Parasites and the skin

Parasitic infections may be confined to the skin or may have skin involvement as part of their pathology.

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This article seeks to familiarise readers with the management of those conditions that are encountered in daily practice and to remind you of those rare and wonderful infestations that you might never see. I will focus on and deal with parasitic infestations and the skin. Skin pathology often provides important clues to systemic infections. This article will discuss common clinical presentations and tabulate the rarer diseases.

Parasitic infestations are common in the tropics due to a combination of heat, humidity and ultimately poor socioeconomic and health care conditions.

Parasitic infections can be solely confined to the skin, as seen with human scabies, cutaneous larva migrans, the chigger flea, cutaneous myiasis and cutaneous leishmaniasis. Parasites not confined to the skin include onchocerciasis, loiasis, the guinea worm, schistosomiasis, cutaneous amoebiasis and the cutaneous involvement in trypanosomiasis.

Common scenarios

Scabies

The common scenario of a child brought to a busy rural outpatient department or public hospital is shown in Figs 1 and 2. The history is that of severe pruritis persisting for a few weeks, worse at night and there are family members or friends with the same affliction as shown in Fig. 1. The diagnosis is scabies until proven otherwise, and treatment consists of topical scabicides.

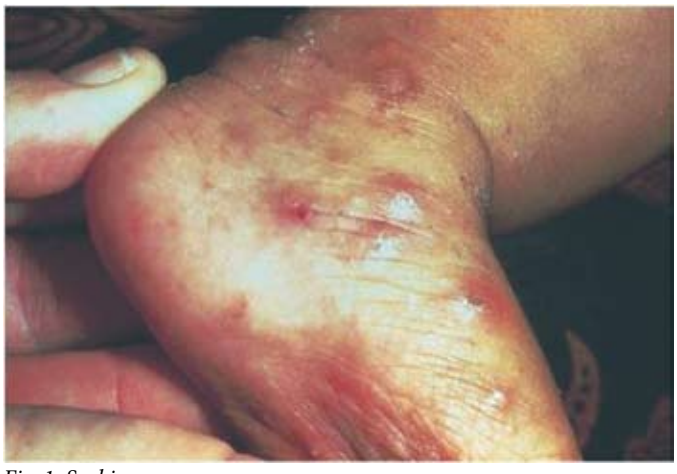

Fig. 1. Scabies.

Human scabies is caused by the host-specific mite *Sarcoptes scabie* var. *hominis*. A hypersensitivity reaction to the mite is responsible for the intense pruritis experienced by infested individuals. This burrowing mite lives its entire life cycle within the epidermis of the skin. Secondary infection with group A *Streptococcus pyogenes* or *Staphylococcus aureus* may occur. Transmission occurs by direct contact and sometimes spreads through fomites. Drug resistance to topical scabicides is occurring.

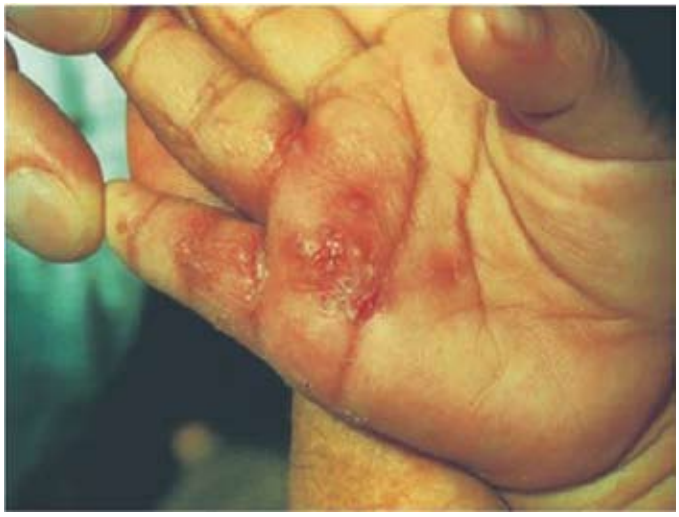

Fig. 2. Scabies.

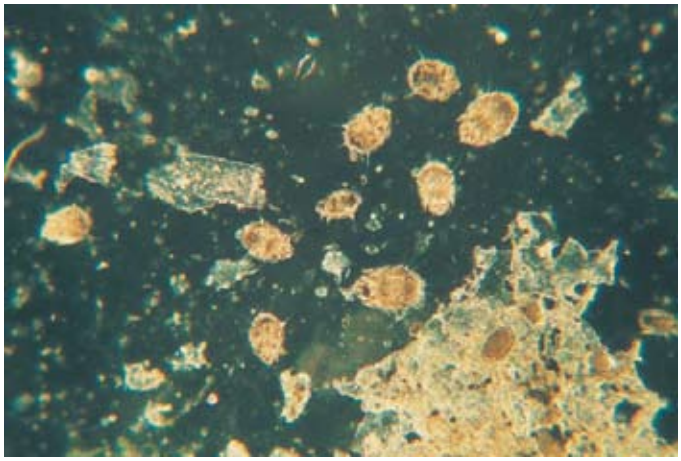

Fig. 3. Smear of skin scraping showing abundant scabies mites.

The diagnosis is confirmed by direct microscopy of skin scraping from a burrow, mounted on a glass slide. The findings are demonstrated in Fig. 3. Dermoscopy, epiluminescence microscopy and skin biopsy are other diagnostic aids. Treatment is shown in Table I.

Treatment of scabies

Effective management of scabies requires the following:

- Treat all contacts.
- Apply scabicides from the neck down over the entire body, especially unaffected intertriginous areas of the skin.
- Avoid using antiseptic such as dettol and savlon.
- Avoid overuse of tetmosol soap, which may worsen existing pruritis.
- Disinfect towels, clothing and bedding.

Table I. Treament of scabies

Drug	Dose	Comment
Gamma-bezenehexachloride: Lindane 1% lotion	Apply and leave on for 8 hours; repeat 1 week later	Contraindicated during pregnancy and in children <2 years of age Resistance is emerging Aplastic anaemia recently reported in children treated with Gambex shampoo
Precipated sulphur 5 - 10%: Tetmosol soap 5%	Apply for 3 consecutive days, then wash off	Safe in children and in pregnancy Preparations include Tetmosol soap 5% Useful for prophylaxis Ineffective in established infestation Sulphur ointments in soft white paraffin effective in children
Crotamiton: Eurax	Apply on 2 consecutive days, repeat in 5 days	Eurax not as effective as the others
Benzyl benzoate 10% lotion: Ascabiol emulsion 25%	Apply for 24 hrs then wash off May need to repeat	Dilute in water for children Safe in pregnancy Rare side-effects Skin irritation
Ivermectin 200 µg/kg	Stat dose Can repeat after a week	Highly effective, especially in Norwegian crusted scabies Can be obtained on a named-patient basis from MSD with permission from the MCC
Pyrethroids: Spregal aerosol	Spray entire body except the face; leave on overnight and repeat one week later Repeated sprays may be needed in HIV+ patients All persons affected in same household to treat at the same time Do in well-ventilated room and avoid any flames Disinfect clothes and bed linen	Esdepallethrin is a pyrethroid pesticide, which is scabicidal Piperonyl butoxide acts by blocking the defense system that the parasite uses to counteract the latter Contraindicated in children <2 years and during pregnancy

- Use systemic antibiotics and/or systemic antihistamines in severe cases.
- Short courses of topical or systemic steroids may be effective in treating post-scabetic pruritis, which is common. Avoid the continuous use of topical antiseptics.
- Use sulphur-based ointments in neonates, infants and in pregnancy.

Norwegian scabies

The second clinical scenario of Norwegian scabies is commonly seen in HIV-positive patients. Fig. 4 shows the eczematous, psoriasiform rash reminiscent of psoriasis.

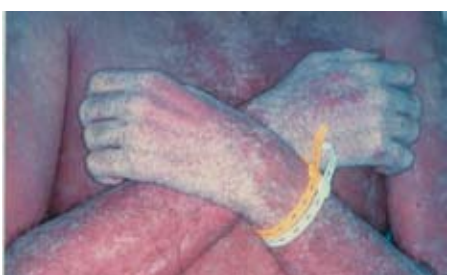

Fig. 4. Psoriasiform rash of Norwegian scabies.

Diagnosis will be assisted by considering the following:

- One or more skin biopsies may be required to confirm the diagnosis.
- This illness is highly contagious and often health care workers become afflicted after contact.
- Norwegian scabies is commonly seen in old age homes and psychiatric facilities.
- The most effective treatment for Norwegian scabies is oral ivermectin, which requires permission for use from the Medicines Control Council.
- Several applications and prolonged use of stronger concentrations of sulphur ointments, Ascabiol or Spregal spray need to be used in these patients to obtain cure.
- Keratolytics and occasionally anti-proliferative agents are needed to clear the hyperkeratosis that is teeming with mites before using the above agents.

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Myiasis

Scenario 3 demonstrates a typical case of myiasis. A backpacker ventured into rural Zimbabwe for a few months and subsequently returned to Johannesburg with numerous boils on his back (Fig. 5). These irritating lesions persisted for approximately 3 weeks and did not respond to topical antiseptics and systemic antibiotics.

Myiasis is caused by the larvae of flies, which lay their eggs on skin or clothing. The eggs hatch and the larvae penetrate the skin. Worldwide the most common flies that

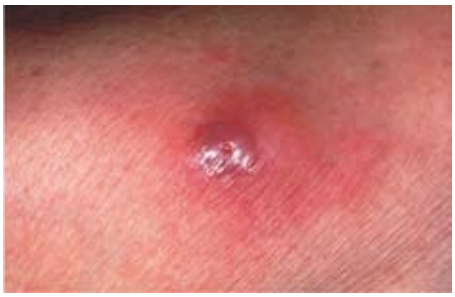

Fig. 5. A boil typical of myiasis.

cause human infestation are *Dermatobia hominis* (human botfly) and *Cordylobia anthropophaga* (tumbu fly).

The route of transmission differs with different flies. The botfly lays her eggs on mosquitoes, which in turn deposit them on warm-blooded mammals. The tumbu fly deposits its eggs on moist clothing, soiled blankets and in sand. In endemic areas people usually iron their clothes after hanging them out to kill the fly eggs.

There are essentially two types of myiasis:

- Furuncular myiasis (Fig. 6), which is what the patient described in our scenario has, usually caused by the botfly.

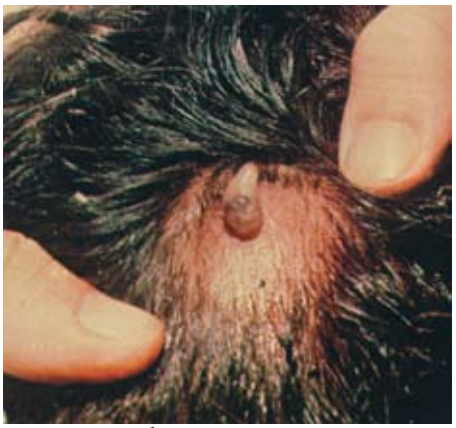

Fig. 6. Furuncular myiasis.

- Wound myiasis (Fig. 7), where larvae are deposited in suppurating wounds or on decomposing flesh. *Cochliomyia hominovorax* is the causative fly in the Americas and *Chrysomia* in Africa.

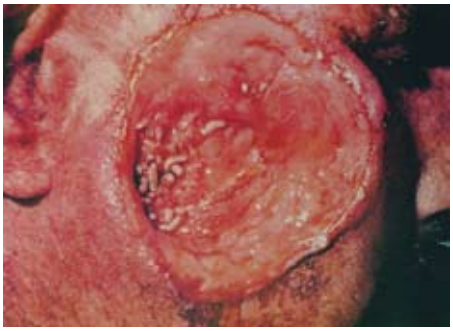

Fig. 7. Wound myiasis

The main aim of treatment is literally to suffocate the larvae. Occlusive ointments such as vaseline are effective as they interfere with the larva's respiration and force it to extrude itself. Alternatively, surgical nicking of the furuncle followed by extraction of the larvae can be curative (Fig. 6).

Topical and systemic antibiotics may be needed to cure any secondary infection. The approach in wound myiasis would be surgical debridement and the principles of surgical management.

Cutaneous larva migrans

In the fourth scenario a young child is brought for a rash on his foot, as shown in Fig. 8. The family had just returned from a coastal holiday. This is typical of cutaneous larva migrans or 'creeping eruption'. The latter term is being used because of the slow crawling movement of the worm, which is visible. This condition is due to the incomplete development of hookworm larvae, whose natural hosts are cats and dogs, in man. The larvae are found in damp soil contaminated by dog and cat faeces. Invasion of human skin usually takes place on beaches, where shoes are seldom worn.

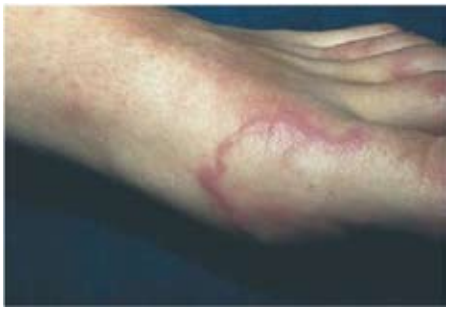

Fig. 8. Cutaneous larva migrans.

Treatment of larva migrans

Spontaneous cure can take place over months. Do not try to catch, freeze or surgically clip the worm. The treatment of choice is a single dose or 3-day course of albendazole.

Alternatively, a 500 mg tablet of thiabendazole is ground up in 25 g of vaseline and applied once a day for 2 days.

Chigger fleas or tungiasis

In scenario 5 a child from a rural, economically poor area of KZN is brought to you. Fig. 9 demonstrates the clinical picture. The primary lesions are black dots, papules, nodules and burrowing excoriations. There is some resemblance to a minor abscess with a central punctum. The child complains of mild discomfort. The differential diagnosis includes infected warts or scabies but the primary lesions of these are fairly typical ofchigger fleas, therefore always consider tungiasis or chiggers in this setting.

This is common in the tropics (endemic in Central and South America, the Caribbean, tropical Africa, India and Pakistan), and is

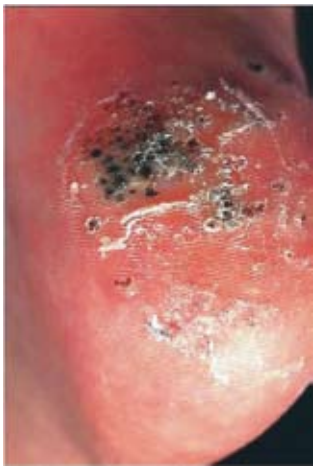

Fig. 9. Heel of foot, showing wart-like lesions.

caused by the wingless flea *Tunga penetrans*. The condition is called tungiasis.

The flea's eggs are found in clusters in soil, from which infestation of the bare-footed patient occurs. The impregnated female burrows itself into the skin of the foot, the toe webs, around the nails and on the heels. The flea's abdomen expands rapidly, forming a large white sphere like a mistletoe berry. Rare complications include gangrene, tetanus and auto-amputation.

Treatment of tungiasis

- Maintaining a high index of suspicion for this condition.
- Removal of the flea with a sterile needle.
- Surgical curettage and electrodessication.
- Topical thiabendazole or ivermectin.
- Systemic thiabendazole or ivermectin.
- Systemic antibiotic cover.
- Tetanus prophylaxis.

Leishmaniasis

In this scenario, a 26-year-old medical doctor visited Israel over a period of a month and returned with a small sore on his upper lip. This increased in size with time. He took an empiric dose of a broad-spectrum antibiotic in addition to a topical antibiotic for 2 weeks, with no response. He had no associated constitutional symptoms. Fig. 10 shows the ulcerating plaque, which is clinically non-diagnostic.

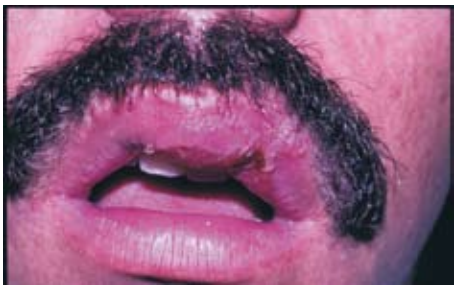

Fig. 10. Leishmaniasis

The differential diagnoses include:

- furunculosis resistant to antibiotics
- an actinic cheilitis (this would occur on the lower lip)
- granulomatous conditions which may be fungal such as sporotrichosis, or mycobacterial such as tuberculosis
- atypical mycobacteria
- syphilis or other sexually transmitted infections
- neoplasias.

However, his visits to the Middle East would make one consider leishmaniasis.

Diagnosis requires the mandatory performance of an adequately sized deep skin biopsy.

The presence of amastigotes in neutrophils is in keeping with leishmaniasis. This doctor had the oriental sore of cutaneous leishmaniasis.

Leishmaniasis is a genus of flagellate protozoa found in Africa, the Mediterranean basin, the Caribbean and Latin America. It is transmitted by the bite of the phlebotomus sandfly.

Dogs and rodents are the intermediate hosts.

There are three forms of leishmaniasis:

- cutaneous leishmaniasis, which is restricted to the skin and is seen more often in the old world, as seen in our patient
- mucocutaneous leishmaniasis, which affects the skin and mucous surfaces and occurs exclusively in the so-called new world (Fig. 11)

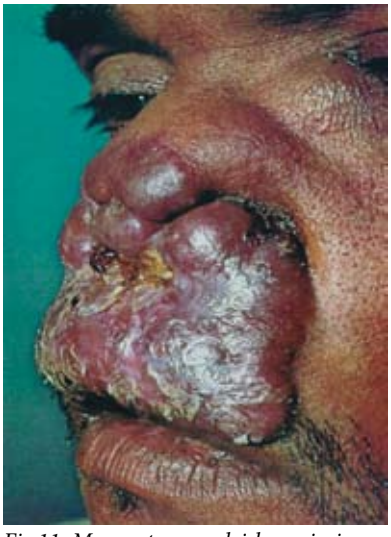

Fig.11. Mucocutaneous leishmaniasis.

- visceral leishmaniasis, which affects the organs of the mononuclear phagocytic system, such as the lymph nodes and spleen.

There are various species and subspecies of *Leishmania*. The commonest old-world form is *L. major* or *L. tropica*.

The clinical picture begins with a small papule at the inoculation site, which enlarges into a nodule or plaque. This may become verrucous or ulcerate. The lesions are often solitary but may be multiple, with the formation of satellites in a lymphatic or sporotrichoid spread. These lesions can resolve spontaneously in people living in endemic areas or may become chronic and disseminate. The latter occurs more often in immunosuppressed patients with poor cell-mediated immunity.

Diffuse cutaneous leishmaniasis develops in the setting of infections with *L. aethiopica* and *L. amazonensis*. After a prolonged time period of years and decades some patients develop mucocutaneous disease. Additional forms of cutaneous leishmaniasis are *L. recidivans*, which follows a sporotrichoid pattern with dry erythematous plaques. *L. recidivans* is characterised by recurrences at the site of an original ulcer, generally within 2 years and often at the edge of a scar.

Diagnosis of leishmaniasis

The diagnosis is confirmed by tissue or skin histology which demonstrates the presence of amastigotes in dermal macrophages. This is sometimes found in dermal scrapings or fine-needle aspirate (FNA) of affected tissue – so-called Leishman-Donovan bodies in large histiocytes. However, in older lesions parasites may not be found. Here the delayed skin reaction test (Montenegro test or Leishman reaction), which uses leishmania antigens to induce a cell-mediated (CMI) response can be an important diagnostic tool.

This test is positive in 50% of patients with cutaneous and mucocutaneous leishmaniasis. It is negative in diffuse leishmaniasis. Another drawback is that the test does not distinguish between past and current infection. Other adjunctive tests are tissue culture, ELISA and PCR.

Treatment of cutaneous leishmaniasis

Treatment depends on the type and severity of infection. Old-world disease is often self-limiting. Severe cases of *L. tropica* and *L. major* can be treated with pentavalent antimonials. New-world disease, e.g. *L. brazilienzes*, can progress to mucocutaneous disease. Treatment of choice is pentavalent antimonials, e.g. sodium stiboglutamate or meglumine antimonials.

Adjunctive treatments for cutaneous and mucocutaneous lesions include heat and cryotherapy, and drugs such as itraconazole, amphotericin B, ketaconazole and allopurinol. Prevention measures include insect repellants, insecticides and destruction of animal reservoirs.

Parasites not confined to the skin include onchocerciasis, loiasis, the guinea worm, schistosomiasis, cutaneous amoebiasis and the cutaneous involvement in trypanosomiasis. These are listed in Table II and depicted in Figs 12 - 14.

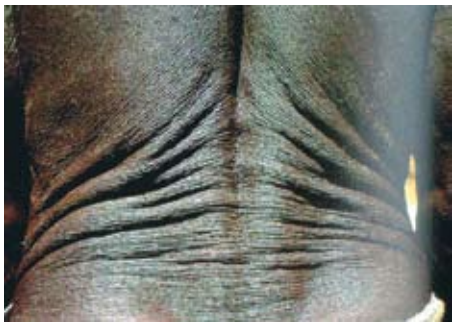

Fig. 12. Skin changes of onchocerciasis. Thickened and excoriated from chronic scratching.

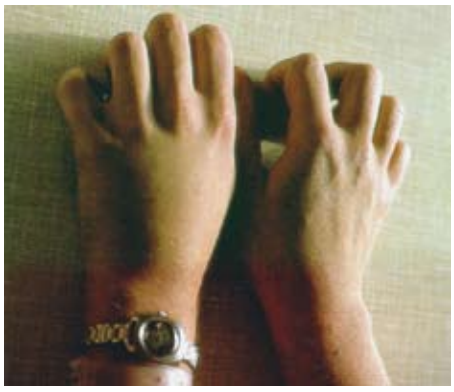

Fig. 13. Calabar swelling of loiasis.

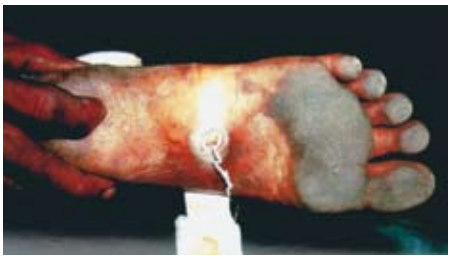

Fig. 14. Typical guinea worm.

Table II. Summary of parasitic diseases not confined to the skin

Disease	Pathogen	Vector	Geographic distribution	Diagnostic tests	Disease and complications	Treatment
Onchocerciasis (Fig. 12)	*Onchocerca volvulus*	Blackflies Simulium Along free-flowing rivers Larvae develop well in aerated water	Equatorial Africa, Central and South America, Yemen	Skin snips for unsheathed microfilaria DEC (Mazotti test)	Chronic pruritis and excoriations Eye involvement with gradual impairment of vision and blindness (river blindness)	Ivermectin effective against microfilaria Adjunctive doxycycline sterilises female worm Add systemic steroids in cases of eye involvement Suramin for adult worms
Loiasis (Fig. 13)	*Loa loa*	Chrysops flies	West and Central Africa	Microscopy of day blood for microfilaria	Calabar swellings (migrating swelling) Transient subcutaneous nodules often on the arm Irritation of eye as an adult worm traverses the sclera	Oral DEC 1 - 6 tabs dly for 2 weeks Repeated courses are necessary Ivermectin
Dracunculosis (Fig. 14)	Guinea worm *Dracunculous medinensis*	Ingest larva in contaminated water containing cyclops	Africa, Latin America, India	Clinical: see adult worm stringing out of skin ulcer	Ingested larva reach the skin, where adult worm literally breaks through	Excision and extraction Metronidazole (anti-inflammatory more than antihelmintic) Wound care
Cutaneous schistosomiasis	*S. haematobium* *S. mansoni* *S. japonicum*	Humans are infected by contact with fresh water The parasite penetrates intact skin Water snails are intermediate hosts	Haematobium North Africa Middle east Sub-Sahara Mansoni Sub-Saharan Africa Middle East Brazil Caribbean Japonicum China Philippines Indonesia	Identification of viable eggs Microscopy of terminal urine in *S. haematobium* Stool in *S. mansoni* and *S. japonicum* Eggs from all on rectal biopsy Serology: does not distinguish acute from past infection	Papules, nodules Cercarial dermatitis (swimmer's itch) Main pathology is granuloma formation around eggs Katayama fever: development of adult worms and the early stages of egg deposition, days to weeks after infection May cause severe systemic reaction including fevers, rigors, myalgia, urticaria, lymphadenopathy and hepatosplenomegaly High eosinophilia Chronic established disease: granulomatous disease affecting all organs	Praziquantel 40 mg/kg/day stat Sometimes repeated Systemic steroids in Katayama fever Avoidance of water in endemic areas Snail control

Table II. Continued

Disease	Pathogen	Vector	Geographic distribution	Diagnostic tests	Disease and complications	Treatment
Trypanosomiasis	American *T. cruzi* (Chagas' disease) African *T. rhodesiense* (acute) *T. gambiense* (chronic)	American: Reduviid bug Occasionally contaminated blood Tsetse fly (Glossina)	Tropical America Tropical Africa	American: In acute stage stage micro exam for trypomastigotes in blood specs Thereafter: PCR African: Detection of trypanosomes in blood film, chancre, lymph node aspirate, buffy coat, bone marrow or CSF PCR	Clinical includes a necrotic chancre at the site of inoculation, pruritis in the later stage, and 'trypanides', more or less discoid or annular erythematous eruptions African Trypanides Cervical lymphadenopathy In American Tryp Affects ANS, GIT and CVS systems Myocarditis is critical in these patients When conjunctiva is the portal of entry oedema of the palpebral and periocular tissue is seen – Romana's sign Chagoma: painful nodule at site of inoculation	Nifurtimox (with gamma interferon) Suramin, pentamidine Eflornithine WHO control measures
Filarial elephantiasis	*Wuchereria bancrofti, Brugia malayi*	Mosquitoes Genus: *Aedes*	Africa, West Indies	Microfilaria in peripheral blood at night	Thickened oedematous skin	Ivermectin Adjunctive albendazole, doxycycline Surgical correction

DEC = diethylcarbamazine; ANS = autonomic nervous system; GIT = gastrointestinal; CVS = cardiovascular system; CSF = cerebrospinal fluid; PCR = polymerase chain reaction.

In a nutshell

- Skin pathology often provides important clues to systemic infections.
- Parasitic infestations are common in the tropics due to a combination of heat, humidity and ultimately poor socioeconomic and health care conditions.
- Parasitic infections can be solely confined to the skin, as seen with human scabies, cutaneous larva migrans, the chigger flea, cutaneous myiasis and cutaneous leishmaniasis.
- Parasites not confined to the skin include onchocerciasis, loiasis, the guinea worm, schistosmiasis, cutaneous involvement in trypanosomiasis.