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# Travelers' tropical skin diseases: Challenges and interventions

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## Abstract

Tropical regions receive a significant part of the traveling population. It is very important that health professionals are familiar with the main tropical skin diseases and able to advice patients appropriately. This article reviews the main tropical diseases of travelers, with an emphasis on diagnosis, management, and prevention. Among others, cutaneous larva migrans, myiasis, tungiasis, Chagas disease, Dengue fever, African trypanosomiasis, filariasis, and leishmaniasis are discussed. Increasing awareness among travelers and health care professionals can help reduce morbidity and mortality. Continued research on new drugs and vaccines is needed to reduce the risks of tropical diseases.

**KEYWORDS**

cutaneous larva migrans, diseases of travelers, leishmaniasis, myiasis, tropical diseases

## 1 | INTRODUCTION

In 2015, it is estimated that 1.2 billion people traveled around the world, and the number of international tourist arrivals is expected to increase by 3.3% a year by 2030 (United Nations World Tourism Organization, 2015). Along with the increasing number of travelers, a significant growth of travel-related illnesses including dermatologic problems is observed (Korzenieweski, Juszczak, & Jerzemowski, 2015). In tropical regions, travelers are exposed to a much larger number of insects (vectors of arthropod-borne diseases) than in temperate climate areas as well as contaminated soil and water (Korzenieweski et al., 2015). Trips to exotic destinations and tropical regions expose the travelers to risks of contracting new illness; therefore, preventive measures are required to reduce these risks.

A research study that was conducted by Freedman et al. included 17,353 travelers and concluded that skin disorders are the third most recurrent health problem that international travelers face when traveling to developing countries, only rating behind fever and acute diarrhea (Freedman, Chen, & Kozarsky, 2016). Skin diseases may affect up to 8% of travelers (Patel & Sethi, 2009). Preventive measures can be provided through travel protocols. People who plan to travel to other countries are advised to ask their health care providers for information about preventive interventions that can be obtained by following protocols such as those reported in Freedman's review. Dermatologists and general practitioners should be prepared to prevent, diagnose and treat tropical dermatological diseases in travelers, and this review aims to assist in this task. Resources and relevant sites on tropical diseases are listed on Table 1.

**Abbreviations:** CLM, cutaneous larva migrans; FDA, Food and Drug Administration; WHO, World Health Organization; YFV, yellow fever

## 2 | VIRAL EXANTHEMS

In a study by Hochedez et al. (2008), Chikungunya (35%) and Dengue (26%) were the major of etiologies of febrile exanthem.

### 2.1 | Dengue fever

Dengue fever is caused by a flavivirus that has five serotypes. It is transmitted by the *Aedes* mosquitos, *particularly A. aegypti*, an insect with an incubation period that varies from 4 to 10 days. It is an emergency disease and according to World Health Organization (WHO), over half of the world's population is at risk of contracting the virus that infects approximately 100 million people per year (Jelinek, 2000; Ligon, 2005; Schwartz, Mendelson, & Sidi, 1996; Tauil, 2002).

### 2.2 | Diagnosis

The diagnosis is made through the clinical and laboratory features, which must include fever and two of the following: severe headache, retro-ocular pain, gastrointestinal distress, myalgia, arthralgia, prostration, skin rash, and leukopenia. Over 50% of patients develop facial erythema, a macular, rubella-like exanthem, and petechiae. Dengue fever presents with one or more of the following manifestations: severe abdominal pain, persistent vomiting, fluid

  

**TABLE 1** Resources and relevant sites on tropical disease

| Resource | Site |
|---|---|
| WHO Emerging Diseases | http://www.who.int/topics/emerging_diseases/en/ |
| Morbidity and Mortality Weekly Report | http://www.cdc.gov/mmwr/ |
| Weekly Epidemiological Record | http://www.who.int/wer/en/ |
| Journal of Emerging Infectious Diseases | http://www.cdc.gov/ncidod/EID/ |
| ProMED-mail | http://www.promedmail.org |
| HealthMap | http://www.healthmap.org/ |
| The International Society of Travel Medicine | http://www.istm.org/ |
| CDC Travelers' Health | http://www.cdc.gov/travel/ |
| International Association for Medical Assistance to travelers | http://www.iamat.org/ |
| Secretariat of Health Surveillance, Brazil | http://portal.saude.gov.br/ |
| Bulletin Épidémiologique Hebdomadaire, France | http://www.invs.sante.fr/beh/index.html |
| Directorate General of Health, Portugal | http://www.dgs.pt/ |
| Eurosurveillance | http://www.eurosurveillance.org |
| TropNetEurop | http://www.tropnet.net/index_2.html |
| TravelMED, Germany | http://www.travelmed.de |
| Statens Serum Institut EPI-NEWS, Denmark | http://www.ssi.dk/sw1928.asp |
| Global Public Health Intelligence Network, Canada | http://www.phac-aspc.gc.ca/index-eng.php |
| British Travel Health Association | http://www.btha.org/ |
| Travel Advisories, Australia | http://smartraveller.gov.au |

accumulation (ascites, pleural, and pericardial effusion), mucosal bleeding, lethargy, postural hypotension, hematuria, petechiae, and hepatomegaly.

Specific laboratory tests include viral isolation and detection of NS1 antigen. Viral isolation (gold standard) is performed in a blood sample taken up to the fifth day of symptoms when viremia occurs. Detection of the NS1 antigen (rapid test) through the ELISA technique that must be collected within the first 3 days of symptoms and the specific antibodies (serology), performed by means of the IgM dosage should be positive after the fifth day of disease.

### 2.3 | Treatment

The treatment is symptomatic and includes rehydration, paracetamol, antiemetics, and antipruritics. The use of acetylsalicylic acid, non-hormonal anti-inflammatories, and drugs with hemorrhagic potential is contraindicated.

### 2.4 | Prophylaxis

Insecticides, barrier measures, protective clothing, bed netting, and insect repellents, as well as immunization, that has a reported efficacy rate of 56.5% (Capeding et al., 2014).

### 2.5 | Vaccine

The dengue vaccine (Dengvaxia®) protects against serotypes 1, 2, 3, and 4 (recombinant and attenuated), and is being developed by Sanofi Pasteur (Aguiar, Stollenwerk, & Halstead, 2016).

### 2.6 | Chikungunya

Chikungunya is an RNA togavirus that has five genotypes. The virus was first detected in Tanzania in 1952. It is transmitted by *A. aegypti* mosquito which can also transmit dengue and yellow fever. The incubation period ranges from 3 to 7 days (Burt, Rolph, Rulli, Mahalingam, & Heise, 2012; Powers & Logue, 2007). Fetal infection can result in microcephaly.

### 2.7 | Symptoms

The eruption of chickungunia is present in half of patients. It affects the torso, and face, and most commonly the limbs. Transient maculopapular eruptions lasting 2–3 days may be associated with pruritus. These authors have observed many cases with atypical cutaneous features mimicking Stevens–Johnson syndrome and toxic epidermal necrolysis, especially in infants and children (Garg, Sanke, Ahmed, Chander, & Basu, 2018). Polyarthralgia is a prominent feature, with symmetrical involvement of joints of midtarsal region, foot, ankles, knees, small joints of hands, wrist, and elbow. There is swelling of joints but no other signs of inflammation (Chaterjee, Sarma, & Hansda, 2017).

### 2.8 | Treatment

There is no vaccine or specific drug against the virus, and the main difference between Dengue and Chikungunya is that the fever and joint pain are more intense in the second. Symptomatic treatment can control the pain and fever.

### 2.9 | Prevention

Exposed skin should be covered with long-sleeved shirts, trousers, and hats. The use of insect repellent as indicated. Travelers should sleep in places that are protected from mosquito nets (Gregianini et al., 2017).

## 3 | CUTANEOUS LARVA MIGRANS

According to Caumes' research with 224 patients, *cutaneous larva migrans* (CLM) is the most frequent dermatosis observed in travelers, followed by bacterial skin infections, arthrop reaction, myiasis, tungiasis, urticaria, febrile rash, leishmaniasis, and scabies (Caumes et al., 1995). With conditions in tropical or subtropical climates favoring the infection, CLM is classically noted in the returning traveler. Over the last decades, reports of CLM in Europe have become more common, probably because of more frequent trips to endemic areas. The most striking fact is the rise of autochthonous cases in southern Europe (Gutiérrez García-Rodrigo, Tous Romero, & Zarco Olivo, 2017).

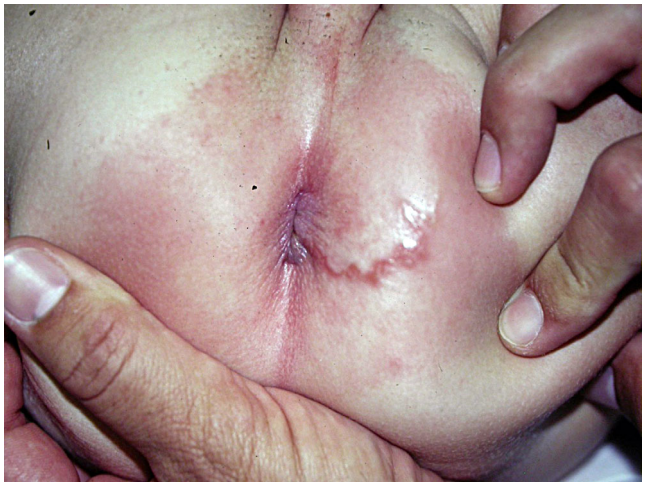

**FIGURE 1** Cutaneous larva migrans: Typical serpiginous erythematous tract developed after infant's contact with sand

CLM is usually the result of skin infection by the larval stages of dog and cat hookworms (in most cases *Ancylostoma caninum* and *braziliense*) but can also be caused by other helminth parasites such as *Strongyloides stercoralis* (Karthikeyan & Thappa, 2002). Clinical examination is fundamental to diagnosis and reveals a typical serpiginous erythematous tract (Figure 1) known as a "creeping eruption" that represents the path of a single larva through the epidermis (Baple & Clayton, 2015). Humans are incidentally infected when there is skin contact with contaminated soil or sand containing filariform larvae. These develop 5–10 days after the rhabtidiform larvae have hatched from the eggs passed in the feces of their infected definitive host (Baple & Clayton, 2015). Once the host is contaminated, the parasite is confined to the epidermis and dermis. It usually undergoes spontaneous resolution within 1–6 months but because of the risk of potential complications, intense pruritus and substantial disease duration, treatment becomes necessary (Baple & Clayton, 2015; Karthikeyan & Thappa, 2002).

### 3.1 | Diagnosis

Since larvae in the skin are rarely recovered, and they are not readily or easily identified when found, the species involved in individual cases is usually unknown. Thus, the diagnosis of CLM is based mainly on the clinical features and typical history.

### 3.2 | Treatment

Although CLM is self-limited, treatment is desired by most patients. Treatment options include topical 10–15% thiabendazole solution three times daily for 15 days, oral thiabendazole 50 mg/kg/day (not exceed 3 g/day) during 3–4 consecutive days, albendazole 400 mg/day for 3–5 days, and ivermectin 150–200 mg/kg as a single dose (Karthikeyan & Thappa, 2002).

### 3.3 | Prevention

Travelers should avoid beaches with dogs and cats, wear sandals at beaches, use towels or deckchairs, walk at the waterline.

## 4 | MYIASIS

Myiasis is defined as the infestation by fly larvae or maggots whose hosts are human and vertebrate animals. Skin involvement, the most common manifestation of myiasis, occurs in three forms: furuncular myiasis, wound myiasis, and migratory myiasis. In furuncular myiasis, the female fly lays eggs on a blood-sucking insect with a quick-drying glue-like substance. Wound myiasis follows fly infestation of open wounds, mucous membranes, and body cavities. Humans are an accidental host in migratory myiasis and acquire the eggs by contact with the horse's coat or by accidental deposition of eggs by the fly onto human skin (Francesconi & Lupi, 2012; Vasievich, Villarreal, & Tomecki, 2016).

Furuncular myiasis typically exhibits one or more erythematous papules or nodules, ranging in size from 0.2 to 2 cm with a central pore or punctum (Figure 2), which is the caudal spiracle of the larva through which it breathes and expels waste—a spontaneous serosanguinous drainage from the site. Sites of predilection are the head and neck, upper shoulders, and chest. Symptoms may include itching, nocturnal lancinating pain at the sites, a sense of movement from within the nodule, fevers, and chills. The disease may also be associated with local adenopathy.

### 4.1 | Diagnosis

Diagnosis is primarily clinical and often strengthened by the travel history (McGraw & Turiansky, 2008). Dermoscopy is a useful diagnostic tool and can be performed after a few hours of occlusion, which forces the larva to make surface (Blaizot et al., 2018). Typical aspects include: breathing spiracles looking like *bird's feet*; a creamy body surrounded by black dots shaped as a *thorn crown*; and a *bubbling* as the larva breathes. If imaging is necessary, multichanneled color Doppler ultrasound with variable and high-frequency probes (upper range 15–22 MHz) and magnetic resonance imaging (MRI) may be used. Ultrasound using lower frequency probes (≤10 MHz) has been used in the identification of *Dermatobia hominis* larvae in soft tissues (Bouer et al., 2016).

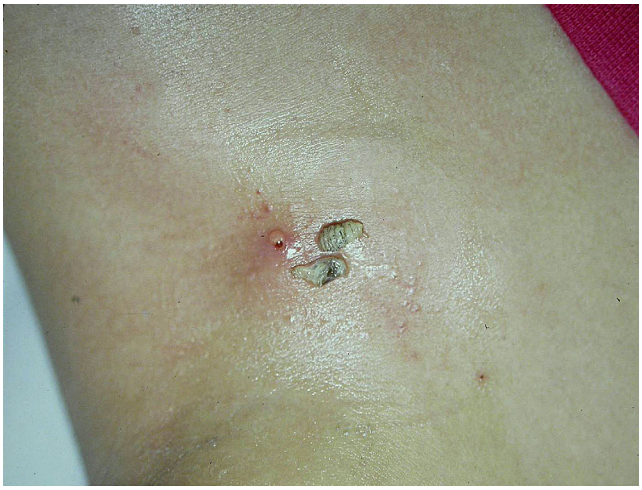

**FIGURE 2** Furuncular myiasis: Erythematous nodule with a central punctum

### 4.2 | Treatment

Treatment includes application of toxic substances to the eggs and larvae, methods producing localized hypoxia to force emergence of the larvae, and mechanical or surgical debridement (Pascoal et al., 2016). The idea of sheltering a larva in the skin is often unbearable and justifies trying a first-hand manual extraction with local anesthesia. Lidocaine or liquid nitrogen can be used both to paralyze the larva and anesthetize the lesion. Secondly, a surgical excision with a scalpel or a punch biopsy might be necessary to remove the whole larva (Blaizot et al., 2018). Adjunctive ivermectin treatment can be considered when extraction may have been incomplete (Blaizot et al., 2018).

Cutaneous lesions usually heal rapidly after the larvae are removed. Bacterial superinfection has been described and is usually secondary to likely scratching. In such cases, a brief course of antibiotic therapy may be indicated. In general, cutaneous infestation, other than in its local effects, is harmless (Safdar, Young, & Andes, 2003).

### 4.3 | Prevention

Because *D. hominis* is a forest-dwelling fly found in Central and South America, individuals who participate in ecotourism and rural outdoor occupations such as archeology and mining in these areas are at risk of developing myiasis. This disease can be prevented through using protection measures to reduce mosquito bites (Boggild, Keystone, & Kain, 2002).

## 5 | TUNGIASIS

Tungiasis, caused by the sand flea *Tunga penetrans*, is an infestation where the female sand flea burrows into the host after mating, takes a blood meal from the superficial dermal blood vessels, and proceeds to extrude more than 100 eggs, which subsequently fall to the ground (Vasievich et al., 2016). While in the host, the flea burrows into the outer layer of the skin and grows considerably, remains in the host for 4–6 weeks, and subsequently dies and is sloughed off by the host over several weeks (Korzenieweski et al., 2015; Vasievich et al., 2016). The fleas inhabit dry and sandy places in rural areas, such as stables and pens, affecting humans and pigs. The infestation is found worldwide, including sub-Saharan Africa, India, and especially in the Caribbean (prevalence 15–50%) (Vasievich et al., 2016).

### 5.1 | Diagnosis

The sand flea favors acral sites, such as toes and subungual skin, and is often acquired when walking barefoot or with open-toed shoes in an endemic area. The disease presents with a papule or nodule, often with an overlying black dot where the flea has entered the host (Vasievich et al., 2016).

### 5.2 | Treatment

A shave or punch excision or sterile needle is usually sufficient in removing the flea (Vasievich et al., 2016). In patients with severe disease, topical ivermectin, metrifonate, or thiabendazole may also be used (Heukelbach, Eisele, Jackson, & Feldmeier, 2003).

## 6 | CHAGAS DISEASE

Chagas disease, also known as American trypanosomiasis, is caused by protozoon *Trypanosoma cruzi* and transmitted by arthropod vectors of the family *Reduviidae* (also known as "kissing bugs") such as the *Triatoma* insect (known as "vinchuca" in Spanish or "barbeiro" in Portuguese) which bites humans most commonly on the face at night (Patel & Sethi, 2009). *Triatoma* sheds feces containing *T. cruzi* protozoa at the site of the bite which are rubbed or crushed into the bite wound to alleviate itching. The parasite then enters the bloodstream and affects internal organs, typically the heart and intestines. Chagas disease affects between 7 and 8 million people and is a neglected tropical disease. It is endemic in some regions of Latin America (Costa et al., 2003).

There are two ways of transmission: vector, that is, the "kissing bug" insect bites humans and lays feces that, when in contact with wounds or mucous membranes, transmit the parasite, and oral transmission, that is, the bug deposits feces on (or is crushed with) food, such as açaí and sugar cane juice (Da Silva Valente, de Costa Valente, & Neto, 1999). Outbreaks of orally acquired acute Chagas disease have been reported in Belém, Brazil and Caracas, Venezuela (Alarcón de Noya et al., 2010).

### 6.1 | Diagnosis

Chagoma is a red, indurated furuncular lesion at the site of inoculation that is accompanied by regional adenopathy and central edema (Patel & Sethi, 2009). It develops in the weeks after the initial bite and persists for weeks afterward. *Romaña's* sign is a classic sign of acute Chagas disease. It occurs due to deposition of parasite—laying feces into the conjunctival sac. Symptoms of acute Chagas disease include fever for more than 7 days, headache, intense weakness, swelling of the face and legs, vomiting, and diarrhea. The acute phase of Chagas may last up to 2 months, after which patients enter the indeterminate, latent phase (Patel & Sethi, 2009). One third of these patients will develop chronic Chagas disease.

### 6.2 | Treatment

Treatment with nifurtimox and beznidazole is recommended (5). Nifurtimox dose in adults is 8–10 mg/kg/d in three divided doses over 30–120 days. Benznidazole is used in adults and children over 12 years of age at a daily dosage of 5–7 mg/kg, divided into two doses, one after breakfast and the other after dinner.

### 6.3 | Prevention

Use mosquito net or window protection, during night activities in forested areas, we recommend the use of repellent, pants and long-sleeved clothing, and foods of plant origin should preferably be pasteurized.

## 7 | LEISHMANIASIS

Leishmaniasis is a relatively common disease in returning travelers, accounting for approximately 5–10% of all travel-related diseases

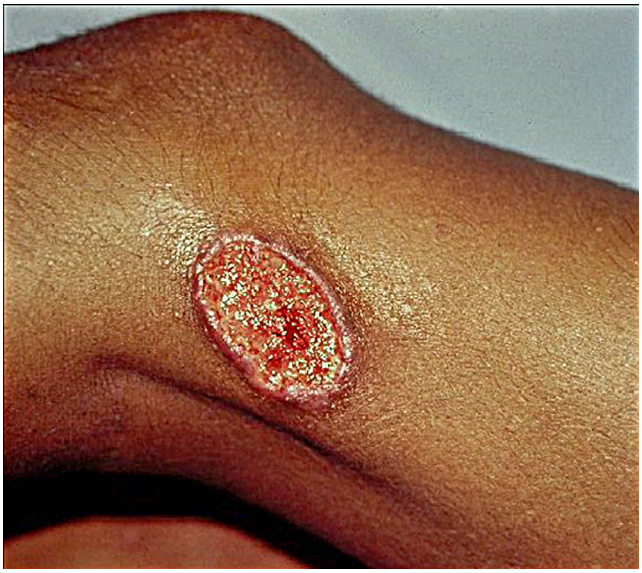

**FIGURE 3** Cutaneous leishmaniasis: An ulcerated plaque with indurated border

(Vasievich et al., 2016). It is caused by a protozoan parasite of the genus *Leishmania* and transmitted by a vector genus *Lutzomyia* (New World disease, i.e., originating from Central and South America) or *Phlebotomus* (Old World disease, i.e., originating from Africa, Asia, Middle East, and Southern Europe). The disease can manifest in four forms: localized cutaneous, diffuse cutaneous (or diffuse anergic cutaneous), mucocutaneous, and visceral. The progression from one stage to another largely depends on the *Leishmania* species and host response to the infection (Desjeux, 2004; Vasievich et al., 2016).

Cutaneous leishmaniasis begins on average after a 3-month incubation period as a solitary well-circumscribed erythematous papule at the site of the sandfly bite (Vasievich et al., 2016). The papule enlarges and forms nodules and ulcerated lesions with indurated borders (Figure 3). In some cases, the patient can present with the lymphangitic form of leishmaniasis. Diffuse (anergic) cutaneous leishmaniasis is a more extensive form of cutaneous leishmaniasis characterized by disseminated flesh-colored papules or nodules. In the New World disease, the most commonly involved *Leishmania* species are *L. mexicana*, *L. amazonensis*, *and L. braziliensis* (Rasti et al., 2016).

Mucocutaneous leishmaniasis is a rare form and can occur 1–2 years after the onset of primary cutaneous disease. It occurs almost exclusively in New World disease. Signs and symptoms of mucocutaneous disease include upper respiratory congestion and hoarseness, epistaxis with erythematous, edematous, and boggy mucosa with purulent drainage (Vasievich et al., 2016). This can lead to mutilating destruction of the mucous membranes and surrounding cartilage; most classically, ulceration of the septal mucosae. The disseminated form of leishmaniasis is known as visceral disease or Kala-azar (black sickness). It is often associated with fever, weight loss, weakness, pallor, hepatosplenomegaly, and lymphadenopathy.

### 7.1 | Diagnosis

The diagnosis is based on detection of amastigotes on tissue smear stained with Giemsa, skin biopsy from the ulcer edge where amastigotes inside macrophages can be found (biopsy is less sensitive than culture), and fine-needle aspiration. The polymerase chain reaction (PCR) offers high sensitivity in species determination which is important because the response to treatment varies among species of *Leishmania.* Other diagnostic methods include the delayed skin reaction test (Montenegro or Leishmanin skin test), serologic test (indirect immunofluorescence), electronic microscopy studies, culture in Novy–MacNeal–Nicolle (NNN) medium, and DNA probes (Khatami, Firooz, Gorouhi, & Dowlati, 2007). A major problem in leishmaniasis treatment is incorrect diagnosis in the presence of small number of parasites in zoonotic leishmaniasis and false-negative results in microscopic diagnosis (Khatami et al., 2007).

### 7.2 | Treatment

Pentavalent antimonials are preferred therapy for cutaneous leishmaniasis (Vasievich et al., 2016). This depends somewhat on the infecting species, as determined by PCR results, and risk of progression to mucocutaneous disease. Side effects of antimonial treatment include nausea, vomiting, diarrhea, fatigue, pancreatitis, cytopenias, and reversible electrocardiogram changes. Oral miltefosine may also be used; it has been shown to be efficacious and relatively well tolerated compared with antimonies. Pentaimidin-diisethionat (Pentacarinat®) i.m., i.v. can also be used. As second-line therapy, liposomal or non-liposomal amphotericin B is effective in patients who do not respond to antimonials (Vasievich et al., 2016. Also, pentoxifylline can be used as an adjunct to pentavalent antimonials (Rasti et al., 2016).

## 8 | SLEEPING SICKNESS OR AFRICAN TRYPANOSOMIASIS

Generally known as sleeping sickness, African human trypanosomiasis is a parasitic infection transmitted by tsetse flies. These flies can be found in 36 countries of sub-Saharan Africa, putting at risk about 60 million people. The infection affects the central nervous system, causing severe neurologic disorders. Untreated disease is fatal (Simarro et al., 2012; Welburn, Fèvre, Coleman, Odiit, & Maudlin, 2001).

### 8.1 | Diagnosis

The first stage of sleeping sickness has nonspecific symptoms, such as fever, headaches, weakness, itching, and joint pain. At this stage, sleep disease is easily treatable but difficult to diagnose. Without treatment, the parasite invades the central nervous system and the second stage of the disease begins which can be characterized by more specific symptoms, such as confusion, violent behavior, or seizures. The disease carries the name of its most striking symptom: patients experience the inability to sleep at night but are often overcome by sleep during the day. It is challenging to diagnose sleep disease before the second stage due to non-specific early stage symptoms. Once the parasite is detected, a lumbar puncture is required to examine the

cerebrospinal fluid. This will determine the stage of the disease and the appropriate treatment.

### 8.2 | Treatment

The type of treatment depends on the stage of the disease. The drugs used in the first stage of the disease are low in toxicity and easy to administer. However, the success of treatment in the second stage of the disease depends on a drug that can overcome the blood–brain barrier. The combination therapy nifurtimox-eflornithine (NECT) is the treatment currently recommended by WHO (Babokhov, Sanyaolu, Oyibo, Fagbenro-Beyioku, & Iriemenam, 2013). NECT is much safer than the previously used melarsoprol; approximately 5–20% of patients treated with melarsoprol died due to complications associated with its toxicity. New molecules are currently in the clinical testing phase in the hope that a safe and effective oral treatment will be developed for both stages of the disease. Currently, MSF is responsible for the efficient supply and distribution of all medicines used to combat sleeping sickness in the world. Prevention efforts, such as vector control, are crucial to keeping sleep disease under control.

## 9 | MANSONELLIASIS

Mansonelliasis' geographic distribution extends from Central America to South America and Antilles. Mansonelliasis is prevalent in the rural area in the city of Coari in Amazon, a location where the research of the Brazilian group of Paulo R. Cunha was done. The group performed two expeditions to tropical areas and residencies on the bank of Brazilian Amazon river. Out of 387 people, 96 (25%) presented positivity for microfilaria in the blood (Mansonelliasis). Mansonelliasis is caused by a filarial nematode known as *Mansonella ozzardi*, whose microfilariae is found in the peripheral blood, and it has specific morphological and biological characteristics. The nematode was first described by Manson in 1897 and was provided with updates by Faust, in 1929. The insect vector is possibly the *Cerqueirellum argentiscutum (simuliidae)* (Medeiros, Pessoa, & Camargo, 2014).

Mansonelliasis may provoke joint pains, headache, skin eruption (red papules), stains, itching, low fever, mental confusion, swollen lymph nodes, and hepatomegaly.

### 9.1 | Diagnosis

In endemic zones, the diagnosis is quickly and easily performed, even in children younger than 4 years old, through the blood smear examination that identifies a large number of microfilarias.

### 9.2 | Treatment

Ivermectin is treatment for *Mansonemma ozzardi.*

### 9.3 | Prevention

Appropriate clothing, special hats, protective barriers, a special kind of net with insecticides and chemicals with repellent properties help reduce the risk of traveler's contact with the parasites and their vectors.

## 10 | SCABIES

Scabies is caused by the female mite *Sarcoptes scabiei var hominis.* It occurs worldwide, and outbreaks are common in tropical areas. It typically follows intimate contact with an infested individual and can spread rapidly in crowded conditions. Travelers going to nursing homes, extended care facilities, schools, and prisons are at higher risk (Chosidow, 2000). The mite usually spreads from an affected individual to others via direct, prolonged, skin-to-skin contact. Short contact with an infected person, such as a quick handshake or hug, usually will not spread scabies. Scabies is spread easily to sexual partners and household members. The most common manifestations are intense, widespread itching, especially at night, and an eruption characterized by scattered inflamed papules, pustules, vesicles, and occasionally larger nodules; hyperkeratosis/crusting may be noted in heavily infested areas (Chosidow, 2006).

### 10.1 | Diagnosis

The diagnosis is established based upon the clinical features including the distribution of the eruption (web spaces of hands, wrists, sides of hands and feet, genitalia, and intertriginous areas) and the presence of intraepidermal burrow(s), the classic lesion of scabies. The diagnosis can be confirmed by identifying the mite or mite eggs or fecal matter (scybala). This can be done by carefully removing the mite from the end of its burrow using the tip of a needle or by examining under the microscope skin scraping for mites, eggs, or scybala. However, a person can still be infested even if mites, eggs, or fecal matter cannot be found; fewer than 10–15 mites may be present on an infested person who is otherwise healthy.

### 10.2 | Complications

The intense scratching can cause erosions that can become secondarily infected with *Staphylococcus aureus* or beta-hemolytic streptococci. Superinfection with *Streptococci* can lead to post-streptococcal glomerulonephritis.

### 10.3 | Treatment

A topical scabicide preparation, most commonly permethrin 5% cream, should be applied for 8–12 hr from the neck down to toes at bedtime, washed off in the morning, and reapplied the same way 1 week later (Vasievich et al., 2016). In addition, when treating infants and young children, the scabicide preparation also be applied to entire head and neck because scabies can affect these areas. Only permethrin or sulfur ointment may be used in infants. Oral ivermectin is a safe and effective treatment; however, it is not FDA-approved for this indication. Oral ivermectin should be considered for patients who have failed topical treatments or who cannot tolerate FDA-approved topical medications. If used for classic scabies, two doses of oral ivermectin (200 μg/kg/dose) should be taken with food 1 week apart. The

safety of ivermectin in children weighing less than 15 kg and pregnant patients has not been established.

### 10.4 | Prevention

Scabies can be prevented by avoiding direct skin-to-skin contact with an infested person or items such as clothing or bedding used by an infested person. Scabies treatment is recommended for household members, particularly those who have had prolonged skin-to-skin contact with an infected person. Household members and other potentially exposed persons should be treated simultaneously with the infested person to prevent reexposure and reinfestation. Bedding that came in contact with the skin and clothing worn within 3 days before treatment should be machine washed and dried using hot water and hot dryer cycles or be dry-cleaned. Items that cannot be dry-cleaned or laundered can be disinfested by storing in a closed plastic bag for several days to a week. Scabies mites generally do not survive more than 2–3 days away from human skin. Children and adults usually can return to child care, school, or work the day after treatment. Clean clothing should be worn after treatment. Both sexual and close personal contacts who have had direct prolonged skin-to-skin contact with an infested person within the preceding month should be examined and treated (Chosidow, 2000).

## 11 | YELLOW FEVER

Yellow fever (YFV) is a disease caused by a virus that transmitted through mosquito bites. Vector-borne transmission occurs via the bite of an infected mosquito, primarily *Aedes* or *Haemagogus* spp. Nonhuman and human primates are the main reservoirs of the virus, with anthroponotic (human-to-vector-to-human) transmission occurring. Travelers are at risk when visiting endemic areas of Africa and South America. Since January 10, 2018 travel-related cases of YFV, including four deaths, have been reported in international travelers returning from Brazil. None of the 10 travelers had received YFV vaccination (Vasconcelos, 2003).

Symptoms take 3–6 days to develop and include fever, chills, headache, backache, and muscle aches (Hamer et al., 2018). About 15% of infected individuals develop serious illness that can lead to bleeding, shock, organ failure, and sometimes death. A traveler's risk for acquiring YFV is determined by various factors, including immunization status, location of travel destination, season, duration of exposure, occupational and recreational activities while traveling, and local rate of virus transmission at the time of travel.

### 11.1 | Diagnosis

YFB diagnosis is established via virus isolation or nucleic acid amplification tests for viral RNA performed early in the disease process. Serologic assays that detect virus-specific IgM and IgG antibodies help in establishing the diagnosis.

### 11.2 | Treatment

There is no specific drug therapy for YFV. Management aims at symptomatic relief and providing life-saving interventions. Rest, fluids, and use of analgesics and antipyretics may relieve symptoms of fever and aching. Care should be taken to avoid medications such as aspirin or nonsteroidal anti-inflammatory drugs that may increase the risk for bleeding.

### 11.3 | Prevention

Avoidance of mosquito bites is the best way to prevent YFV. YFV can be prevented by taking a relatively safe, effective vaccine that is administered as a single subcutaneous injection. YFV vaccine is recommended for individuals aged ≥9 months who are traveling to or living in endemic areas (Querec et al., 2006; Staples, Bocchini, Rubin, & Fischer, 2015).

## 12 | LYMPHATIC FILARIASIS

Lymphatic filariasis is caused by thread-like worms *Wuchereria bancrofti* (90%) and *Brugia malayi* (10%). It is considered a neglected tropical disease, and the affected regions are located mainly in Asia, Africa, the Pacific Islands, some of the Caribbean Islands, and South America. Lymphatic filariasis is carried by different species of mosquitos. The adult worms live only in the human lymphatic system. Lymphatic filariasis spreads from person to person via mosquitoes. People with the disease can suffer from lymphedema and elephantiasis and in men, swelling of the scrotum, called hydrocele. Lymphatic filariasis is a leading cause of permanent disability worldwide. In endemic zones the exposure is universal, yet most people are asymptomatic (Dreyer, Norões, & Figueredo-Silva, 2000; Figueredo-Silva & Dreyer, 2005).

### 12.1 | Diagnosis

The standard method for diagnosing active infection is the identification of microfilariae in a blood smear by microscopic examination. The microfilariae that cause lymphatic filariasis circulate in the blood at night (nocturnal periodicity). Blood collection should be done at night to coincide with the appearance of the microfilariae, and a thick smear should be made and stained with Giemsa or hematoxylin and eosin. Serologic techniques provide an alternative to microscopic detection of microfilariae for the diagnosis of lymphatic filariasis. Patients with active filarial infection typically have elevated levels of antifilarial IgG4 in the blood and these can be detected using routine assays. ELISA techniques can be utilized in diagnosis.

### 12.2 | Treatment

Treatment starting with low doses of diethylcarbamazine 6 mg/kg for 12 days or single dose ivermectin (microfilaricidal) 200–400 μg/kg.

### 12.3 | Prevention

Avoiding mosquito bites is the most effective way to prevent lymphatic filariasis. The mosquitos that carry the microscopic worms usually bite between the hours of dusk and dawn. Persons at risk in endemic areas should sleep in an air-conditioned room or under a mosquito net at night. Also, they should wear long sleeves and

trousers and use mosquito repellent on exposed skin between dusk and dawn.

## 13 | CONCLUSIONS

Tropical skin diseases can be observed in travelers after returning to their home country. The dermatologist should be aware of the broad and variable manifestations of tropical diseases. Pre-travel counseling on tropical diseases should be encouraged as it can contribute to establishing a diagnosis and management plan promptly. Early diagnosis and intervention are crucial to decreasing morbidity and mortality in affected individuals. Obtaining the recommended vaccines is a very important means of protection from tropical diseases. During the trip, travelers should always have a first-aid kit with them and utilize means of protect from insect bites.

### CONFLICT OF INTEREST

The authors declare no conflicts of interest.

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