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THERAPY IN PRACTICE

Got the Travel Bug? A Review of Common Infections, Infestations, Bites, and Stings Among Returning Travelers

Matthew P. Vasievich[1] · Jose Dario Martinez Villarreal[2] · Kenneth J. Tomecki[1]

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Abstract The popularity of international travel continues to increase among Americans, even though they often experience subsequent illness on return from their journey. The pathogens responsible are not necessarily endemic to the destination itself but are often the result of poor sanitary conditions or activities engaged in while away. Skin disease ranks third among all medical concerns in returning travelers. This review addresses the pathogenesis, epidemiology, clinical presentation, and treatment of the most common skin diseases in returning travelers: insect bites and bedbugs, cutaneous larva migrans, scabies, tungiasis, myiasis, leishmaniasis, viral exanthems, and marine envenomation. Primary care physicians and dermatologists should be familiar with these illnesses and a general approach to their evaluation and management.

Key Points

Arthropod assault and infestation is a source of significant morbidity in returning travelers.

Travelers should bring proper protective clothing and insect repellant when travelling to areas where biting insects are vectors of disease.

Travelers should be acquainted with the hazards associated with local marine life if they plan on entering the water.

✉ Matthew P. Vasievich
vasievm@ccf.org

[1] Department of Dermatology, Cleveland Clinic Foundation, 9500 Euclid Ave, Cleveland OH, USA

[2] Department of Internal Medicine, University Hospital, "José E. González," Universidad Autónoma de Nuevo León, Monterrey, Mexico

1 Introduction

Skin disease is one of the most common medical sequelae after travel abroad. Americans travel all over the world, but most commonly to Mexico, Canada, Jamaica, and the Dominican Republic as well as Western Europe [1]. The restoration of diplomatic ties with Cuba may make this a novel and popular travel destination as well. However, emerging destinations such as Brazil, Peru, and countries in south and southeast Asia such as Myanmar, Cambodia, and India are becoming increasingly sought-after vacation spots [2].

This review discusses the cutaneous manifestations of diseases commonly seen in returning travelers from the western hemisphere, including new trends in US citizens traveling abroad as described by the GeoSentinel database of illness in international travelers [3]. The diseases are not necessarily endemic to a particular region but may occur as a result of variability in sanitary conditions, travel to forested areas, or rustic accommodations.

2 Insect Bites

Insect bites are a common complaint in returning travelers. Bites typically result from an insect trying to obtain a blood meal from a host. Clinically, they appear as erythematous edematous papules that can be distributed singly, grouped, or generalized, depending on the amount of accessible skin and the number and type of insects biting. A robust host response to the bite can produce a bulla. Pruritus often results either as a direct result of the bite or as a host response to proteins injected into the skin by the insect.

One of the most common biting insects encountered by travelers is the bedbug. Bedbugs (genus *Cimex*) are sub-

centimeter reddish-brown flat arthropods found throughout the world. They live within the cracks, crevices, and seams of mattresses and chairs and generally only come out onto the resting surface to obtain a blood meal from a host. Bedbugs are more common in poor areas, but can potentially be found anywhere humans sit, rest, or sleep, including hotels, theaters, airplanes, workplaces, and schools. They feed when the host is sleeping or otherwise distracted and still. The bite is painless and occurs on exposed skin—primarily the face, neck, hands, and arms. The papules of bedbug bites are often in a linear pattern as a bug probes multiple sites looking for a suitable blood vessel on which to feed (Fig. 1). Other signs of bedbug infestation include specks of feces on bedsheets, wallpaper, or box springs. Specks of blood on sheets are also suggestive. Bedbugs can reside in luggage, which can result in infestation of a traveler's home on their return. Thus, suitcases should be kept off the floor and closed when not in use. Once a building is infested with bedbugs, eradication is difficult. Measures to remove bedbugs include heat treatments, as bedbugs cannot tolerate temperatures above 50 °C, and insecticides, though resistance to some compounds has been reported [4].

Describing the biting patterns of other specific insect species encountered in rural or wilderness areas is not possible within the confines of this review. However, if travelers do plan on spending significant amounts of time in these areas, they should be familiar with the most likely species to be encountered and bring appropriate protective clothing and insect repellant. The papules and pruritus from the bites themselves are generally self-limited, and treatment is aimed at symptom control. Usually mid-potency topical steroid creams or ointments are appropriate, with oral antihistamines as needed. For severe or generalized bites, a short course of oral corticosteroids or an intramuscular corticosteroid injection could be used.

3 Cutaneous Larva Migrans

3.1 Pathophysiology

Cutaneous larva migrans is a skin disease caused by the hookworms *Ancylostoma braziliense*, *Ancylostoma caninum*, and *Uncinaria stenocephala*, natural parasites of dogs and cats. Humans are a dead-end host. The worms themselves are approximately 0.5 mm in diameter, but are occasionally longer. The parasites are excreted in the host animal feces, often on soil or sand (beaches) and subsequently acquired by direct skin contact with individuals laying out on beaches or walking barefoot along the water. The worms invade the skin but lack a secreted collagenase enzyme required to penetrate the basement membrane and thus become trapped in the epidermis, which results in serpiginous erythema and pruritus [5].

3.2 Epidemiology

Cutaneous larva migrans is one of the most common skin diseases seen in returning travelers, accounting for about 10 % of all skin disease. It is most often acquired on beaches in Asia, Africa, South and Central America, and the Caribbean [3].

3.3 Clinical Presentation

The incubation period for the disease is 5–15 days, after which extremely pruritic linear or serpiginous erythematous tracts appear, which represent the path of single larva through the epidermis (Fig. 2). The tracts are most commonly found on the feet, followed by the thighs and legs or buttocks. The average number of tracts per patient is three. The rate of progression of the path is approximately 2–3 cm/day. The parasite in the skin causes a hypersensitivity reaction in the host, which causes the pruritus. Laboratory

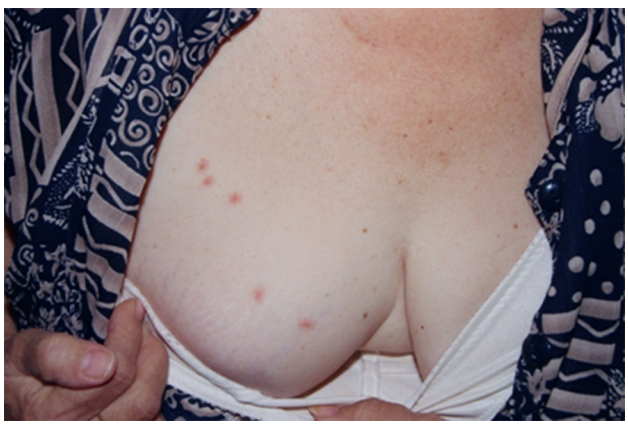

Fig. 1 Bedbug bites

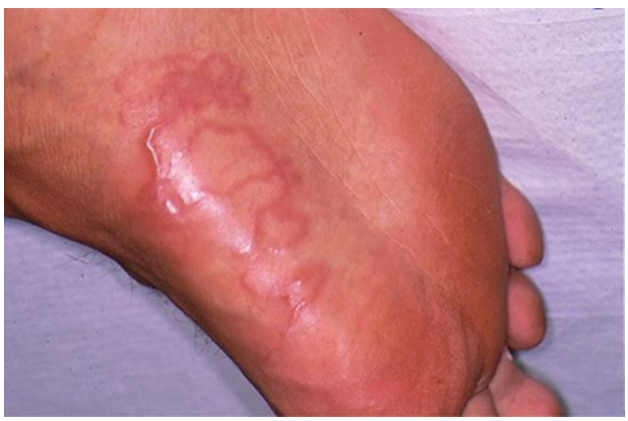

Fig. 2 Cutaneous larva migrans

findings can include peripheral eosinophilia and elevated serum immunoglobulin (Ig)-E. Differential diagnosis includes pili migrans, migratory myiasis, larva currens, scabies, gnathostomiasis, and strongyloidiasis. The diagnosis of cutaneous larva migrans is made clinically. There have been reports of dermoscopy and confocal scanning laser microscopy being used to aid in identifying the worms, though the sensitivity of these techniques has not yet been established. Possible complications include superinfection/impetiginization, bullae, and papular urticaria [6]. Loffler's syndrome is a systemic disease of unknown etiology that consists of migratory pulmonary infiltrates and peripheral eosinophilia. In rare cases, it has been associated with cutaneous larva migrans. Whether this represents direct invasion of the lungs by the worm or a systemic response to the infestation is yet to be determined [7].

3.4 Treatment

Cutaneous larva migrans is a self-limited disorder that usually resolves within 2–3 months as the parasites die within the epidermis. As such, treatment may not be necessary though will likely be desired by most patients. A single dose of ivermectin (12 mg in adults, 150 μg/kg in children) is curative in 80–100 % of cases; alternatives are albendazole 400 mg as a single dose or topical thiabendazole 10–15 % three times daily for 15 days. Cryotherapy of the advancing edge of the tracts is not usually effective [8].

4 Scabies

4.1 Pathophysiology

Scabies is caused by the female mite *Sarcoptes scabiei var hominis*, which burrows into the stratum corneum after mating and lays eggs in 1–2 days within the burrow. The larvae subsequently hatch within 3 days, emerge from the skin, and then burrow again to mature. The host typically mounts an immune response against the mite, which accounts for the pruritus [9, 10]. In naive hosts, symptoms can take 2–6 weeks to occur, whereas in non-naive patients, symptoms typically occur within 12–72 h [11].

4.2 Epidemiology

Scabies occurs throughout the world and typically follows close intimate contact with an infested individual. Sexual transmission is common in adults. Mites can be passed from mother to child, particularly in nursing infants. Occasionally, the mite can also be contracted by sleeping in a bed previously used by an infected individual [12].

Scabies can be fulminant and pronounced as crusted scabies in which the parasite burden grows exponentially and results in thick crusted plaques. Those at risk for crusted scabies include aboriginal Australians; patients with Down's syndrome, leprosy, or human T-lymphotropic virus (HTLV)-1 infection; and those who are immunosuppressed or have a sensorineural deficiency such as patients with spinal cord injury or Parkinson's disease. Patients with crusted scabies are usually asymptomatic as a result of an impaired immune response or itch sensation [13, 14].

4.3 Clinical Presentation

Scabies typically presents with erythematous papules or nodules with linear burrows in flexural areas, finger and toe web spaces, and in the groin [9] (Fig. 3). In the crusted variant, thick hyperkeratotic crusted plaques develop on the trunk or extremities. Differential diagnosis for conventional scabies includes dyshidrotic eczema, folliculitis, papular urticaria, cutaneous larva migrans, and arthropod assault. Diagnosis is made by identifying mites, eggs, or feces on a mineral oil prep of the keratotic debris obtained by scraping a burrow with a scalpel blade. Alternatively, a burrow ink test can be conducted by gently rubbing a fountain pen on one of the papules, covering it with ink. When excess ink is wiped away with alcohol-soaked gauze, the remaining ink will track down the scabetic burrow. The head of the mite can be appreciated on dermoscopy; it appears as a black triangular dot, known as the 'delta-wing-jet' sign. The body of the mite is translucent and cannot be seen. A scabies infestation can become superinfected from the persistent scratching or from skin breakdown, especially in crusted scabies [15].

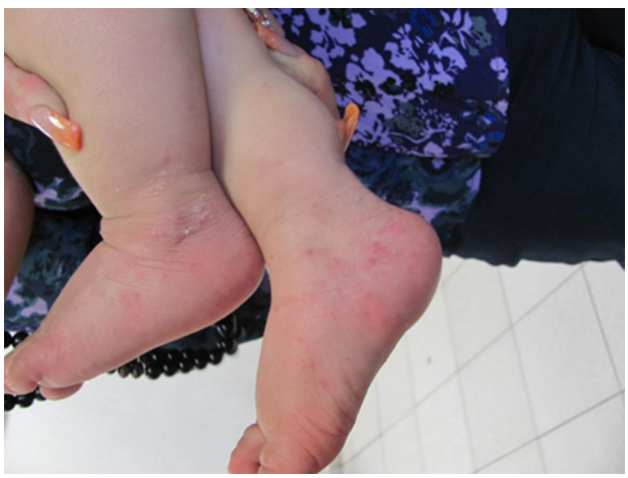

Fig. 3 Scabies

4.4 Treatment

Treatment is permethrin 5 % cream applied for 8–12 h from the neck down at bedtime, washed off in the morning, and repeating 1 week later. The reapplication is necessary because the permethrin is not efficacious against eggs of the parasite, only the live mites. Alternatively, ivermectin 200 μg/kg in two doses 2 weeks apart is effective in 90–100 % of patients. Second-line treatment for the infestation includes 10 % topical crotamiton or topical 1 % lindane, though lindane has been associated with neurotoxicity after repeated use. Household contacts also should be treated to prevent re-infection. Patient clothing and bedding should be washed in hot water or sealed in plastic garbage bags for 1 week if laundering the items is not possible. Itch can persist for several weeks after treatment; antihistamines can be used [16].

For crusted scabies, the United States Centers for Disease Control (CDC) has published a treatment protocol based on expert opinion in which permethrin cream is applied daily for 1 week then twice-weekly coupled with ivermectin 200 μg/kg on days 1, 2, 8, 9, and 15. Ivermectin can also be given on days 22 and 29 if needed. Cure is ascertained by clinical improvement and confirmatory negative skin scrapings [17].

5 Myiasis

5.1 Pathophysiology

Myiasis has three forms: follicular/furuncular myiasis, wound myiasis, and migratory myiasis. Follicular or furuncular myiasis is caused by one of four different types of flies: the human botfly *Dermatobia hominis*; the Tumbu fly *Cordylobia anthropophaga*; *Cuterebra* species, known as rabbit or rodent bot flies; and *Wohlfahrtia vigil* and *W. opaca* [18, 19]. In furuncular myiasis, the female fly lays eggs on foliage or deposits them on a blood-sucking insect with a quick-drying glue-like substance. When the insect lands on a host for a blood meal, or the host brushes against the foliage, the eggs hatch and the larvae quickly burrow under the skin of the host, where they mature and feed on the host blood for 5–10 weeks. Once the larva matures, it emerges, drops to the ground, and continues development to a mature fly in the soil [20].

Wound myiasis follows fly infestation of open wounds, mucous membranes, and body cavities. Flies that cause wound myiasis worldwide are *Cochliomyia hominivorax*, *Chrysomya bezziana*, and *Wohlfahrtia magnifica.* The female fly lays eggs around the edges of wounds or on mucous membranes, particularly the nose and orbit. Eggs hatch in 1–2 days, and larvae feed on tissue for approximately 1 week, which can increase the size of the wound. The spines on the body of the larvae serve as an anchor to the wound base, which can make removal of the larvae difficult. Infestation of the wound produces a characteristic odor that attracts more flies to lay eggs. After maturing, the larvae fall to the ground to complete maturation to an adult fly. The entire life cycle is approximately 24 days [18, 19].

Migratory or creeping myiasis is caused by the flies *Gasterophilus intestinalis* and *Hypoderma ovis* and *lineatum*. The female fly lays eggs on the leg hairs of the distal extremities of horse or cattle. The eggs remain dormant until the animal bites or licks the areas where the eggs are laid. The larvae subsequently hatch and burrow into the oral mucosa; from there, they are ultimately swallowed and remain in the gut for 8–11 months until they pass through the feces to the ground where they pupate. 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. On hatching, the larvae burrow into the epidermis and migrate 1–30 cm per day; however, they ultimately die as they are unable to pupate [18, 21, 22].

5.2 Epidemiology

Myiasis is commonly seen in travelers returning from sub-Saharan Africa, Latin America, and the Caribbean, less so from northeast Asia, and represents 1–5 % of illness in returning travelers from these regions. However, cases of furuncular and migratory myiasis have also been reported across North America and Southern Europe [3, 18, 19].

5.3 Clinical Presentation and Diagnosis

Furuncular myiasis (Figs. 4, 5) typically exhibits one or more erythematous papule or nodule, ranging in size from 0.2 to 2 cm with a central pore or punctum, which is the caudal spiracle of the larva through which it breathes and expels waste—a spontaneous serosanguinous drainage from the site. Favored sites 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. Diagnosis is primarily clinical and often strengthened by the travel history. If the furuncle is closely observed, movement can often be appreciated around the punctum, and the spiracle can be appreciated on dermoscopy. If imaging is necessary, ultrasound or magnetic resonance imaging (MRI) may be used. Differential diagnosis includes an inflamed cyst, abscess, folliculitis, or other arthropod bite. If the nodules are submerged in water and the organism is alive, air bubbles may appear at the punctum [23, 24].

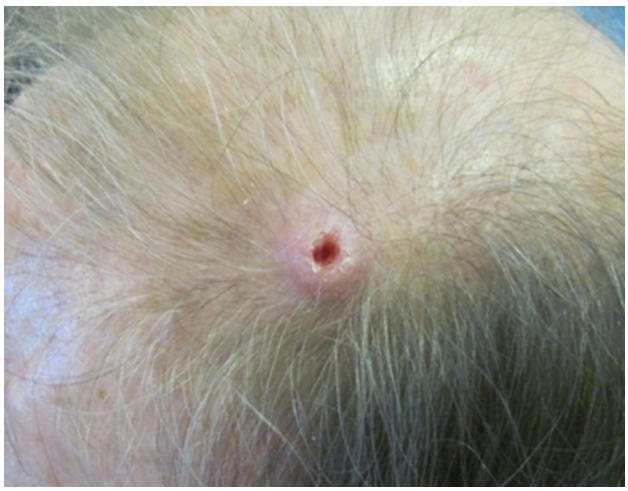

Fig. 4 Furuncular myiasis

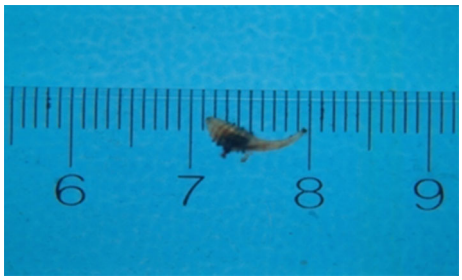

Fig. 5 Botfly larva

Migratory myiasis is characterized by pruritic serpiginous linear tracts that form as the larva burrows through the skin. The tracts may resemble the helminthic infection cutaneous larva migrans, with a similar differential diagnosis, though migratory myiasis travels more slowly and can persist in the skin for months, unlike the helminthic infestation. Fly larvae are also larger and can be visualized with mineral oil and dermoscopy [18].

Wound myiasis is characterized by the presence of larvae in an open wound or mucous membranes. In severe cases, fever, chills, pain, superinfection of the site, and peripheral neutrophilia or eosinophilia may occur [25].

5.4 Treatment

Furuncular myiasis is treated with occlusion of the punctum with vaseline for 24 h, thus suffocating the organism, causing it to emerge from the skin. An eccentrically placed cruciform incision can be helpful in removal, as incision directly over the punctum can result in transection of the larva and incomplete removal [23, 24, 26]. Migratory myiasis can be treated surgically with removal of the larva from the leading edge of the tract using a sterile needle or local anesthetic followed by incision and removal of the larva [19]. Irrigation and debridement is necessary for wound myiasis. For large wounds, general anesthesia may be necessary [18].

6 Tungiasis

6.1 Pathophysiology

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. While implanted in the host, the flea grows approximately 2000-fold in size, remains in the host for approximately 4–6 weeks, and subsequently dies and is sloughed off by the host. The parasite can also be carried by animal hosts, including dogs, pigs, cows, and rats, which can lead to persistence of the organism in rural communities despite eradication efforts [27–29].

6.2 Epidemiology

Tungiasis is found worldwide in both the eastern and the western hemispheres, including sub-Saharan Africa, India, Pakistan, and especially in the Caribbean, where disease prevalence is 15–50 %. In endemic areas, burden of disease can be particularly high in individuals with multiple organisms [3, 28].

6.3 Clinical Presentation

The sand flea favors acral sites, most commonly the toes and peri-ungal 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. The differential diagnosis includes a callus, clavus, friction blister, or traumatic hematoma. As the organism grows and becomes symptomatic, itching may result in superinfection. In communities without established vaccination programs, infection with *Clostridium tetani* can also occur [30, 31].

6.4 Treatment

Treatment for tungiasis is physical removal of the organism; a simple shave or punch biopsy procedure is usually sufficient. Otherwise, a sterile needle may be used. Alternatively, in patients with significant disease, topical ivermectin, metrifonate, or thiabendazole may also be used [32].

7 Leishmaniasis

7.1 Pathophysiology

Leishmaniasis is a relatively common disease in returning travelers, accounting for approximately 5–10 % of all travel-related diseases [3]. Caused by a protozoan parasite of the genus *Leishmania*, leishmaniasis is broadly classified into 'Old World' and 'New World' disease. Old World leishmaniasis originates from Africa, Asia, the Middle East, and Southern Europe; New World disease originates in Central and South America, occasionally as far north as Texas [33–35]. Leishmaniasis in returning travelers to the USA has increased in recent years, owing to increased travel abroad and to the activities of the US military and contractors in the Middle East. Old and New World leishmaniasis can be classified into 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 [33].

The life cycle of *Leishmania* in the human host starts with the bite of the female sandfly of either the *Phlebotomus* (Old World) or *Lutzomyia* (New World) genera. The flagellated promastigote form of the protozoa is transferred from the proboscis of the sandfly to the tissue of the host, where it is rapidly phagocytosed by macrophages, dendritic cells, and neutrophils. The parasite then transforms into the non-flagellated amastigote form and multiplies in the cytoplasm of the host cells. If the host is again bitten by a female sandfly, the parasites are transferred to the gut of the fly, transform back into the flagellated promastigote form and subsequently migrate to the proboscis of the fly in preparation to infect the next host. The *Leishmania* genus is divided into *Leishmania* and *Viannia* subgenera if the protozoa replicates in the foregut or mid- to hind-gut of the sandfly, respectively. The fly itself is smaller than a mosquito and inaudible; it has a painless bite that typically occurs on exposed skin of the head, neck, and extremities between dusk and dawn. Animal reservoirs include rodents and dogs [36].

7.2 Epidemiology

Global prevalence of both Old World and New World leishmaniasis is estimated at 12 million, with an annual incidence of approximately 2 million, of which 1.5 million are thought to be cutaneous leishmaniasis. Mortality is estimated at 20,000–30,000 annually, second only to malaria for diseases caused by a protozoan [37]. More than 90 % of New World cutaneous disease originates from Brazil and Peru; Old World disease originates mostly from Iran, Afghanistan, Syria, and Saudi Arabia [38, 39]. New World disease is typically limited to Central and South America; disease is uncommon in the USA, although occasional case reports have come from Texas and Oklahoma [40].

7.3 Clinical Presentation

Cutaneous leishmaniasis (Fig. 6) begins after a 1- to 2-month incubation period as a solitary well-circumscribed erythematous papule at the site of the sandfly bite. The papule enlarges and forms larger nodules and plaques with indurated borders that may ulcerate. Cutaneous leishmaniasis can be caused by numerous species in both Old World and New World disease [39]. Diffuse (anergic) cutaneous leishmaniasis (Fig. 7) is a more extensive form of cutaneous leishmaniasis characterized by disseminated flesh-colored papules or nodules; however. *Leishmania mexicana* usually causes an ulcer. Primarily a New World disease, diffuse cutaneous leishmaniasis is caused by *L. mexicana*, *L. amazonensis*, and *L. venezuelensis* species [33, 41]. Mucocutaneous leishmaniasis is a rare form, almost exclusively in New World disease, and can occur 1–2 years after the onset of primary cutaneous disease. Signs and symptoms of mucocutaneous disease include upper respiratory congestion and hoarseness, often epistaxis with erythematous, edematous, and boggy mucosa with purulent drainage. This can lead to mutilating destruction of the mucous membranes and surrounding cartilage; most classically, ulceration of the septal mucosae. Progression rates to mucocutaneous leishmaniasis range from 1 to 10 % [37, 42, 43]. The disseminated form of leishmaniasis, known as visceral disease or Kala-azar (black sickness) is caused by migration of infected tissue macrophages through the reticuloendothelial system. Patients exhibit fever, weight loss, weakness, pallor, hepatosplenomegaly, and lymphadenopathy. Visceral leishmaniasis can originate from either Old or New World

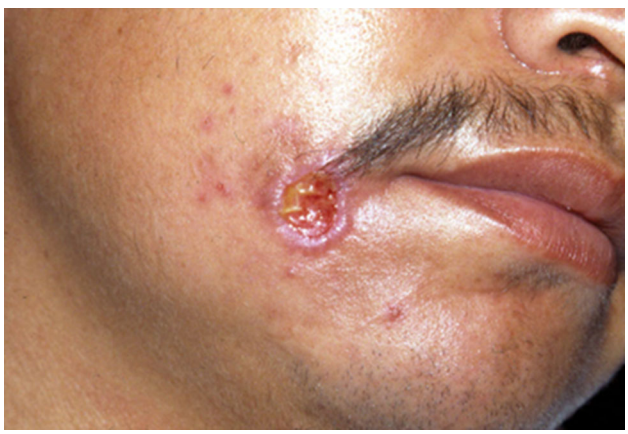

Fig. 6 Cutaneous leishmaniasis

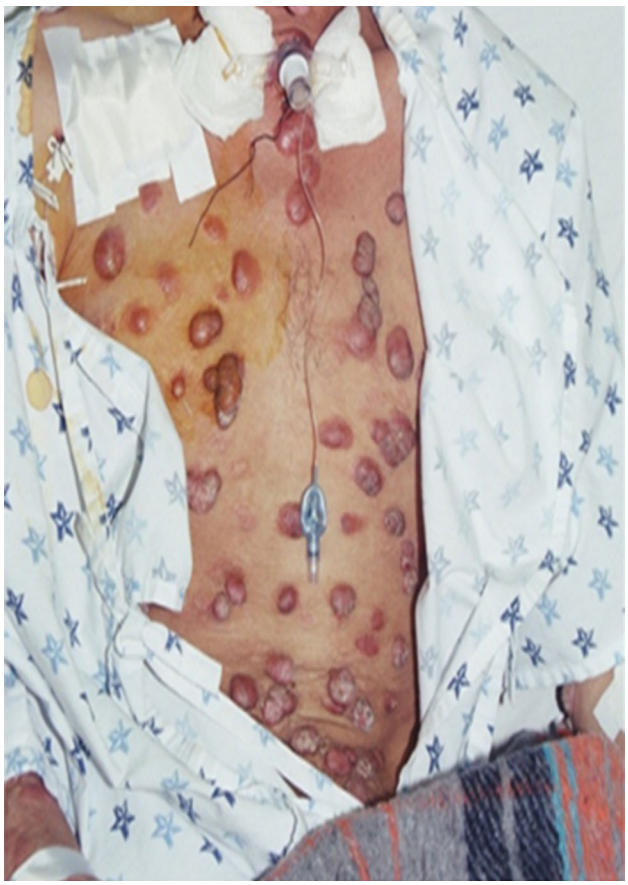

Fig. 7 Diffuse anergic cutaneous leishmaniasis

disease, with patients from Bangladesh, Brazil, Ethiopia, India, Sudan, and South Sudan accounting for >90 % of cases [37].

The differential diagnosis for cutaneous leishmaniasis is broad and includes malignancy, mycobacterial, non-mycobacterial, and fungal infections of the skin and arthropod assault. A good travel history is essential for making the diagnosis. Skin biopsy reveals a dense dermal mixed infiltrate with histiocytes, lymphocytes, neutrophils, and plasma cells. Intracellular amastigotes may be present within inflammatory cells, though sensitivity for the organism is highly variable, ranging from 19 to 77 % [37, 44]. Polymerase chain reaction (PCR) helps to confirm the diagnosis, either with a skin biopsy specimen or fine needle aspirate. PCR can also identify the organism, which can guide choice of treatment [45].

7.4 Treatment

Treatment of leishmaniasis can be difficult. As a general rule, pentavalent antimonials—either intravenous sodium stibogluconate or intramuscular meglumine antimoniate, both 20 mg/kg/day for 3–4 weeks—are preferred therapy for cutaneous leishmaniasis. This depends somewhat on the infecting species, as determined by PCR results, and risk of progression to mucocutaneous disease [46]. All patients with New World leishmaniasis should receive systemic therapy as well as disseminated mucocutaneous or visceral Old World leishmaniasis. Side effects of antimonial treatment include nausea, vomiting, diarrhea, fatigue, pancreatitis, cytopenias, and reversible electrocardiogram (ECG) changes. Cure rates can range widely from 52 to 95 % with antimonials [47–49]. Oral miltefosine may also be used in both Old World and New World disease as it has been shown to be efficacious and relatively well tolerated compared with antimonials [50]. As second-line therapy, liposomal or non-liposomal amphotericin B are effective in patients who are non-responsive to antimonials. Liposomal amphotericin B is preferred because of its shorter dosing regimen and better side effect profile. For uncomplicated localized disease in Old World leishmaniasis, topical therapies such as paromomycin, cryotherapy, or intra-lesional antimonials can be effective and helpful [51, 52].

8 Viral Exanthems

8.1 Dengue

Dengue is an RNA flavivirus that is transmitted from person to person by the *Aedes aegypti* and *Aedes albopictus* mosquitos. The shipping industry and air travel have led to both mosquito species now being present throughout the world, though *A. aegypti* primarily resides in the tropics. The mosquitos can live in forested, rural, and urban areas. They will also enter homes, so travelers are susceptible even if they do not spend significant amounts of time outdoors. Dengue outbreaks have largely been confined to the tropics, though cases have been reported in the Americas. After inoculation of a host by a mosquito bite, the incubation period for the virus averages 4–7 days. Symptoms are somewhat non-specific and include fever, headache, severe myalgias, arthralgias, and gastrointestinal symptoms. Cutaneous manifestations occur in about half of patients and consist of diffuse erythema or a morbilliform rash with islands of sparing (Fig. 8). Petechiae may also be noted on the skin. Diagnosis is by reverse-transcription PCR (RT-PCR) or immunoassays to detect antibody titers against the virus. Treatment is aimed at symptom control, as the disease is usually self-limited and fevers resolve within 2–7 days, though fatigue can persist following infection. Complicated dengue can occur with re-infection in the form of dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS). Mortality rates for DHF or DSS patients are <1 % in centers experienced in treating the disease [53].

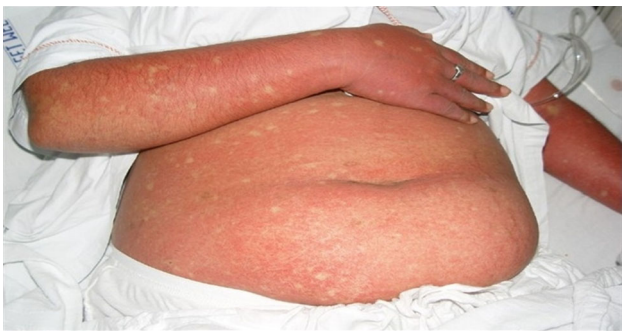

Fig. 8 Morbilliform exanthem with islands of sparing associated with dengue virus infection

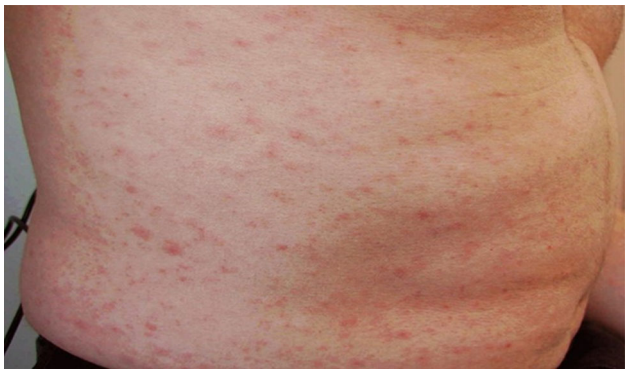

Fig. 9 Pink- to salmon-colored papules associated with chikungunya virus infection

8.2 Chikungunya

Chikungunya virus is an RNA togavirus transmitted by the same *Aedes* mosquito species as dengue. Outbreaks are again generally confined to the tropics, though cases have been reported in temperate climates such as northern Italy and the mid-western USA. Similar to dengue, the virus incubates for 3–7 days. Symptoms include high fever, symmetric polyarthritis, and gastrointestinal symptoms. Pruritic light pink papules that can progress to bullae occur in 40–50 % of patients 2–5 days after the fever (Fig. 9). The arthritis can be chronic, with more than half of patients reporting joint pain for longer than 18 months. Diagnosis is made by RT-PCR or serologies for IgM or IgG against the virus. Treatment is primarily supportive; non-steroidal anti-inflammatory drugs (NSAIDs) and oral corticosteroids are used for the joint pain associated with the virus [53].

8.3 Zika

Zika, like dengue, is an RNA flavivirus transmitted by *Aedes aegypti*, *A. africanus*, and *A. albopictus* mosquitos. The virus was first isolated in Uganda in the late 1940s. Scattered cases had been reported in returning travelers from Africa and Southeast Asia in the late 2000s and early 2010s. The first case in South America was reported in Brazil in 2015, and the virus has spread rapidly since, with cases reported in approximately 29 countries or territories in the Americas as of early 2016. Zika infection is asymptomatic in most cases. Patients who are symptomatic experience mild fever, arthralgias in the small joints of their hands, headache, non-suppurative conjunctivitis, and a morbilliform rash (Fig. 10). Diagnosis is by serum or urine RT-PCR or RT-PCR of amniotic fluid in pregnant women. Complications of Zika virus infection include Guillain–Barré syndrome and microcephaly in the fetus of pregnant women infected in the first trimester. Treatment is again primarily supportive with preventive measures, such as insect repellant and protective clothing, paramount in anyone travelling to areas where they might be exposed to Zika virus [54].

9 Marine Envenomation

Marine envenomation is a common complaint in travelers to North America, Australia, and Oceania, as well as travelers to the Middle East and the Mediterranean region of Europe, and accounts for 5–10 % of travel-related complaints from these regions [3]. Symptoms from most stings are acute and require immediate medical attention; however, it is important to continue care in the returned traveler after initial treatment at the time of injury.

9.1 Stingray

Stingrays are large flat fish that live and feed on the bottom of fresh and salt waters in tropical and subtropical climes worldwide, including several species in the USA. Most stingray stings occur in victims unintentionally startling or stepping on the fish. The stinger is a barbed apparatus located on the dorsal aspect of the tail, which whips forward and embeds in the skin. The stinger has an associated gland that coats it in venom and mucus. The sting itself is intensely painful, and the stinger often breaks off, leaving fragments lodged in the victim. Pain and edema peak within 30–90 min and can last 1–2 days. Nausea, vomiting, weakness, and balance disturbances may also occur [55]. Treatment consists of obtaining hemostasis of the wound and immersing the wound in hot water (110–114 °F or 43–46 °C) to ameliorate the pain; infiltration with local anesthetic may be helpful. The wound should be explored for stinger debris, cleaned, debrided, packed, and left to heal by secondary intent. The patient should receive tetanus toxoid prophylaxis and possibly a prophylactic antibiotic to prevent soft tissue infection [56, 57].

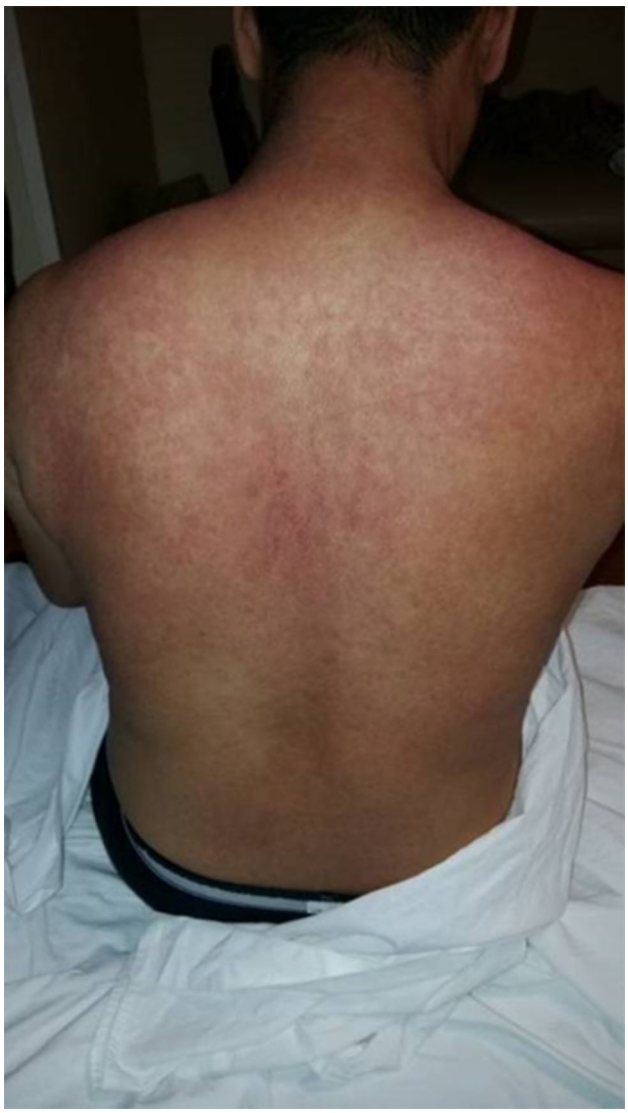

Fig. 10 Morbilliform exanthem associated with Zika virus infection

9.2 Spiny-Finned Fish

Lionfish, stonefish, and weever fish are spiny-finned fish that are commonly implicated in marine stings, not only in travelers/swimmers but also (particularly lionfish) in individuals with saltwater aquariums [58]. Stonefish live in tropical waters and on reefs, particularly off the coasts of Australia and India. They feed near the ocean floor and are reddish to gray-brown colored as camouflage with their surroundings. Stings often occur from the victim stepping unknowingly on the fish. Weever fish are sandy-brown fish, 6–12 inches in length, that inhabit shallow waters around Europe; they are bottom dwellers, with stings occurring when a victim steps on them. Lionfish are striped fish indigenous to the reefs of the Indian and western Pacific Oceans, though their numbers have also been increasing in Caribbean waters [59]. Lionfish are not aggressive to swimmers, and stings usually occur in fishermen or aquarium owners [58]. A spiny fish sting is similar to a stingray sting. Pain peaks within the first 30–90 min and generally resolves within 1–2 days. Nausea, vomiting, and weakness can occur, likely as a result of the pain from the sting. Treatment is similar to a stingray attack as described above [60]. Of note, anti-venom is available for stonefish stings, often at clinics near waters the fish inhabit. Dosing of the anti-venom should correlate with the number of puncture wounds observed: intramuscular 2000 U for 1–2 punctures, 4000 U for 3–4 punctures, and 6000 U for $\geq$5 punctures. There is a risk of anaphylaxis to the anti-venom, and a serum-sickness reaction can occur within 3 weeks of anti-venom administration. The anti-venom is not FDA approved [61].

9.3 Box Jellyfish

The box jellyfish (*Chironex fleckeri* or commonly, the 'sea wasp') lives in the Indian and Pacific Oceans off Australia and south-east Asia. The animal measures approximately 12 inches in diameter, and its tentacles are several meters in length, each with thousands of nematocysts, which are hollow needle-like structures that deploy from a spring-like apparatus and inject venom into the victim. The box jellyfish is one of the most venomous animals in the world. Human death can occur within seconds to minutes of contact with the toxin. The mechanism of action of the jellyfish toxin is unknown but is thought to circulate in the blood of the victim after a sting and create pores in the plasma membrane of cells, causing a potassium efflux and cardiovascular collapse and/or respiratory failure [62, 63]. Box jellyfish stings produce flagellate edematous purpura that may evolve to bullae and necrosis. Fever, nausea, vomiting, and headache may occur. Initial treatment is stabilization of hemodynamics and respiratory status. Application of 5 % acetic acid (white vinegar) helps to decontaminate the victim from undischarged nematocysts. Freshwater and physical rubbing can exacerbate the sting by triggering undischarged nematocysts. After decontamination, box jellyfish anti-venom should be administered, coupled with cold packs topically and analgesia as needed. Topical or systemic corticosteroids and antihistamines may also be helpful [64, 65].

9.4 Portuguese Man-of-War

The Portuguese Man-of-War (*Physalia physalis*) is found worldwide. The organism is actually a colony of smaller polyp-like organisms that floats on the surface of the ocean using a gas bladder filled with nitrogen and carbon monoxide gases. It has approximately 40 tentacles, some 30 m in length, to ensnare its prey. The Man-of-War floats

passively on the ocean, propelled only by the wind and waves; as such, it occasionally can be found washed up on beaches. Man-of-War stings are characterized by flagellate erythema from nematocyst discharge and associated severe pain that can last up to several hours. Nausea, malaise, headache, delirium, and gait ataxia can occur. Hypotension and respiratory failure are uncommon but may occur in severe stings. Treatment for Man-of-War stings is similar to that for box jellyfish stings, but no anti-venom is available [56, 65].

9.5 Sea Bather's Eruption

Sea bather's eruption is a pruritic dermatitis caused by the larvae of the thimble jellyfish (*Linuche unguiculata*), which are present throughout the world, though commonly found in the waters off Florida and in the Caribbean. The dermatitis develops when larvae, which measure only 1–2 mm in diameter, become trapped beneath the swimwear of the victim and cause pruritic erythematous edematous papules and wheals resembling urticaria (Fig. 11). The papules and wheals occur within a day of exposure to the larvae and can persist for up to 2 weeks. Severe reactions can cause headache, fever, chills, nausea, and vomiting. Treatment is primarily supportive with acetic acid decontamination followed by symptomatic treatment with topical corticosteroids and oral antihistamines. If symptoms are severe, systemic steroids may be effective [66, 67].

10 Dermatologic Conditions That Worsen During Travel

It is worth mentioning that individuals with chronic dermatologic diseases should be aware that travel may exacerbate their condition. In patients with rosacea, many of the common triggers of the disease such as sunlight, spicy foods, alcoholic beverages, and caffeine are commonly consumed in greater amounts while traveling and on vacation. Patients with acne and seborrheic dermatitis who travel to warmer climes may flare with warm weather, sweating, and sunscreen creams that can cause follicular occlusion. Acne patients are particularly at risk for this, as many of the topical and systemic medicines given for acne vulgaris cause photosensitivity and merit aggressive sunscreen use. These travelers should also be conscientious to bring appropriate over-the-counter and prescription medications with them on their trips, as 1- to 2-week lapses in therapy may also result in a flare. In patients with psoriasis, the converse may be true as someone whose home is in a sunny locale may experience a psoriasis flare when visiting cooler regions where longer clothing is appropriate for warmth and the extremities are not exposed to ultraviolet (UV) light from the sun, which suppresses formation of psoriatic plaques. Lastly, it is important to note that even conscientious travelers who make an earnest attempt to pack the topical medicines necessary to maintain their dermatologic condition may have larger tubes and containers of creams and ointments confiscated due to security regulations pertaining to quantities of creams, liquids, and gels that may be carried onto an airplane. If the patient is not prepared to check their bag, they may forfeit their medicines and experience a flare on their trip.

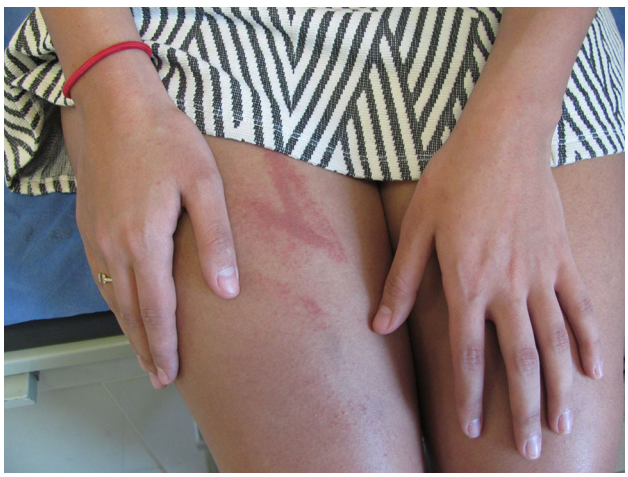

Fig. 11 Sea bather's eruption

11 Summary

Skin disease is a common complaint in returning travelers. This review has addressed the most common diseases that affect individuals returning from Western hemisphere destinations as well as from emerging destinations such as Southeast Asia. Key for diagnosis of a travel-related skin complaint is familiarity with potential disease vectors, hazardous animals, or marine life in the locale visited by the patient. Travelers should understand the risks of the natural world in their locale of travel, and patients should be counselled to take appropriate steps to avoid contact with insects or marine life that may cause harm. Thus, travelers may experience an enjoyable journey. Bon voyage!

Compliance with Ethical Standards

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