Zoonotic parasites with significant importance for tropical dermatology

Review Article

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ABSTRACT

Zoonotic parasitosis is well known to predominate among poor societies in tropical and sub-tropical zones. Despite the comprehensive emergence and re-emergence of cutaneous zoonotic parasites, the clinical recognition of these types of infections is limited. Although most prevalent in tropical and sub-tropical zones, parasitic zoonosis in skin was reported in developed countries under suitable conditions. The non-specific symptomatology and lack of meticulous reports for the real burden were notable. Dermoscopy is a recent promising alternative in the diagnosis of cutaneous parasitosis, particularly by arthropods and helminths. In zoonotic protozoa, histopathological examination remains a cornerstone for proper diagnosis. Challenges in medical treatment are present in several cases; therefore, the search for alternatives is important. The present review aims to underline the epidemiological data, clinical pathology, and updates in the diagnosis and management of these dermal parasitic infections.

Keywords: Dermoscopy; histopathological; medical treatment; parasites; skin; zoonosis.

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INTRODUCTION

Tropical parasites are characterized by striking biology that causes health problems. Parasitic infections affect societies worldwide with substantial mortality rates, and overwhelming economic concerns[1]. Several studies navigate parasite flows and networks revealing the emergence and re-emerging of parasitic zoonosis[2-5]. Circulation of zoonosis is facilitated by exposure to biological, physical, and chemical factors. This might be due to the specific style of the work environment; for example, farmers in comparison to other professional groups suffer tick-borne diseases and zoonosis. In addition, low health education and limited accessibility to health care afflict several societies[6]. Particular exploration for more efficient anti-parasitic drugs mandates funding that is not always present in developing countries[7].

Dermatological zoonosis is characterized by miscellaneous lesions that can range from allergic dermatitis to physical trauma. These cutaneous lesions may be associated with mild local reactions or are related to severe systemic illness[8]. In this context, our scope was to define the common tropical parasites of zoonotic origin that cause dermatological lesions regarding their epidemiological data, clinical pathology, and updates in the diagnosis and management.

To launch the current review, the following objectives were intended: the available epidemiological data regarding different zoonotic parasites, the remarkable clinical pathology, the evaluation of diagnostic methods, and the updates and challenges of their management. Then, the draft protocol was reviewed in several series of discussions. Articles were gathered using the Egyptian Knowledge Bank (EKB), PubMed, Web of Science, and Google Scholar. Inclusion criteria were: peer-reviewed studies, systematic and narrative reviews, as well as case reports published in the period between 1990 and 2022. We also cited earlier studies if they were the definite source of data. Exclusion criteria were: research articles with closed access or online articles not related to a definite site of the database.

Zoonotic arthropods

Myiasis (*myia*, Greek word for "fly)

The chief zoonotic potential occurs in humans through skin invasion by the developing larvae (maggots) of Diptera. Infested larvae feed on dead or living tissues of the skin[9]. The recorded prevalence of the disease relies only on published case reports. Myiasis is considered a differential diagnosis of skin diseases in tropical poor communities due to bad hygiene and poor housing conditions[10,11]. However, human myiasis may also occur in countries with enhanced hygiene, as reported from New York[12], and in France[13]. Notably, in temperate countries, myiasis occurs in summer when fly breeding is at its maximum[14]. Predisposing factors involve skin cancers, diabetes, and vascular diseases[10,15]. Nosocomial infection with *Sarcophaga* larvae was recorded revealing the ability of maggots to reach to

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purulent wounds through dressings[16]. Prevalence is more frequent at the extremes of age due to bad self-hygiene[15,17].

The three main clinical types are known; migratory (creeping), localized furuncular (also follicular/nodular/boil-like) and wound (traumatic) myiasis. In the first two types, the temperature of the skin triggers the eggs to hatch. The larva pierces intact skin (obligatory myiasis) neighboring to a hair follicle to create a breathing pore in the skin while it is growing up. The condition starts as intensely itchy lesions similar to a mosquito bite. Over time, the lesions increase in size and are non-healing[17].

The furuncular type is mainly produced by larvae of *Cordylobia anthropophaga, Dermatobia hominis, Cuterebra* spp., and *Wohlfahrtia* spp. Furuncular lesions repeatedly discharge bloody-serous or sero-purulent fluid through the central punctum (pore) and then subsequently ulcerates[18]. The migratory type is usually caused by *Gasterophilus* spp. (horse botfly) and *Hypoderma* spp. (cattle grub) and the lesions are in the form of tunnels underneath the skin. In wound myiasis, larvae of obligatory myiasis such as *Chrysomya bezziana* (Old World Screwworm) (obligatory myiasis) feed on necrotic tissue and then invade through healthy living tissue causing devastating tissue damage that might be fatal[19]. In *Cochlyomyia* (New World Screwworm), eggs are laid at the site of the wound and hatched larvae invade through the bed of the wound into the neighboring healthy tissues[20]. Larvae of *Calliphora* and *Lucilia* are classified as facultative myiasis that feeds on the necrotic tissue without invading healthy tissues[21,22]. Interestingly, facultative myiasis is manipulated in some societies for maggot therapy[23]. A recent report demonstrated the beneficial use of *M. domestica* as an alternative to other commonly used larvae in maggot debridement therapy being more available and easier to breed. Moreover, it was confirmed that chitosan which is extracted from the cuticles of larvae was superior in accelerating wound closure[24].

Examination usually reveals the clear movement and intermittent protrusion of a small white structure from the central pore. The upper and lower limbs, the scalp, the forehead, and the back are the most affected sites[17]. With steady persistent pressure for 30 sec, the live larva expels without need for forceps, and the species is identified by size and morphology of posterior spiracles. Enlarged regional lymph nodes may be clinically present[25]. So far, species that cause non-specific (facultative) myiasis such as *Lucilia sericata* can be cultured in axenic media containing non-viable flesh as clean granulation tissue starves the larvae. Culture and sensitivity for bacteria and pathological examination are mandatory. Mixed perivascular infiltrates involving eosinophils are characteristic pathological findings.

Spontaneous expulsion may occur; yet the main goal of treatment is the whole elimination of maggots. Spines covering the body of the larva may anchor into surrounding tissues making surgical extraction a difficult task. Minor remnants can induce superinfections or granuloma formation. Alternatively, asphyxia of the larva through occlusion of the central pore of the lesion by heavy oil or paraffin is mostly recommended[26]. Eczematous pruritic halos surrounding lesions may exist post-treatment[27].

Entomodermoscopy made the colorful visualization, imaging, and whole extraction of maggots through the depth of the stratum corneum more feasible than the traditional methods[28]. The dermoscopy is of two types: 1) polarized dermoscopy which is a handheld device that does not need direct skin contact; 2) non-polarized dermoscopy which functions by direct skin contact. Both types have light-emitting diodes for illumination and a magnification lens[29]. By dermoscopy, *Dermatobia hominis* appeared as a creamy-white object with a posterior segment in the form of a bird's feet-like structures bounded by a thorn crown[30]. Dermatoscopy also allowed the diagnosis of cutaneous myiasis on top of *Pemphigus vulgaris* lesions where the posterior spiracle was visualized in detail and captured[31].

Association with bacterial furunculosis or skin malignancy is challenging for treatment. In the latter condition, routine wound care may lead to bleeding. In neck lesions, bleeding might involve the carotid artery or one of its subdivisions[32]. Moreover, ectoparasitic drugs like lindane, malathion, and ivermectin have severe adverse effects when used for open wounds. Recently, phytochemicals have been explored for the treatment and prevention of myiasis[33].

Tungiasis

Originally, *T. penetrans* is a blood-feeder ectoparasitic flea that was related only to the American countries. It was transmitted to Africa during the period between the 17th and 19th centuries with the slave trade and later in 1872 with the increased shipping of sand from Brazil to Angola[34]. Tungiasis is prevalent in rural poor areas with sandy soil and banana plantations[35]; for instance, in Ethiopia, Cameroon, Tanzania, Kenya, Nigeria, Rwanda, and Uganda[36-38]. In Brazil, the disease doesn't show seasonal variations due to different rainfall patterns. In Kenya, cases increase when only muddy ponds are present as a source of water[39]. Pigs, dogs, and cats are the reservoir hosts[40-43]. It is predominantly higher in boys than in adults due to less exposure or acquired immunity that occurs with age[36,44]. An outbreak of pruritic boil-like lesions among soldiers of a UN peacekeeping battalion was reported[45].

Notably, *T. penetrans* is the smallest species of fleas (1 mm)[46]. According to the Fortaleza Classification

infestation with *T. penetrans* was divided into five clinical stages[47]. Stage I: (30 min to hours), the lesion is a red spot (1 mm). Stage II: (one to two days later), the skin becomes erythematous, and the parasite increases in size to be more evident (whitish/ pearl-like nodule) with a central black pore (the anogenital opening). Stage III: (up to three weeks) the arthropod increases in size to the maximum forming a round or watch glass-like patch. The lesion is painful simulating a foreign body with hyperkeratosis and desquamation. Stage IV: (three to five weeks) the lesion involutes and becomes covered by a black crust containing dead fleas. Stage V: (six weeks to several months), the lesion develops into a residual scar[47].

Typically, sites are the heels, the soles, and the periungual area of the toes. Also, other parts of the body such as the hands, elbows, neck, buttocks, and genitalia can be infested[48]. In complicated cases, the parasite triggers intense inflammation, suppuration, and ulcer formation that ends in gangrene, and difficulties in walking[49].

Histopathological examination is not indicated for diagnosis in endemic areas. However, in traveler's medicine, it is recommended to settle the diagnosis after return from the endemic areas[50]. Lesions may take the shape of a tumorous growth from which histological sections reveal the presence of pseudo-epitheliomatous hyperplasia[51], infestations with hundreds of fleas, eggs, and chitinous remnants[34,52] that might be associated with *Staphylococcus aureus* and other aerobic and anaerobic bacteria (involving *Clostridia* spp.)[53]. The site of *T. penetrans* larval extraction may be contaminated with *C. tetani*, causing tetanus in non-vaccinated individuals[54].

In endemic areas such as northeast Brazil, tungiasis is not considered a health hazard, even in children with severe disease. In addition, it is not well recognized among physicians. Elimination of the flea requires a sterile needle and topical antibiotic. However, attention against rupture of the parasite during extraction is required to avoid severe inflammation[34]. Niridazole at 30 mg/kg body weight was successful among infected children[55]. Ivermectin (a single oral dose of 0.2 mg/kg BW) and thiabendazole (25 mg/kg body weight daily for 10 d) showed efficacy in several patients[56]. Inhabitants in Fortaleza (Brazil) apply a blend of candle wax, kerosene, and medicinal volatile oils or plant extracts on the lesions[57]. To protect against complications, application of disinfections, close follow-up, and removal of the invading fleas are required[58]. Insecticidal spraying of the infested soil and treatment of infected domestic animals may prevent infestations[58]. Recently, several topical applications have been developed such as dimeticones[59], mixtures of neem seed and coconut oils[60], and tea tree oil-based gel[61].

Scabies

The origin of zoonotic scabies or "pseudo-scabies" is the direct contact with infected dogs, foxes, cattle, and horses. The majority of reports originated from North America and Asia. In a prior study, the most affected age group ranged from 20-40 years and higher incidences were seen in males[62,63].

Scabies is characterized by a short incubation period and a self-limiting course. The establishment of a diagnosis traditionally relies on clinical signs[62]. Zoonotic scabies involves topographic regions such as palms, web of fingers, head, and neck; particularly body areas in contact with animals are foremost affected[64,65]. Clinical signs range from itching to intense pruritic papulovesicular rash[66]. Typically, itching with nocturnal exacerbations start within hours of infection compared with human scabies that needs weeks[67]. Dysbiosis may contribute to the pathology of *Sarcoptes scabiei* triggering the growth of *S. aureus* and the *S. pyogenes* related to group-A Streptococci (GAS)[68].

Adult stages, eggs, and fecal pellets can be identified by microscopic examination. Using dermoscopy at 40x magnification, the 'jet with contrail' sign appears in the form of a small brown triangular structure (anterior part of mite) trailed by a linear segment (a burrow with or without eggs, and fecal pellets). Similar signs with different terminologies for the anterior part of the mite were identified[69] such as: 1) the "delta glider" sign[70]; 2) the "hang-glider" sign[71]; 3) the "circumflex accent like sign"[72]; 4) the "delta-wing" sign[73]. Dermoscopic diagnosis can be enhanced by applying ink to the lesion, revealing a live mite[74]. More clues include: 5) "grey-edged line sign" (pigmented and beaded curved arrow on the outer rim of the tunnel the center of which is filled with bluish-white structures (the feces of the mite that contains melanin); 6) "wake sign" (scales configured in the form of a wake on the water surface formed by a moving body); 7) serpiginous hyper-pigmented thread-like structures (consistent with pigment on histology)[70].

Applying 24 h topical lindane treatment[75], ivermectin[76], and 5% permethrin cream[77] is recommended. Clinical resolution usually occurs within 2-3 weeks post-treatment[78]; yet resolution of pruritic rash after massive exposure to mites is difficult. Symptomatic treatment involves sedative antihistamines and/or soothing cream, to relieve itching, lessen scratching and maintain sleeping[76]. Treating infected animals in contact with humans is essential.

Trombiculiasis

Larvae of the family Trombiculidae (chigger mites) infest skin while the adults exist and reproduce in the soil. Larvae emerge from eggs as obligatory parasites of warm-blooded hosts, involving mainly rodents and

dogs; however, humans can be accidental hosts[79]. *Leptotrombidium delicense* was described as an "endangered species"[80].

A study reported pinnal lesions and deformities in infected patients. In addition, alopecia, inflammation, necrosis, ulceration, scarring, and diminution of pinna mass, may occur. Up to 25–100% of affected pinnae were reported. Pathological examination shows hyperkeratosis and acanthosis with diffuse neutrophilic and granulocyte infiltrations. The superficial epidermis reveals the penetrating mouth part of the mite with focal necrosis whereas the dermis shows inflammatory infiltrates composed of necrotic granulocytes and multifocal hemorrhages[80].

The resulting cutaneous lesions are non-specific in the form of scaly plaques, urticarial, or mild erythema. By video dermoscopy, the larva appears as a bright red-orange oval body with three long pairs of legs strongly attached to the skin[79,81]. Dermoscopy also showed the presence of perifollicular, brown lesions with a central hypo-pigmented area giving a 'sunburst appearance'[82].

Oral rupatadine (10 mg) and topical fusidic acid 0.03 % /triamcinolone bentonite with 2 % cream were recommended as a daily treatment. As prophylaxis, clothes should be washed at a minimum temperature of 60°C to kill the parasitic larvae[83].

Ticks

A wide range of genera was determined involving *Rhipicephalus, Amblyomma, Haemaphysalis,* and *Hyalomma* across numerous countries. South Africa, Tanzania, Zambia, Zimbabwe, Madagascar, Angola, Mozambique, and Comoros are chiefly endemic regions. The highest estimates are found in cattle followed by sheep and goats[84]. In Canada, *Dermacentor* followed by *Amblyomma* and *Ixodes* are the most prevalent genuses[85]. Increasingly, erythema and rash due to rickettsiosis in the Slovak population and Lyme disease in the United States have been reported[86]. Ticks burrow into the soil to protect themselves from high temperatures and rainfall; thus, can endure and persist in the environment[87].

Ticks are blood-feeder ectoparasites and thus can transmit a diversity of viral and bacterial infections to their hosts. Pruritus due to tick infestations and the associated cutaneous manifestations due to co-transmitted diseases are of clinical significance. For instance, Lyme disease is characterized by secondary disseminated erythema migrans[88], and purpura fulminans that is depicted by extensive bleeding lesions and dermal vascular necrosis[89], and Rocky Mountain spotted fever and Japanese spotted fevers which are additionally manifested by skin rash[90].

Prompt diagnosis and treatment by early removal (within 48 h) can incumber transmission of tick-borne-diseases[91]. Dermoscopy is efficient in the identification of various stages of ticks involving adults, nymphs, and larvae. Besides, it can evaluate absolute tick removal. Ticks are characterized by their grey oval bodies and their eight legs. The mouth parts are implanted inside the skin and surrounded by a pink/red halo[92,93]. di Meo *et al*.[94] also recognized the presence of tense brown globular granules that seemed to be tick droppings, while Suh *et al*. identified fragments of the mouth parts of the arthropod inside the skin due to incomplete removal[95]. Visualization of many ticks at a site has also been reported[96]. Dervis *et al*. recommended dermoscopy to differentiate tick bites from other skin lesions such as haemangioma, nevus, and wood splinter[97].

Mechanical techniques involving rotation with forceps and chemical methods through applying gasoline and petroleum jelly or heat on nymphs/female ticks were all reported[98]. Remnants of mouthparts in the skin may lead to the formation of foreign body granuloma[99], and protection against associated bacterial and viral infections is essential. For instance, Lyme disease is co-treated with Doxycycline and Minocycline[100].

Zoonotic protozoa

***Leishmania* species**

It is a blood protozoan transmitted by sandflies (genus *Phlebotomus* in the Old World and genus *Lutzomyia* in the New World). According to the WHO, cutaneous leishmaniasis is an uncontrolled, emerging, and neglected parasitic disease that affects millions every year[101]. Several environmental and human behavioral factors changed the distribution of leishmaniasis[102]. The co-evolution of the parasite included a wider range of reservoir hosts such as the old-world rodents[103], cats[104], dogs[105], and different species of the sand-fly vector[106]. Children in endemic regions are more prone to infection[107]. Although neutrophils are the leading cells enrolled after infection, monocytes share in the pathogenesis of the disease by acting as a reservoir cell that pools the parasite for cell-to-cell transmission[108]. Clinical types of zoonotic leishmaniasis involve ulcerative leishmaniasis, lymphangitis, lymphadenitis, and tuberculous forms. The final course of the disease is formulated according to the Th1-Th2 balance[109] (Fig. 1).

Skin lesions occur predominantly on the arms and legs, whereas the head is the least common area. When the lesion is present on the skin of the feet purulent discharge in addition to lymphangitis, and lymphadenitis are present. Facial lesions develop rapidly and are characterized by their uneven edges and deeper wounds. Notably, cutaneous leishmaniasis appears to be sporadic in HIV patients; where Kaposi sarcoma shows manifestations similar to cutaneous leishmaniasis[110].

Clinical features are unspecific. Hence, pathological, parasitological, molecular and serology assays are routinely useful to establish diagnosis. Molecular PCR techniques are the most sensitive, but they are limited

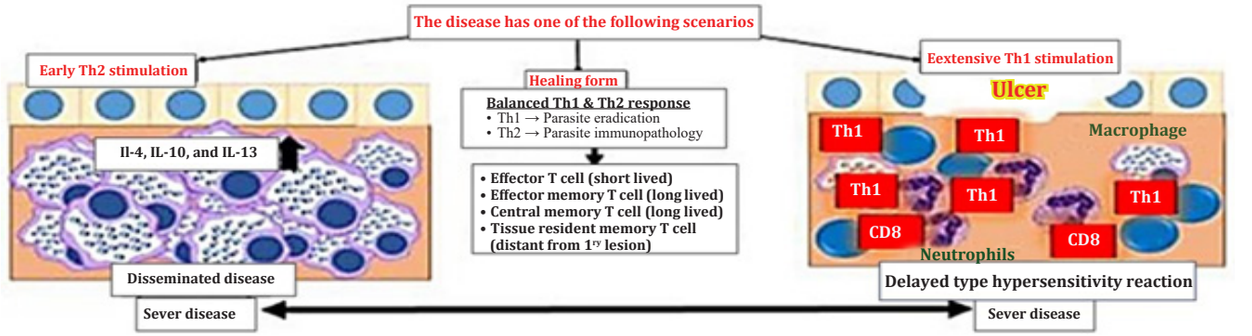

Fig. 1. Clinical types of cutaneous leishmaniasis according to the immunological background (Elsaftawy *et al.*)[109].

to research centers. Hence, serology remains the key tool for diagnosis including immunochromatography test (ICT), enzyme-linked immunosorbent assay (ELISA), immunofluorescence antibody test (IFAT), western blot, and direct agglutination test[111,112]. Monoclonal antibodies are also used for the identification of New World Leishmania species[113].

Current anti-leishmanial therapies include antimonials, pentamidine, miltefosine, amphotericin B, and paromomycin. El Saftawy *et al.*[114] underlined the presence of several factors that hamper the healing of lesions such as virulence factors of the parasite; few of which are targeted by current drugs. Moreover, drug efficacy is declining in immune-suppressed subjects, due to generation of side effects, and occurrence of drug resistance. Other issues are the parasitic burden and interactions of immune responses that rely to a great extent on the species of the parasite. Thus, raised arguments for new therapeutic modalities involve laser therapy and novel drug delivery systems such as metallocomplexes. Nd:YAG and CO_2 lasers are reported to enhance healing speed; however, successful laser therapies rely on the collaboration of several factors including host, parasite and laser issues[114,115].

Toxoplasma gondii

This ubiquitous tissue protozoon is of cosmopolitan prevalence as detected by specific anti-*Toxoplasma* IgG antibodies (1-100%). Occurrence relies on socioeconomic and hygiene levels, soil humidity, and environmental contamination with oocysts[116]. Toxoplasmosis increases with age and in warmer and humid climates[117] and is more prevalent in males than in females[118]. In the Far East, some countries have the lowest seroprevalence (~1%) whereas, in some European and South American regions, the disease has the highest seroprevalence (>90%). A Brazilian study highlighted the serious high risks for the acquirement of infection during pregnancy and hence fetal transmission[116]. According to the Centers for Disease Control and Prevention (CDC), seroprevalence in the USA ranges from 11% to 22.5%[119]. Immunosuppression and transplantation of hematopoietic stem cells particularly in children show a higher rate of transmission[120]. Elsaftawy *et al.*[109] inquired if the epidemic of COVID-19 was associated with hidden increases in *T. gondii* transmission as both pathogens are zoonotic.

Cutaneous toxoplasmosis was recorded in felines[121]. Blind administration of steroids in pets deteriorates their immunological condition and leads to the multi-organ spread of the parasites[122]. In humans, cutaneous toxoplasmosis is chiefly asymptomatic; yet, in acquired toxoplasmosis, lymphadenopathy might be present in association with papular rash, scattered prominent erythematous macules (3–4 mm), hard painful nodular lesions, or ulcers[123,124]. Lesions appear on the back, abdomen, shoulders, palms, and soles[125,126]. One study reported a case of toxoplasmosis in a febrile patient with skin manifestations[127]. Moreover, Amir *et al.*[128] demonstrated cutaneous lesions as a part of systemic toxoplasmosis. However, neural, and respiratory involvement obscures a definite diagnosis[129]. Cutaneous toxoplasmosis may mimic drug reaction or a graft-versus-host disease in patients with a history of recent transplantations[130].

In an infected feline, a skin nodule showed free and clustered parasites, necrotizing granulomatous panniculitis, and vasculitis with intracellular parasites in endothelium cells, fibroblasts, and macrophages[131]. In a cutaneous complication of immunosuppression state, diagnosis of toxoplasmosis was difficult due to the morphologic similarity to *Leishmania* and *Histoplasma* spp.[128]. Molecular identification, electron microscopy, and immunohistochemistry are valuable to identify the parasite[132]. Notably, serologic methods are not useful in severely immunosuppressed patients[122].

In a report, Yang *et al.*[126] recommended early diagnosis and treatment of this fatal opportunistic parasite in order to improve prognosis. In another report, sulphadiazine and pyrimethamine failed to resolve the skin lesions and instead, the patient developed new lesions[125]. In addition, sulfadiazine hypersensitivity, immunosuppression, and relapses were all reported[133].

Zoonotic helminths

Strongyloides stercoralis

This exceptional nematode can distinctively molt within the host, convert into an infective larva stage, and cause internal autoinfection. Strongyloidiasis exists as a latent infection in immunocompetent individuals, while in immunocompromised patients, it is a fatal disease with hyper infection of disseminated forms[134]. In a 2013 report, 30-100 million subjects were allegedly infected with *S. stercoralis* worldwide[135]. *S. stercoralis* involves two genetic populations, one that infects dogs and humans (zoonotic) and another one that entirely infects dogs[136,137]. Zoonotic infection with *S. fuelleborni* has been recently reported[138]. Parasite mapping is related to Africa and South-East Asia[139], Columbia[140], and the Abua area of Rivers State in Nigeria[141]. However, recent studies revealed its existence in Australia, Thailand, France, Belgium, Bulgaria, and Switzerland showing the devastating spread of the parasite[142,143].

Primary strongyloidiasis occurs through penetration of skin by infective larvae found in contaminated soil. Then the larvae pass to the lungs through the bloodstream. In the alveoli, they emerge to pass to the trachea and the upper gastrointestinal tract. Once in the intestines, larvae molt into adult females and reproduce asexually *via* parthenogenesis producing eggs. Thereafter, hatched larvae pass in the stool with or without eggs[144].

In endemic areas, chronic strongyloidiasis shows several skin manifestations. The most typical is larva currens which is a serpiginous, oedematous, and creeping urticarial eruption. In hyperinfection syndrome, these manifestations are accelerated[145]. In disseminated strongyloidiasis, the distinctive skin lesions are extensive petechiae and purpura[146]. In severe conditions skin lesions might be associated with neurologic involvement[147].

Serpiginous dermatological lesions with unexplained eosinophilia and pulmonary and gastrointestinal manifestations are characteristic[148]. Stool examination can be performed by three methods: (i) the sedimentation-zinc chloride flotation (ii) the Baermann technique, and (iii) the sodium acetate acetic acid formalin concentration method[138]. Nevertheless, stool examination has low sensitivity. Greaves *et al*. [139] recommended serology for the diagnosis and follow-up of the disease.

Dermoscopy introduced in the diagnosis of disseminated strongyloidiasis[149] revealed homogenous purpuric areas corresponding to leukocytoclastic vasculitis[150]. Fluorescence-advanced video dermatoscopy also has recently been introduced to differentiate strongyloidiasis. It manipulates a monochromatic light-emitting source to inspect the skin and relies on the capacity of the endogenous particles to attain certain wavelengths and release fluorescence. The images are captured in real-time by means of greyscale whereas black color means no fluorescence and white shows the highest fluorescence[151].

Thiabendazole is the drug of choice for strongyloidiasis; however, the undesirable adverse effects limit its use. Therefore, albendazole and ivermectin are frequently prescribed[139]. Challenges in treating disseminated and hyper infections are the associated immunosuppressed status, sepsis, progressive myopathy, acute respiratory failures, and physical decline. Basile *et al*.[152] showed that histologic findings of skin biopsies from these patients exposed many interstitial and intravascular filariform larvae. Krishnamurthy *et al*.[153] stressed on the importance of cautious resolve before starting immune-suppressive remedies in patients from endemic areas.

Cutaneous larva migrans (CLM)

Animal hookworm is a nematode belonging to the Ancylostomatidae where the infection is caused by the larvae stage. Infection occurs mainly in poor societies in tropical and subtropical regions. Larval stages present in soil contaminated with feces from cats and dogs invade the skin of bare feet[154]. Climate changes increased incidences of CLM cases, particularly in non-tropical areas[155]. Larvae cannot invade the basal layer of human skin and remain in the epidermis without completing their lifecycle, accordingly untreated skin lesions are self-limiting within months[156]. Atypical presentations in the form of secondary infection and bullous formation may occur[157].

On dermoscopy, larvae are distinguished as segmental lustrous brownish structures in yellow to red serpiginous tracts, or yellowish to brownish spots and dotted vessels[149]. Ivermectin and Clobetasol cream are recommended. The challenge to controlling infection is that it has never been considered a health hazard in poor communities[158].

Gnathostomiasis

This is a systemic nematode infection caused by *Gnathostoma* spp. through eating raw fish. In humans, infections are accidental, and the ingested parasite is only a larva or immature adult[159]. In carnivorous mammals, the parasite reaches the adult stage (definitive hosts). *G. spinigerum* is encountered in the majority of human infections. Other zoonotic species include *G. nipponicum, G. hispidum, G. doloresi*, and *G. binucleatum*. Endemic countries involved are Thailand, Japan, and Vietnam, in Southeast Asia, and Ecuador, Peru, and Mexico in the American content. In the United States, cases are mostly recorded in immigrants from Southeast Asia. In tropical Australia, infections were diagnosed using serology. Cases have been recently reported in Brazil[160], Colombia[161], and Korea[162].

The worm is characterized by a prominent cephalic bulb and a body covered with spines. In humans, the advanced L3 larvae or immature adults invade the stomach to reach the liver, skeletal muscles, and subcutaneous tissues. Cutaneous presentations involve four forms of creeping eruption, nodular migratory panniculitis, pseudo-folliculitis, and a mixed form[159]. During the initial migration of larvae, eosinophilia is commonly detected and then it subsides in chronic cases. These swellings are painful, erythematous, and itchy and may reappear over years if the patient was not treated. In case the parasite migrates near the skin surface it can emerge spontaneously or be easily extracted[163]. Seriously, the condition might be associated with visceral gnathostomiasis (larva migrans-profundus), neuro-gnathostomiasis with fatal eosinophilic meningitis, and myeloencephalitis[164], or ocular gnathostomiasis[165]. Gnathostomiasis differs from CLM, especially when lesions are present in uncommon areas for CLM e.g., breast or other covered parts of the body)[163].

Úraga *et al*.[159] reported dermoscopic findings involving the presence of cylindrical brown structures and pink halos consistent with the gut and exoskeleton of the larvae. Skin manifestations are related to the infiltration of eosinophils. The larva is usually present in the dermis and is definitely discriminative from the larvae of CLM (smaller larva and tunnel at the epidermis), and from *S. stercoralis* larva (may migrate in the deep dermis and is relatively smaller than *Gnathostoma*)[163].

Histologically, *Gnathostoma* spp. is often differentiated on the basis of the specific morphology of the epithelial cells of the parasite's gut. Serological detection involving ELISA for IgG antibodies against L3 has been introduced[166]; however, it is characterized by low sensitivity and cross-reactivity with other nematodes[167]. A western blot assay against crude preparations of *G. spinigerum* has been industrialized for research in European and Asian countries. Since the early 2000s, sequencing of ribosomal DNA was performed for the molecular analysis of gnathostomiasis[168,169]. Notably, neuroimaging findings cannot be considered diagnostic but may reflect CNS involvement. However, MRI is better than a CT scan in distinguishing *Gnathostoma* from *Strongyloides* larvae[167].

For treatment, albendazole is usually prescribed[170]; however, low cure rates and recurrences occur frequently. Ivermectin is an alternative choice and repetition might be required after 7 d[171]. Recurrences may occur for up to two years with the initial therapy or, with the combination of albendazole and ivermectin. Thus, treatment may necessitate multiple courses of albendazole, up to 4 courses, and a prolonged period of follow-up. Sometimes, local relapses are in the form of transitory inflammation or itching without larval migration; hence, conservative anti-histaminic therapy is required. New migratory lesions refer to the persistence of living parasites which would require the initiation of a new course of treatment. Challenges also involve cerebral edema; hence oral steroids are advisable[172].

Dirofilariasis

A member of the Nochtiella subgenus, *D. immitis* is known as the canine heartworm and is mainly transmitted *via* mosquitos. Dirofilariasis is endemic in the European continent and Asia mainly in the southern eastern zone. In addition, it has been frequently reported in Africa. The increased veterinary awareness reduced the prevalence of *D. immitis* prevalence in dogs. Yet, climate changes have been considered as the main spreading factor that predisposed dirofilariasis via new competent and invading species of mosquitos e.g., *Aedes albopictus* and *Ae. koreicus*. Moreover, the prevalence of asymptomatic dogs particularly in the recently colonized areas resulted in new spreads[173].

Dirofilariasis in humans occurs accidentally and is a dead end. Pathogenesis mainly involves migration of the larvae and the host's immune response. The endosymbiont *Wolbachia* spp. bacteria complicate immunopathogenesis through shifting Th2 pathway to the Th1[174]. The parasite however was speculated to exert immune evasion *via* cysteine protease inhibitors (also, cystatins) that modulate host immune reaction by triggering IL-10 production whilst IL-12 and TNF-α are reduced[175].

Human cutaneous dirofilariasis is recognized as a migrating subcutaneous tumor[176]. Gravely, surgeons frequently misdiagnose dirofilariasis for malignancy and distress the patients with mismanagement. Therefore, acquaintance with the typical location of the parasite in the face around the eyes and its migratory nature, recognition of the endemic regions of the nematode, and the proper recording of travel history are all necessary to establish a correct diagnose. High-resolution ultrasonography enhances visualization of the motile nematode in the subcutaneous nodules[177]. *Dirofilaria* spp. are distinct from other nematodes through its characteristic morphology under light and scanning electron microscopy. Histological assessment of the subcutaneous nodule following its excision confirms diagnosis. DNA-based analyses is also beneficial[178].

Surgical excision of the subcutaneous nodule or the extraction of the helminth is the best approach in the management of dirofilariasis. Ivermectin and other anti-helminthic treatments are of unclear outcomes; yet, they may hamper migration of the nematode[178,179]. Doxycycline proved efficacy to eliminate circulating *Wolbachia* in blood[174].

Schistosome dermatitis/swimmers' itch

Cercarial dermatitis (swimmers'/ bather's itch) is a trematode infection caused by non-human *Schistosoma* species. It occurs in subjects washing or swimming in ponds inhabited by snails infected with the miracidia of birds' schistosomes[180]. In Europe, it is mostly recorded in lakes largely occupied with aquatic vegetation[181]. Also, expanding riverbeds and dams created good circumstances for this zoonotic parasitosis to emerge[182]. With a similar ecosystem, in the southwest of the United States, dermatitis-causing schistosomes were increasingly reported suggesting the probability of dermatitis outbreaks[183].

The lesion is present in the form of dermatological allergic, tender, and painful erythematous rash (1-2 mm)[184]. The most severe symptoms that occur in childhood compared to adults, may be in the form of bronchial reactions[185]. The differences in the course of the disease seemed to rely on the state of the immune system[184].

Histopathology reveals the presence of cercariae within the epidermis close to the stratum granulosum mixed with eosinophils and minor perivascular lymphocytic infiltrates in the dermal layer[186]. The investigators speculated the daily use of Triamcinolone cream with hydroxyzine. Follow-up is important in order to confirm the complete disappearance of symptoms and resolve of eruptions[186].

CONCLUDING REMARKS

1. Despite the widespread zoonotic cutaneous parasitosis in tropical and sub-tropical zones, several reports were conducted from developed countries with suitable environments.
2. Extension in epidemiology was seen in scabies, toxoplasmosis, Gnathostomiasis, and *Schistosoma* dermatitis.
3. The real burden of zoonotic parasitosis is not well recognized due to lack of studies in this field.
4. Nonspecific symptomatology may obscure the correct diagnosis.
5. Recently, dermoscopy is an expectant alternative in the diagnosis of cutaneous parasitosis caused by arthropods and helminths. The procedure allows visualization of the detailed features of the parasite that invaded the skin; thus, confirming a diagnosis.
6. In zoonotic protozoa, histopathological examination remains the basis for proper diagnosis. Challenges in medical treatment were recognized with several parasites; therefore, the exploration of alternatives is necessary.

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