

Recently, topical 5% imiquimod (Aldara) applied to cutaneous lesions, in addition to conventional therapies, has been shown to be effective with a higher proportion of lesions healing in a shorter time.[16]

For extensive cutaneous lesions, treatment should be systemic as for MCL or VL (see below).

MCL / VL / Extensive CL

For these forms of leishmaniasis treatment should be systemic. Traditionally, pentavalent antimonials (sodium stibogluconate and meglumine antimoniate) have been used IV (filtered and given slowly over 5 minutes) or IM at a dose of 20mg/kg/day for 20-40 days (10 days for CL). Patients not infrequently develop gastroin-

Fig 4: *Characteristic ulcer of cutaneous leishmaniasis*
First published in Morris-Jones S & Weber M. New Engl J Med 2004:
350; 2313-4. Copyright © [2004] Massachusetts Medical Society.

testinal symptoms, arthralgia, headache, abnormal liver function, raised amylase, low platelets and a disordered heart rhythm. Rarely, bleeding from mucous membranes and substernal pain may result. Rarely, when MCL is treated, a severe inflammatory reaction can result in significant tissue swelling in the oropharynx that should be managed with systemic corticosteroids. Although reasonably high cure rates of 76% are reported,[17] there are a number of problems with antimonial therapy. The drug is not universally available; its side effect profile may limit adherence, as does the duration of required treatment. Recently, the rapid emergence of resistance to pentavalent antimonials has been reported, particularly in the Indian Sub-Continent.

Second line therapy varies according to drug availability and costs. Pentamidine IM has been used, 3-4mg/kg on alternate days for a maximum of 10 injections, to treat VL or MCL, and twice weekly for CL, until the lesions resolve clinically. Initial responses are often good but high relapse rates are reported with visceral disease, and the treatment may need repeating.[17] Adverse effects include hypotension (which may be severe, so that all patients should be treated lying down), hypoglycaemia, pancreatitis, arrhythmias, leucopenia, thrombocytopenia and acute renal failure.

Amphotericin B may also be used to treat leishmaniasis. The drug is administered IV at 1mg/kg/day for 10-21 days or, if available as the liposomal formulation, at 3mg/kg/day for 5 consecutive days, followed by a single dose one week later. Treatment doses and durations may need to be increased in the context of co-infection with HIV.

Paromomycin (an aminoglycoside) given IM daily for 21 days, at a dose of 11mg/kg, has shown comparable results to amphotericin B in some studies.[18]

Standard treatments for VL/MCL are mostly cumbersome, expensive and often with a poor toxicity profile, underlining the urgent need for inexpensive, safe alternative therapies. The most promising development in the treatment of VL has been in India where oral miltefosine, 100mg daily (2.5mg/kg/day) for 4 weeks, seems to be highly effective. Cure rates for visceral disease of 94% at 6 months are reported.[19] The main drawbacks are compliance and teratogenicity (pregnancy must be avoided during the treatment and for 2 months afterwards).

VL / HIV Co-infection

Dual infection with HIV and VL poses particular clinical problems. Diagnosis may be more difficult as presentations are atypical, and parasites are more frequently found in unusual locations. Relapse and mortality rates are both higher than in VL alone, and drug toxicities, especially of the antimonials, are similarly more common in co-infected patients. Where highly active antiretroviral therapy (HAART) is available, successful sustained suppression of viral replication may allow secondary prophylaxis to be considered.

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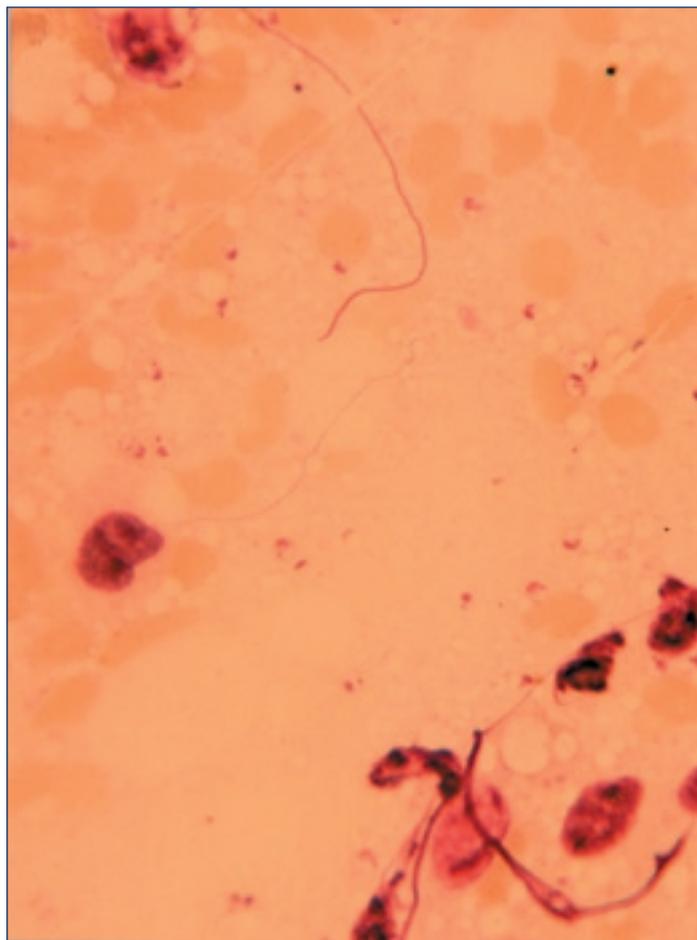
Fig 5: Leishmania amastiogtes seen on a slit skin smear taken from the edge of a cutaneous ulcer

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Question 1:

This fisherman's son complains of intense itching on the buttock area. He is not itchy elsewhere:

a) *What do you see?*

b) *What is the diagnosis?*

c) *What is the treatment?*

Question 2:

This patient is itchy all over. She keeps noticing these straight linear raised streaks that last about 2 hours, then go down, but may appear somewhere else:

a) *What is the diagnosis?*

b) *How would you treat it?*

Question 3:

This child has a serpiginous wheal on his buttock. It has been there for about an hour. Earlier today he had a similar one on his back and yesterday one on his abdomen:

a) *What is the diagnosis?*

b) *What is it caused by?*

c) *How would you treat it?*

REGIONAL DERMATOLOGY TRAINING CENTRE RESEARCH REPORTS

Provided by Dr Barbara Leppard

Why are Namibian men reluctant to wear condoms?

The late Yvonne Siyongo
Caprivi, Namibia

In sub-Saharan Africa, the prevalence of STIs and AIDS is higher than anywhere else in the world. To look at one possible reason for this, Yvonne interviewed 125 male government workers from Katima Mulilo in Caprivi, Namibia and 11 local pastors - to ask about their knowledge and attitudes to the use of condoms. The government workers were asked if they knew what a condom was, if they knew how to use one, if they had ever used one, if they used one regularly, and if not, why not. The pastors were asked what they were teaching their congregations about the use of condoms. Seventy-four percent (74%) of male government workers knew what a condom was and how to use it, but 39% had never used one. The reasons given for not using them included:

- They are unnatural
- They reduce sexual satisfaction
- Their partner did not like them
- Their partner would assume they were being unfaithful if they used them
- They are anti-Christian
- Traditional herbs are a better protection against STIs and AIDS than condoms
- Traditional herbs are better for family planning than condoms
- They can only be obtained at the local hospital or health clinic.

All pastors said that their church taught that individuals should be chaste before marriage and faithful afterwards. Therefore, there was no need for the use of condoms.

She concluded that there was a good case for the education of both men and women in the use of condoms in order to stop the spread of STIs and AIDS and that they should be more widely available, e.g., in shops as well as health centres and hospitals. The church needs to emphasise the importance of behaviour change as the real way to prevent STIs and AIDS.

Efficacy of partner notification in Swaziland

Mpumelolo Dlamini
Mbabane, Swaziland

Contact tracing and partner notification is an important part of the strategy for treating STIs all over the world. In the STI clinic at Mbabane Government Hospital, this is done by giving the patient a slip of paper to give to their sexual contacts, advising them to attend the clinic.

In a study of 318 patients attending the clinic (60% male, 40% female), only 27% of their contacts attended. Contacts of patients who had multiple sexual partners were less likely to respond to this method of partner notification than those with only one partner. These findings indicate that this type of partner notification is not an effective method of contact tracing, particularly for individuals with multiple sexual partners. The clinic needs to revise its strategy if they want to be more successful.

Self medication in patients attending clinics for STIs in Cameroon and Zambia

Marie Michelle Boum
Yaounde, Cameroon

Charles Hakoma
Mazabuka, Zambia

Self medication in patients with STIs can result in:

- Drug resistance
- Increased incidence of HIV if the wrong treatment is given (STIs are known to facilitate transmission of HIV)
- Pelvic inflammatory disease leading to ectopic pregnancy and sterility
- Vertical transmission of disease to the unborn child leading to ophthalmia neonatorum, congenital syphilis and HIV infection.

Both studies looked at the extent of self medication in patients attending an STI clinic. The investigators used a questionnaire to find out why patients treated themselves rather than going to an STI clinic, who they asked for advice and what treatment they had used.

Results

Both studies concluded that the government of their respective countries needed to look at the fees charged for attending clinics and having the relevant tests done. There was also a need to make sure that the appropriate drugs were both available and affordable in the clinics. In addition, the general public needs education about the need for proper treatment of STIs in order to prevent spread of these diseases - to prevent complications and as a means of reducing the incidence of HIV/AIDS.

	Zambia	Cameroon
Number of patients interviewed	112	150
% of patients attending the STI clinic who had already treated themselves	80%	72%
Reasons for self medication	• High fees at clinics • Lack of drugs at clinics • Long waiting time at clinics	• High fees for lab tests at clinics • Lack of drugs at clinics • Long waiting time at clinics • Didn't want wife/husband to know about the problem
Treatment used	Antibiotics 19% Traditional medicine 36% Traditional medicine and antibiotics 30% Other drugs 15%	Antibiotics 77% Traditional medicine 11% Other drugs 12%
Received advice from	Relatives and friends	Relatives and friends

QUIZ: ITCHY LINES   ANSWERS

Answer 1:

a) Serpiginous wheals

b) Cutaneous larva migrans, creeping eruption. Caused by various nematode parasites, e.g., Ancylostoma braziliense

c) Ivermectin or albendazole

Answer 2:

a) Dermographism or physical urticaria

b) Antihistamines, like chlorpheniramine, might help, but also avoiding things that encourage urticaria, such as some pain killers. This lady was taking ibuprofen for abdominal pains she was experiencing during menses. It can also happen with codeine.

Answer 3:

a) Larva currens

b) Threadworm or human parasitic roundworm called Strongyloides stercoralis

c) Ivermectin, albendazole or thiabendazole

HIV RELATED SKIN DISEASES AND SEXUALLY TRANSMITTED INFECTIONS IN AFRICA

An Illustrated Guide

Merja Kousa and Cornelus Sanders
(Kopijyvä Oy, Jyväskylä, 2006)

ISBN 952-92-0163-X

This compact ring-plan handbook is an excellent summary of common skin manifestations of HIV-AIDS, including concise text and a summary of currently available treatments. This is accompanied by profuse illustrations, mostly of good quality, and many derived (with acknowledgement) from the Regional Dermatology Training Centre, Moshi, Tanzania. Most of the treatment recommendations are very sensible (although I would not advocate 'potent corticosteroid ointments' for psoriasis in general). Certainly the book will prove of great value to health care workers in Africa (and other parts of the world, for that matter). The book is currently available, for the cost of postage, from the Teaching Aids at Low Cost (TALC) website – www.talcuk.org - together with many other reasonably priced books.
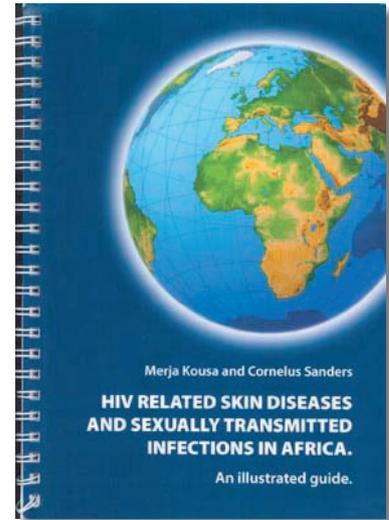
Chris Lovell

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Community Dermatology

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