CHAPTER 10.2

Parasitic infections of the skin

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10.2.1 INTRODUCTION

A wide range of parasitic infections can involve the skin and subcutaneous tissues. Depending on the species of parasite, this involvement may be transient, the parasite passing through the skin on its migration to the blood stream and so to a specific target organ, or the infection may be localised to the skin. In the latter infections, the skin may be the primary site of infection or there may be a secondary invasion of the skin. All parasitic groups (protozoa, trematodes, cestodes, nematodes and arthropods) have species which can involve the skin or subcutaneous tissues:

10.2.2 PROTOZOAN INFECTIONS

Protozoan infections, in which there may be a transient skin phase following infection through the cutaneous route, include African trypanosomiasis (sleeping sickness due to *Trypanosoma brucei rhodesiense* and *T. b. gambiense*) and South American trypanosomiasis (Chagas' disease, due to *T. cruzi*). In the former disease, a vesicular lesion develops about five days after an infected tsetse fly bite and is known as a Sleeping Sickness Chancre. In Chagas' disease the lesion is similar and is termed a chagoma. In Chagas' disease too, there may be a marked, often unilateral, periorbital oedema termed Romana's Sign and which is due to the irritation of the conjunctiva by the infected assassin bug faeces and the penetration of the infecting trypanosomes.

Protozoan infections, which cause localised skin lesions, include various forms of cutaneous leishmaniasis. Commonly recognised species include *Leishmania tropica* (Oriental Sore); *L. braziliensis* (Espundia*)* and *L. mexicana* (Chiclero Ulcer) but there are many species and subspecies of the genus *Leishmania* causing regional variations of cutaneous leishmaniasis [1]. The geographical distribution of cutaneous leishmaniasis is wide, encompassing Africa, the Middle East, the Mediterranean, SE Asia, Asia and Latin America[1,2].

The clinical picture is also variable. In Old World cutaneous leishmaniasis, the lesions are typically single or few in number, while in some of the New World forms of the disease, lesions tend to be diffuse or mucosal in distribution[1]. In cutaneous leishmaniasis, the lesions tend to eventually heal but leave extensive and, disfiguring scarring. *Leishmania donovani* and *L. infantum* are the causes of visceral leishmaniasis, which involves the liver and spleen. However*, L donovani* can cause secondary skin lesions, especially post treatment.

Entamoeba histolytica, the cause of amoebiasis, can also involve the skin in the form of *amoebiasis cutis*. These lesions develop as secondary lesions from direct extension of an amoebic liver abscess or from the rectum[3]. *Balamuthia mandrillaris* , one of the free-living limax amoebae, while usually recognised as involving the CNS[4], may also invade the skin causing extensive skin lesions[3]

Diagnosis of protozoan skin infections usually involves a biopsy from the edge of the lesion with histological identification of the protozoa. Cutaneous leishmaniasis can also be diagnosed by culture on media such as NNN Medium[1].

In the treatment of severe cutaneous leishmaniasis, stibogluconate sodium 20mg antimony/kg/day iv or im as a single dose or in two divided doses for 20 - 28 days can be used, but serious side effects are common [5,6]. Alternative treatments include meglumine antimonite, itraconizole, ketoconazole or liposomal amphotericin B [1,5,6].

Amoebiasis cutis is treated with metronidazole as for invasive amoebiasis (Chapter 7) but there is no known and accepted, effective treatment for *Balamuthia mandrillaris* infections, which, fortunately, are extremely rare.

10.2.2 TREMATODE INFECTIONS

Trematode infections involving the skin are few. Thus in schistosomiasis invading cercariae may cause a transient allergic rash lasting 1-5 days while penetrating the skin (known as cercarial dermatitis) en route to the blood stream. This tends to occur most frequently with *Schistosoma japonicum*, less frequently with *S. mansoni* and rarely with *S. haematobium*.. It is more often seen in previously unexposed people who acquire a heavy infection while swimming. The non-human schistosome species (mostly avian species) can also cause a cercarial dermatitis – known as swimmer's itch or pelican itch. These latter infections are diagnosed clinically, including a history of exposure to infected water. They are self-limiting and never develop beyond the skin itching stage. They can thus be treated symptomatically with antihistamines to relieve the itch.

10.2.3 CESTODE INFECTIONS

A number of tapeworm species may cause subcutaneous lesions in their larval stages. Thus tapeworm cysticerci (*Taenia solium*), hydatid cysts (*Echinococcus granulosus*) and sparganum larvae (*Spirometra* spp) may all be seen as forming cysts under the skin.

Cysticercosis: This occurs when humans swallow the eggs of the pork tapeworm, *Taenia solium*. The eggs may be on salad plants contaminated with infected human faeces (heteroinfection); they may be ingested from the patient's own contaminated fingers (external autoinfection) or the eggs may be regurgitated up in a proglottid from a worm in the patient's intestine and then re-swallowed from the stomach (internal autoinfection)[7,8].

The eggs hatch and the onchosphere larvae migrate through the intestinal wall to settle at various locations through the body where they develop into cysticerci. In some cases, cysticerci may settle in the subcutaneous tissues, appearing as small lumps under the skin. Diagnosis can be made by identifying the larva (known as *Cyticercus cellulosae*) microscopically after biopsy or on histological section[7]. Once they have calcified, cysticerci can be seen on x-ray, CT scan or MRI. Diagnosis can also be confirmed serologically (eg EIA). Treatment for subcutaneous cyticercosis is usually unnecessary but where cysticerci lodge in a vital organ (eg the CNS) then treatment with albendazole (15mg/kg/day in three doses for 8-15 days) or praziquantel (20mg/kg tid for 14 days) can be used[5,6]. Corticosteroids are often recommended to be used in conjunction with anthelmintic treatment[5].

Hydatid: Humans acquire hydatid cysts (the larval stage of the tapeworm, *Echinococcus granulosus*) when they ingest eggs passed by dogs and other canids such as the dingo in Australia. The common sites for hydatid cysts in humans are the liver and the lungs but the CNS, the spleen and other organs may be involved[7]. Subcutaneous cysts may occasionally be encountered. These cysts may grow to a large size and so impair the function of the organ (or even destroy it) in which they are sited. Under the skin they cause little clinical discomfort but if ruptured (eg in a fall), anaphylaxis and the formation of metastatic cysts may occur. Diagnosis is clinical, radiological or using ultrasound and confirmed by antibody serology. Suspected hydatid cysts should never be aspirated or biopsied due to the danger of metastatic spread. Treatment is surgical removal or, if inoperable, the use of albendazole (400mg bd by mouth for 28 days or longer). Repeat treatments may be necessary, with 14 days rest periods between each course)[5,6].

Sparganosis: This is infection with the sparganum (plerocercoid) larva of one of the pseudophyllidean tapeworm species. The larvae may occur in the eye, the CNS, muscle or in the subcutaneous tissues, presenting as nodules or a mass in the organ involved. While sparganosis has been recorded sporadically from many parts of the world, the commonest species is *Spirometra mansonoides* in Asia with infection resulting when humans accidently ingest the minute crustacean, *Cyclops,* with water. The cyclops contains the procercoid larva of the cestode. Infection may also be acquired from ingestion of infected fish, amphibians, reptiles, birds or even pigs[9,10]. The most severe form of sparganosis is that caused by *Sparganum proliferatum.* The life cycle of this parasite, as is the case of most species causing human sparganosis, is unknown. The latter species is unusual in that the sparganum proliferates in human tissues. Diagnosis is clinical with a definitive diagnosis of sparganosis relying on parasite identification. No effective drug therapy is known and treatment relies on surgical removal [5,9,10].

10.2.4 NEMATODE INFECTIONS

Various nematode species, often zoonotic species infecting humans accidentally, can cause subcutaneous lesions. Nematode species infecting the skin include *Gnathostoma* and the filarial species, *Loa loa*, *Onchocerca volvulus*, *Mansonella streptocerca* and

Dirofilaria spp. Additionally, many nematode species, which infect through the skin, may cause skin lesions at the point of entry.

Thus the human hookworm species, *Ancylostoma duodenale* and *Necator americanus*, may cause a transient itchy papular rash (ground itch/dew itch) at the site of penetration. Although they mostly never mature to adults in the human host, the dog and cat hookworms (*Ancylostoma braziliense, A. caninum* etc), may cause serpiginous itchy tunnel-like lesions in the skin (sandworm/cutaneous larva migrans/creeping eruption) lasting for weeks or months. So too, *Strongyloides stercoralis* larvae can cause similar lesions (*larva currens* rash) while penetrating the skin when autoinfection occurs. In the latter case, the rash lasts a few days before disappearing as the infective larvae of the *Strongyloides* enter the circulation to perpetuate the infection. Such *larva currens* rashes may occur at irregular intervals for months or years after leaving an endemic region, sometimes for 20 –50 years or longer.

Gnathostomiasis is caused by various species of nematode belonging to the genus *Gnathostoma*. Transmission usually occurs after ingestion of fresh water fish or sometimes snakes[8]. Clinically, gnathostomiasis presents as swellings, which might be migratory, in the tissues including subcutaneous tissues[5].

10.2.4.1 Filarial infections

10.2.4.1.1 Onchocerciasis ("river blindness")

Onchocerciasis is caused by infection with the filaroid *Onchocerca volvulus.* It is endemic in 37 countries in West, East and Central Africa, the Arabian peninsular, and parts of Central and South America. The global burden is estimated to be around 126 million, mostly in Africa [11]. Onchocerciasis is the second most common infectious cause of blindness after trachoma and causes immense misery and socio-economic loss in infected communities, especially in areas of heavy transmission where infection occurs early in life. The fear of blindness drives people away from the best arable land near the rivers and streams where the vector black flies, (*Simulium* spp.), breed. Work output is reduced because of the number of sight-impaired people in the community. *O. volvulus* follows the general life cycle of filarial nematodes but there are important differences between it and the life cycle of *W. bancrofti* and *Brugia* species: *Onchocerca volvulus* microfilariae are found in the skin not the blood and they do not do not have sheaths. The adult worms live in the subcutaneous tissue and not the lymphatics. The vectors are black flies (*Simulium* spp.) not mosquitoes.

The main clinical features of onchocerciasis are: dermatitis (onchodermatitis), skin nodules, and eye lesions [11]:

Dermatitis: Various combinations of rashes, hyper-pigmentation, atrophic changes and micro-abscesses are seen. There is intense itching and secondary bacterial infection may occur during scratching. Some patients may suffer from hyperreactive onchodermatitis or "sowda" where itching is so intense that daily activities are impaired and normal sleep

patterns are disrupted[12]. Chronic dermatitis causes premature aging of the skin and patches of de-pigmentation (a condition called "leopard skin"). Atrophy of subcutaneous tissue in the groin area may lead to sagging of the skin, the so called "hanging groin". The skin lesions of onchocerciasis are sometimes confused with contagious skin diseases by local populations and sufferers may be stigmatised by their community.

Nodules: Nodules are subcutaneous granulomas resulting from tissue reactions around adult worms. They are usually less than 2cm in diameter, firm to the touch, mobile and neither tender nor painful. They tend to be commoner on the head in Latin America and around the groin ('hanging groin') in Africa.

Eye lesions: The exact cause of the eye lesions has yet to be determined but a number of factors are known to be involved: tissue reaction to microfilariae migrating through the eye, allergic reactions to parasite secretions, and the impact of other irritating agents such as ultraviolet light, dust and smoke. There is progressive loss of vision that can lead to irreversible blindness.

As is the case in lymphatic filariasis, cellular immune responses against a range of antigens are reduced and IgG4 dominates the isotype pattern in classical onchocerciasis [13,14]. Tetanus vaccine response is impaired but not enough to prevent the production of a protective response [15]. By contrast, the immunological profile in the sowdah variant is analogous with the hyperreactivity seen in the TPE variant of lymphatic filariasis with a marked increase in antibody levels and increased cellular responses to filarial antigens[16].

The most common method of diagnosis is by the use of skin snips. These should be obtained from the iliac crests and lower legs of African patients and from the shoulders of patients from the Americas. The classic method is to clean the skin with an alcohol swab, leave to dry, and then elevate a portion of the skin with the point of a needle and cut out a portion 2-3 mm in diameter with a scalpel or razor blade. The biopsy should be blood-free. The biopsy is placed into a drop of water/saline on a microscope slide, covered with a cover glass, and examined microscopically after around two hours. If the person has onchocerciasis motile microfilariae will emerge from the skin. The number of microfilariae per gram of skin can be determined by weighing the biopsy and counting the number of microfilariae [11]. Antigen and antibody assays, rapid card tests and PCR techniques for the diagnosis of onchocerciasis are under development but are not routinely available as yet [17-19].

The first drug to be successfully used for the treatment of onchocerciasis was suramin which was introduced in the late 1940's. It kills both adult worms and microfilariae, but is extremely toxic and severe side effects, including some deaths, were seen in up to 30% of treated patients. Diethyl-carbamazine (DEC) was also introduced in the 1940's. It is very effective in killing microfilariae but again, severe side effects are a problem and it increases the risk of eye damage. Ivermectin is now the drug of choice. It is given orally as a single dose of 150-200μg/kg body weight at 3 - 12 monthly intervals[5,6]. Adverse reactions still occur in about 20% of treated patients but they are usually mild and self-limiting and confined to transient increase in skin rash and itching, soft tissue swelling,

arthralgia, fever, and lymphadenitis. Some patients may experience peri-orbital oedema and rarely, hypotension, bronchospasm, and bullous eruptions. Adverse reactions are more common during the first round of treatment and reduce in prevalence and seriousness with later rounds because of the reduced number of microfilariae [11]. It is recommended that where infection involves the eye, oral corticosteroids (1mg/kg/day) should be given for several days before ivermectin is started[5]. Over the last two decades a control programme for onchocerciasis has been underway in Africa and the Americas, based on the community-wide, mass administration of ivermectin and vector control measures. As a result, onchocerciasis has been eliminated as a health problem in 11 West African countries, and the rest of the endemic areas of that continent, and those in the Americas are being progressively brought under control[11, 20-22]. Nodules on the head are associated with an increased risk of eye damage and extensive nodulectomy campaigns have been carried out for many years in the Americas and have been effective in reducing the prevalence of blindness. In Africa, the nodules tend to occur on the trunk, pelvic areas and upper legs rather than the head and nodulectomy is not an effective means of control[11,22].

10.2.4.1.2 Loasis

Loiasis, caused by infection with *Loa loa* infects 3 to 13 million people in Western and Central Africa. It is also the most common filaroid parasite seen in travellers and other expatriates. It is transmitted by the bite of female flies of the genus *Chrysops* and has a typical filaroid life cycle. The adult worms actively migrate through subcutaneous tissue and sheathed microfilariae appear in the blood during the day. Many of the infected individuals are asymptomatic despite having circulating microfilariae. Expatriates seldom develop microfilaraemia but they can suffer from a range of allergic symptoms such as pruritis, urticaria, and transient angiodema or “Calabar swellings. Calabar swellings can occur anywhere on the body but are most common on the face, arms and hands. Loiasis is often accompanied by marked eosinophilia and high serum IgE. Eosinophilia and a history of travel to a *Loa loa*-endemic area is often the first indication that someone may have the disease. Sometimes a migrating worm may be observed crossing the conjunctiva giving rise to the common name “eyeworm”. Patients may be alarmed, but apart from mild transient local inflammation the worm causes no long-term damage to the eye [23]. Renal involvement, as revealed by haematuria and/or proteinuria may occur in up to 30% of loiasis cases and may be exacerbated by treatment [24]. The most serious complication is encephalitis. It is most commonly precipitated by treatment of individuals with microfilarial counts >5000 per ml of blood and is caused by a rapid increase in antigen shed from the dying microfilariae.

A definitive diagnosis is obtained if an adult worm is removed from the eye by surgery or if characteristic microfilariae are obtained from blood collected during the day[23,25]. Microfilarial density can be low and it is advisable to use concentration tests as per diagnosis of lymphatic filariasis. PCR-based tests have been developed but are only available at a few specialised centres[25,26]. Testing for antifilarial antibody is of little value in endemic populations but is of value in expatriate cases were the absence of such antibody makes loiasis unlikely [25].

Medical practitioners are urged to obtain up to date information from a tropical medicine specialist or the Centers for Disease Control in the USA before treating a case of loiasis because there is a risk of severe and even fatal complications and drug treatment regimes are under review. DEC is effective against both adult worms and microfilariae and is the drug of choice in amicrofilaraemic cases or when microfilaria density is light, including expatriate cases. It is given at a dose building up to 9 mg/kg of body weight for 21 days[5,6]. Multiple courses may be required and recrudescence may occur years later. Mild side effects are common and include Calabar swellings, pruritis, nausea and fever. The treatment irritates the worms and the may be observed moving around under the skin. It must be stressed that treating *Loa loa* in patients with microfilaraemia carries some risk, and if the microfilaraemia density is over 5000 per ml the risk of severe or even fatal shock, renal failure and encephalitis is very high. "Graduated doses" of DEC and pre-treatment with antihistamines and steroids are recommended[5,6] to try and reduce the incidence of severe adverse reactions but experience shows that they do not prevent encephalitis [27]. The removal of microfilariae by apheresis has been used to lower the risk but the technology is not available in many *Loa loa*-endemic areas [28]. Ivermectin (200 ug/kg stat, repeated every 6 – 12 months)[5] kills microfilariae effectively but it too has been reported to cause adverse effects [24]. Trials with albendazole are underway and the early results look promising. Adverse effects do not occur because the microfilariae decrease slowly over several months rather than rapidly as is the case with DEC and Ivermectin but there is still work to been done on defining the dose and treatment regime [29,30].

10.2.4.1.3 Mansonellosis

Mansonellosis is caused by filarial nematodes belonging to the genus *Mansonella. Mansonella perstans* (formerly *Dipetalonema/Acanthocheilonema perstans*), is transmitted by a number of species of midges belonging to the genus *Culicoides*, and infects monkeys and apes as well as man. The parasite is endemic across Central Africa, Tunisia, Algeria, the Northern Coast of South America, and parts of Brazil and Argentina. It is often considered a commensal but is better regarded as a low-grade pathogen. This species of the genus does not infect the skin, but can cause an allergic response from the host and can be associated with angioedema (similar to the Calabar swelling in loiasis), pruritis, fever, headache, arthralgia and right upper quadrant pain. Eosinophilia and elevated serum IgE are common. The unsheathed microfilariae, which show no periodicity, are found in the blood but concentration techniques may be required to find them. Antifilarial antibodies are elevated. The standard treatment is DEC at 8-10mg/kg body weight for 21 days but there is little evidence that it effective and multiple courses may be required [99]. Mebendazole given at 100mg per day for 30 days is effective but ivermectin and albendazole are not [23].

Mansonellosis can also be caused by two other species of the genus. *Mansonella ozzardi* is confined to the New World with a wide distribution in Northern South America, the West Indies, and the Caribbean. It is transmitted by either *Culicoides* spp midges or simuliid blackflies while *Mansonella streptocerca* (formerly *Dipetalonema streptocerca*)

is found in both humans and monkeys in Central Africa. Adult worms live in the thoracic and peritoneal cavities and the sheathless, aperiodic microfilariae are found in blood and skin. Most cases are asymptomatic but there are reliable reports of marked eosinophilia, urticaria, pruritis, lymphadenopathy, headache, joint pains and asthma-like symptoms, especially with *M. ozzardi* [23]. DEC and albendazole are ineffective but ivermectin reduces the microfilarial load and symptoms [32].

10.2.4.1.4 Dracontiasis

Dracontiasis is infection with the Guineaworm, *Dracunculus medinensis*, a filarial nematode which is unlike the other worms of this group in that it is transmitted through the ingestion with water, of the intermediate host, a small freshwater crustacean, *Cyclops.* The infection has been widespread, but often localised, in Asia and the Middle East in the past, but the WHO has hopes that it will be eradicated in the near future.

The long, thin adult worms settle under the skin and muscular tissues (often on the leg) and the female worm extrudes her posterior end through a blister/ulcer when the limb is immersed in water. The worm liberates larvae into the water where they are taken up by the *Cyclops*.

The lesions can be extremely painful and diagnosis is clinical, noting of the end of the female worm emerging from the skin or by x-ray. Treatment involves the use of metronidazole 250mg td for 10 days for adults or thiabendazole 25-37.5 mg/kg bd for 3 days[5,6].

10.2.4.1.5 Miscellaneous Zoonotic filariases involving the skin

A number of animal, mosquito-borne filaroids cause occasional infections in humans, the most common being species of *Dirofilaria,* especially the dog heartworm *D. immitis.* Their life cycle cannot be completed in humans and the parasites in incorporated in a granuloma or a calcified lesion. They can be associated with skin lesions but if they occur in the lung they can be mistaken for tuberculosis or the “coin lesions” of malignancy. Occasional patients may present with eosinophilia and allergic symptoms. Diagnosis is usually made microscopically after excision and histology. Antifilarial serology results are variable [33].

10.2.5 ARTHROPOD INFECTIONS/INFESTATIONS OF THE SKIN:

Arthropods are hard bodied animals with jointed legs. They comprise insects, mites, ticks, spiders and crustaceans. The medical significance of various species relates to their blood-sucking habit; their ability to serve as vectors to transmit other microorganisms; their role as intermediate hosts in the life cycles of other parasites and in some cases, the fact that they are venomous/toxic[34].

Arthropods can thus be either temporary or permanent ectoparasites and infest the host (i.e. live on the surface of the skin of the host) or they can infect the host (live within the skin of the host). Some might be better described as micropredators as they actively hunt their host (eg mosquitoes; tsetse flies).

Where they transmit a disease-causing organism, the transmission may be mechanical, as when the microorganisms contaminate the feet or mouthparts of the arthropod, or biological, where the microorganisms pass through a biological cycle in the vector.

10.2.5.1 Insect infections/infestations

Many insect species can infest/infect the skin[34,35]. This can vary from a simple accidental bite (eg from a thrips or collembolan[34,36]) to a more severe infection (eg from a fly maggot, jigger flea or a louse infestation). Thrips and collembolans are free-living, plant feeding insects which occasionally bite humans, become encapsulated in small nodules in the skin or cause a pruritic rash. Insects which suck human blood include the sucking lice (Anoplura); blood-feeding bugs (Hemiptera); fleas (Siphonaptera) and the blood-sucking flies (Diptera). Some lepidopteran species (caterpillars of moths) have irritating hairs which can cause an allergic rash on contact with the skin and some beetles such as the meloids and staphylinids (blister beetles), *Paederus* spp. (containing paederin), *Lytta* spp.(the Spanish fly, containing cantharidin) and *Epicauta* spp. can also cause blistering on contact with the skin[34].

10.2.5.1.1 Louse (Anoplura) infestations

Three species of lice infest humans - the bodylouse (*Pediculus humanus corporis*), the head louse (*P. h. capitis*) and the pubic or crab louse (*Pthirus pubis*)[34,35]. Animal lice do not attack humans, these insects being very host specific. Transmission is by close head contact (headlice); close contact and sharing of blankets and clothing (bodylice) and sexual contact (crablice).

Lice feed on the blood of the host and their feeding can cause itching, inflammation and skin discoloration (Vagabond's Disease). Of the three species of human louse, only the body louse is a significant vector of disease, transmitting epidemic relapsing fever (*Borrelia recurrentis*), epidemic typhus (*Rickettsia prowazekii*) and trench fever due to *Bartonella* (= *Rochalimaea*) *quintana*.

Diagnosis of pediculosis and phthiriasis is by direct observation – detecting the lice or their eggs (nits) on the hairs of the head (headlice) or the pubic, under arm, chest, and beard hairs or on the eyelashes (crablice). In the case of the bodylice, which are morphologically identical to the headlice, the adult lice can be found on the body or the clothing of the victim. Eggs of bodylice are laid in the seams of clothing.

Treatment and control of headlice involves the use of specially formulated, low concentrations of insecticides such as 0.5%malathion (Maldison) or pyrethroids (eg 1% permethrin)[35,37,38] for cases and close contacts (especially family members). Ivermectin

200ug/kg orally as a single does is also reported to be effective[6]. Alternatively, repeated use of standard hair conditioners can be used combined with rigorous inspection and combing of the hair with a fine tooth comb to remove adult lice and eggs[38]. Careful and repeated examination of the hair is an integral part for successful treatment and control as is treatment of close contacts. For pubic lice, treatment of the patient and all sexual partners with 1% permethrin is the usual approach, although 1% lindane is still suggested as an alternative by some authorities[6]. For bodylice, treatment of the body with pyrethrin or benzyl benzoate is suggested but for control[5], fumigation of clothing, blankets etc is required.

10.2.5.1.2 Blood-feeding bugs (Hemiptera)

Blood-feeding bugs include the assassin (cone-nosed) bugs belonging to the genera *Rhodnius*, *Triatoma* or *Panstrongylus* and the bedbugs (*Cimex* spp)[34]. The cone-nosed bugs of Latin America are vectors of *Trypanosoma cruzi*, the cause of Chagas' disease. They transmit the protozoan to the human host through their faeces, contaminating the bite wound, often on/near the conjunctiva. The bugs often feed around the eye and the infected faeces are irritant, resulting in the diagnostic sign for early South American Trypanosoamiasis, Romana's Sign.

Bedbugs inhabit houses, feeding on the sleeping inhabitants. Their bites can be itchy but they are not known to be significant vectors of any disease.

10.2.5.1.3 Fleas (Siphonaptera)

Many flea species bite humans, suck blood and may cause allergic reactions of varying degrees of severity[34]. The common fleas attacking humans are the dog and cat fleas (*Ctenocephalides canis* and *Ct. felis*); the human flea (*Pulex irritans*) and rat fleas of the genus *Xenopsylla*. The bite of these insects is irritating but not of much clinical significance. Of great importance, however, is the fact that fleas of the genus *Xenopsylla* can serve as vectors of *Yersinia pestis*, the causative agent of Bubonic Plague, and *Rickettsia typhi*, the cause of murine typhus.

Another flea worthy of consideration in tropical areas is the Jigger (Chigoe) flea, *Tunga penetrans*[34,35]. This flea differs from other fleas attacking humans in that the female flea burrows into the skin and lives there as a permanent parasite within an ulcer-like lesion. The female of this flea lives in skin lesions in pigs, rodents and other animal hosts in tropical South America, and it has been introduced into Africa and India as a result human movements. Eggs laid by the female flea fall onto the ground and develop into free living larvae which pupate and emerge as adults. Mating occurs and the females find a host, burrow into the skin, often on the foot and between the toes. The life cycle then repeats itself. Diagnosis is by finding the embedded female flea and treatment consists of removal of the flea taking care to avoid the risk of secondary bacterial infection including tetanus.

10.2.5.1.4 Blood-feeding flies and myiasis (Diptera)

Blood-feeding flies comprise the biting midges (*Culicoides* spp. vectors of perstans filariasis); black flies (*Simulium* spp, vectors of onchocerciasis); tsetse flies (*Glossina* spp, vectors of African trypanosomiasis); horseflies (*Chrysops* spp., vectors of loiasis) and the mosquitoes, belonging to the dipterous family, Culicidae)[34]. One can classify these insects as temporary ectoparasites (such as mosquitoes, blackflies, biting midges and tsetse flies or.alternatively, mosquitoes and tsetse flies are sometimes classified as micropredators as they actively seek out their hosts.

Myiasis is infection of the human host with fly larvae (maggots)[35,39,40]. A range of blowflies (Calliphoridae) have maggots that can, if the opportunity arises, attack human flesh. Most of these derive from eggs or maggots deposited in open wounds on the skin – especially septic wounds. The maggots then live in the wound until mature, drop off and pupate on the ground. Most of these flies attack putrid flesh only, and in fact these specific species of fly maggots have been used in the past (and are being used once more today) in the treatment of such wounds because they attack only dead tissue and thus clean up the wound. Additionally, their secretions contain antibacterials which help resolve and prevent infection of the wound. Other species, such as the screw worm flies (eg *Callitroga* spp), however, are invasive and can attack healthy skin or wounds and destroy healthy skin often causing severe invasive damage.

Of particular interest in relation to human myiasis, however, are the Putsi (or Tumbu) fly of tropical Africa and the Human Botfly (*Dermatobia hominis*) of Central and tropical South America. Of these, the Putsi fly lays its eggs on clothing laid out to dry on the ground while *Dermatobia hominis* lays its eggs on female mosquitoes or ticks. The eggs hatch as a result of the warmth from the human body when the clothes are worn or when the tick or mosquito feeds. The maggots develop in pustular, boil-like skin lesions for about 7-10 days, after which the fully developed larva emerges, drops to the ground and pupates, eventually emerging as the adult fly. Mostly these skin lesions are benign, although scarring can result when the maggot drops out of the skin. Sometimes, however serious complications or even death may result when a maggot on the head penetrates the skull and invades the brain. Heavy maggot fly infections have been misdiagnosed as chickenpox or even, in the past, as smallpox.

Various forms of myiasis are recognised[39]:

- Cutaneous myiasis – calliphorid blowflies; putsi flies, human botflies
- Gastrointestinal myiasis – calliphorid blowflies
- Ophthalmomyiasis – sheep nasal bot fly, *Oestrus ovis*[41]; screw worms
- Genitourinary myiasis – calliphorid blowflies.
- Sanguinivorous myiasis – the Congo Floor Maggot

Sanguinivorous myiasis due to *Auchmeromyia luteola* (The Congo Floor maggot) in Central Africa is different from the others in that the maggots behave rather like bedbugs. They inhabit rodent burrows and occasionally houses with mud floors and come out at

night while the host sleeps to take a blood meal before retreating back into the floor cracks to moult[39,40].

Diagnosis of myiasis is clinical, with the species identification being based on the morphology of the posterior spiracular plate. Treatment is removal of the maggot – for skin lesions, smearing with Vaseline prior to squeezing out the maggot may be used.

20.5.2 Arachnid infections/infestations

The arachnids are 8-legged arthropods. The group includes spiders, scorpions, ticks and the mites. The first two are medically important as some species are venomous, while the latter two groups can be ectoparasitic blood-suckers and in some cases important disease vectors[34].

10.2.5.2.1 Mites

A number of animal parasitic mites can at times attack humans, often causing unpleasant allergic reactions which may be severe in some patients[34]. These mite species vary from the ubiquitous, non-parasitic housedust mite (*Dermatophagoides* spp.), allergy to which is widespread and an important cause of asthma-like attacks, to the wide range of animal parasitic mites that bite humans - trombiculid mites and bird or rodent mites, such as the genera *Cheyletiella* (dogs; cats; rabbits), *Ornithonyssus* (birds) , *Liponyssoides* (mice), *Dermanyssus* (birds) and *Leptotrombidium* (rodents) [34]. The latter species is the vector of Scrub typhus (Tsutsugamushi Fever) due to *Orientia tsutsugamushi*. Grain workers may also sometimes be attacked by free-living, mites of the genus *Pyemotes* which are universally present in barley, wheat, cotton seed, grasses and hay and stored tobacco[34]. These people often present with a mild to severe skin eruption which in some cases can even superficially resemble chickenpox[34].

Demodex folliculorum is the hair follicle mite of humans. It is a common parasite of humans and while mostly of no or little clinical significance, it can block hair follicles causing a ‘blackhead-like’ condition or a roseaceous skin rash[34].

The scabies mites are by far the most important parasitic mites of humans and scabies is the most important mite infection of humans. The cause is the human scabies mite, *Sarcoptes scabiei*. The animal scabies mites can sometimes infect humans, but most cases and outbreaks of scabies are due to the human strain. Clinical features of scabies involve two components – the tunnels in which the mites live (which occur predominantly on the wrists, the elbows, the ankles and around the thigh and genital region) and an allergic rash. The mite lesions itch unbearably and the rash tends to cover the body, but spare the face. Thus as a diagnostic rule of thumb, any person presenting with an itchy rash which covers the body but spares the face, can be considered to have scabies until proven otherwise. A problem which can arise from scabies is secondary infection of the lesions with *Streptococcus pyogenes* and the development of glomerular nephritis.

Transmission of scabies is by close personal bodily contact (hand holding; sexual contact etc) and blankets and clothing are not important in transmission. Scabies is worldwide and epidemics and pandemics seem to occur every few years.

While diagnosis can be confirmed by isolation of the mites from the burrows[42], this is not a practical procedure in most cases and diagnosis is usually clinical.

Treatment for scabies usually relies on 25%benzyl benzoate but alternatives include permethrin 5%, lindane 1% (contraindicated in pregnancy and during lactation), and 10% crotomiton[5,6,35.] Usually a single application is adequate, but sometimes a second treatment is needed after about 4-6 weeks if the infestation is not cleared. Care must be taken to avoid over-treatment as the rash, which is a hypersensitivity reaction to the mite and its waste products in the skin, may well remain after successful killing of the mites. This often convinces the patient (and the doctor!) that the treatment has failed. A good strategy to overcome this, is to treat with benzyl benzoate by covering the whole body except the face with the medication, and then giving the patient crotomiton (Eurax) cream to apply to spots where itching persists over the next few weeks. The crotomiton will relieve the itch and has some miticidal activity. All contacts also need to be treated if the infestation is to be controlled. Ivermectin 200ug/kg as a single oral dose has also been used for the treatment of scabies, but should be used with caution in the elderly because of increased toxicity[5] Mass treatments may be necessary to control scabies outbreaks in institutions.

10.2.5.2.2 Ticks

Ticks can be separated from the mites by their complex mouthparts, with a characteristic many-hooked hypostome. Two major families of ticks can be recognised, the Argasidae (soft ticks or tampans) and the hard ticks, the Ixodidae.

10.2.5.2.2.1 Argasid ticks

The argasids or tampans are blood-sucking ectoparasites of animals. They are known as soft ticks because the body is covered by a soft, leathery integument. They live in sand or the burrow of their usual animal host and feed intermittently. When humans intrude into their environment, they will attack the human host. Important species include *Ornithodoros moubata* and other species of the genus, which are the vectors of *Borrelia duttonii*, a spirochaete harboured by rats and which causes endemic relapsing fever in humans. The bat tampan of Africa, *Argas brumpti*, is found in caves inhabited by bats. When humans are bitten by this species, extensive bruising of the skin results from the powerful anticoagualant in the tick saliva. Such bites can cause an itchy rash, often lasting for weeks or even months[43].

10.2.5.2.2.2 Ixodid ticks

These are known as the hard ticks as they have a hard shield on the dorsal surface. There are many genera and species, the best known of which are the genera *Amblyomma,*

Aponomma, *Boophilus*, *Dermacentor*, *Haemaphysalis*, *Hyalomma*, *Ixodes*, and *Rhipicephalis*[34]. Tick bites are more of a nuisance that a hazard, but the complex mouthparts including the hypostome, anchor the tick firmly to the skin while it feeds and this makes removal a matter of care to avoid leaving the mouthparts embedded in the skin when the tick is removed. Removal of a tick is usually easily achieved by grabbing the tick as close to the skin as possible and removing it with a gentle pull. The applicationm of vaseline to the attached tick can help in the process of removal. For removal of small ticks (including the minute immature larval ticks), a pair of forceps can be of great help.

Tick bites can be irritating and can itch due to injected saliva. Mostly, however they are of minor clinical significance unless they become secondarily infected, which tends to happen especially if the mouthpart are detached during removal in which case a localised ulceration may develop. The engorging females of some species, however, may produce a salivary toxin which can paralyse the host and death will result unless the tick is removed. In humans, the patient develops a localised or ascending paralysis reminiscent of poliomyelitis from which it must be differentiated by finding the tick – which may be embedded in such obscure locations as the ear canal or eardrum, in the hair, under the armpit etc. Recovery commences with the removal of the tick. Such tick paralysis is recorded from Europe (*Ixodes* spp., *Rhipicephalis* spp*., Hyalomma* spp., *Boophilus* spp., *Haemaphysalis* spp.); the USA (*Dermacentor* spp., *Ixodes* spp.); Africa (*Ixodes* spp.; *Hyalomm*a spp.*)* and Australia (*Ixodes holocyclis*)[34]. In Australia, antivenom is available,

Various species of these hard ticks are important vectors of viral illnesses such as Congo-Crimean Haemorrhagic Fever, several viral encephalitides; rickettsial diseases such as Rocky Mountain Spotted fever[34], the Australian Spotted Fevers[44], Boutonneuse Fever; the spirochaetal Lyme disease and the protozoan disease of babesiosis[34].

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