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REVIEW ARTICLE

The Infectious and Noninfectious Dermatological Consequences of Flooding: A Field Manual for the Responding Provider

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Abstract Meteorological data show that disastrous floods are increasingly frequent and more severe in recent years, perhaps due to climatic changes such as global warming. During and after a flood disaster, traumatic injuries, communicable diseases, chemical exposures, malnutrition, decreased access to care, and even mental health disorders dramatically increase, and many of these have dermatological manifestations. Numerous case reports document typical and atypical cutaneous infections, percutaneous trauma, immersion injuries, noninfectious contact exposures, exposure to wildlife, and exacerbation of underlying skin diseases after such disasters as the 2004 Asian tsunami, Hurricane Katrina in 2005, and the 2010 Pakistan floods. This review attempts to provide a basic field manual of sorts to providers who are engaged in care after a flooding event, with particular focus on the infectious consequences. Bacterial pathogens such as *Staphylococcus* and *Streptococcus* are still common causes of skin infections after floods, with atypical bacteria also greatly increased. *Vibrio vulnificus* is classically associated with exposure to saltwater or brackish water. It may present as necrotizing fasciitis with hemorrhagic bullae, and treatment consists of doxycycline or a quinolone, plus a third-generation cephalosporin and surgical debridement. Atypical mycobacterial infections typically produce indolent cutaneous infections, possibly showing sporotrichoid spread. A unique nontuberculous infection called *spam* has recently been identified in Satowan Pacific Islanders; combination antibiotic therapy is recommended. *Aeromonas* infection is typically associated with freshwater exposure and, like *Vibrio* infections, immunocompromised or cirrhotic patients are at highest risk for severe disease, such as necrotizing fasciitis and sepsis. Various antibiotics can be used to treat *Aeromonas* infections. Melioidosis is seen mainly in Southeast Asia and Australia, particularly in rice farmers, and can remain latent for many years before presenting as the host's immunocompetence wanes. It can present with a variety of skin findings or as a nonspecific febrile illness, and preferred treatment consists of ceftazidime or a carbapenem with trimethoprim/sulfamethoxazole (TMP/SMX) for 2 weeks, then continuing TMP/SMX for at least 3 months. Leptospirosis is a waterborne zoonosis that is often prevalent after heavy rains or flooding. Different forms exist, including *Fort Bragg fever*, which produces a distinctive erythematous papular rash on the shins. Doxycycline is often sufficient; however, volume and potassium repletion may be necessary if renal involvement exists. *Chromobacterium violaceum* infection may occur after open skin is exposed to stagnant or muddy water. Cultured colonies produce a unique violacein pigment, and treatment typically consists of a carbapenem. Both typical and atypical fungal infections are increased in the flooding disaster scenario, such as dermatophytosis, chromoblastomycosis, blastomycosis, and mucormycosis. Appropriate antifungals should be used. In addition, land inundated with water expands the habitat for parasites and/or vectors, thus increased

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vigilance for regional parasitic infections is necessary after a flood. Lastly, noninfectious consequences of a flooding disaster are also common and include miliaria, immersion foot syndromes, irritant and allergic contact dermatitis, traumatic wounds and animal bites, and arthropod assault, as well as exacerbation of existing skin conditions such as atopic dermatitis, psoriasis, and alopecia areata due to increased stress or nonavailability of daily medications.

Key Points

The rate and severity of severe flooding events is increasing, likely due to climatic changes caused by global warming.

This extensive review particularly focuses on the infectious consequences that increase after a flooding disaster.

Noninfectious consequences are also increased, and include miliaria, immersion foot syndromes, contact exposures, traumatic wounds and bites, arthropod assault, and exacerbation of underlying skin conditions.

1 Introduction

Over the past decade, the world has experienced several natural disasters associated with catastrophic flooding. Hurricanes, tsunamis, and unusually heavy rains have inundated enormous swaths of habitable land, thus displacing large populations and bringing injury, illness, and death (see list of recent severe floods in the online electronic supplementary material). News agencies report mainly the immediate, direct effects of the floods, such as death toll, traumatic injuries, destruction of private and public property, and disruption of social infrastructure. Additionally, interruption of sanitation and water supply creates enormous public health challenges. These effects alter—in some cases, irrevocably—nearly all aspects of a society, such as transportation, communication, food production and distribution, housing, education, and family relationships.

The rate of natural disasters seems to be increasing, with some sources citing a threefold increase from 2000 to 2009, when compared with 1980–1989 [1]. Disastrous floods, specifically, appear to be more frequent and more severe in recent years, an observation supported by data (Fig. 1) from the Dartmouth Flood Observatory and the International Disaster Database [2, 3]. This may be due to reporting bias since many recent disasters have occurred in densely populated areas and worldwide media coverage has markedly increased; however, other observers point out that climatic changes, such as global warming, may be at least partly responsible for the increase in frequency and magnitude of weather events. The Intergovernmental Panel on Climate Change (IPCC) found that each of the last three decades has been successively warmer at the earth's surface than any preceding decade since 1850, and they forecast an increase in global surface temperature of 1.5–2.0 °C by 2100 [4]. Warmer temperatures increase evapotranspiration, which generates more atmospheric moisture, followed by a rise in global average annual rainfall, thereby leading to more intense floods [5].

The Centre for Research on the Epidemiology of Disasters (CRED) defines a natural disaster as "an unforeseen and often sudden event causing great damage, destruction, and human suffering that overwhelms local capacity" [3]. According to the CRED International Disaster Database, of the 13,431 recorded natural disasters from 1900 to 2014, water-related disasters such as tsunamis (geophysical), floods (hydrological), and storms (meteorological) were the most common at 8266 (61.5 %), affecting 4.6 billion people, causing over 8.6 million deaths, and totaling almost $2 trillion in damages [3]. On an annual basis, floods are often the most frequent and deadly natural disasters, such as in 2004 when floods accounted for 7 of the top 10 disasters by death counts [6].

1.1 Cutaneous Consequences

The medical and public health literature published after disasters such as the South Asian tsunami and Hurricane Katrina shows an increasing awareness of the health effects of exposure to flood waters. The health implications include traumatic injuries, communicable diseases, exposure to pollutants, malnutrition, mental health disorders, and decreased access to care. As an acute event unfolds, the scale of trauma and property loss can quickly become matched by nontraumatic medical problems, many of which have dermatological manifestations. For example, in the days immediately following the 2004 Asian tsunami, an Indonesian hospital reported 265 dermatologic disorders among 235 patients presenting to an outpatient clinic and emergency room. The majority of conditions were new-onset and the most common category was infection/infestation (32.5 %), although closely followed by eczemas (29.8 %), and traumatic injuries to the skin (29.4 %) [7].

In the hope of providing a practical resource for the provider rapidly responding to a flooding event, this article will extensively review the various dermatologic consequences of flooding, particularly focusing on the myriad of possible infectious disease consequences. We will describe

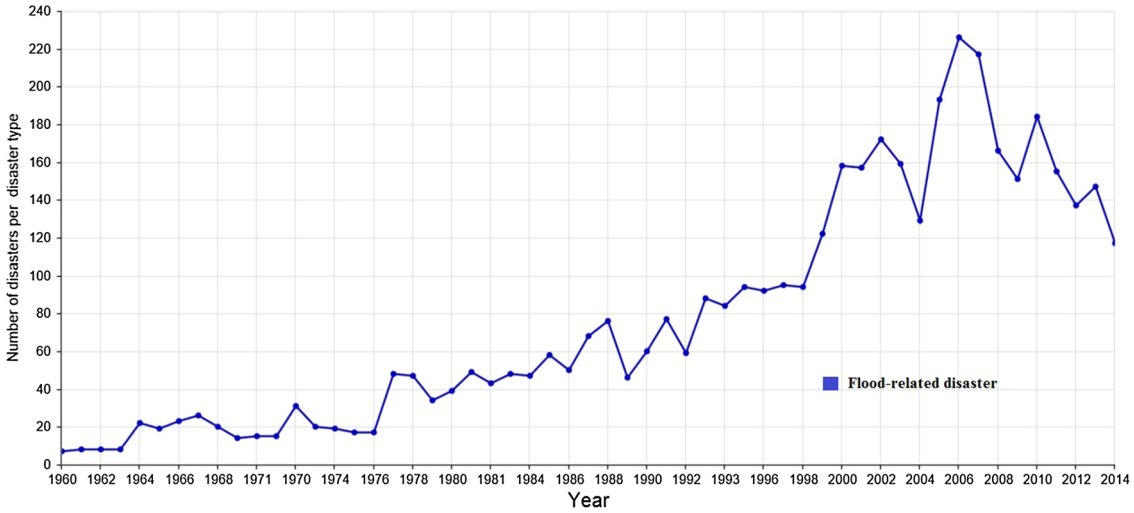

Fig. 1 Data from the International Disaster Database showing an increase in severe flooding events

the usual presentation of each condition, the distinctive clinical and epidemiologic aspects associated with flooding, and the prompt diagnostic, therapeutic, and public health measures to consider when particular pathogens are suspected or confirmed [8]. We will also discuss noninfectious consequences such as miliaria, percutaneous trauma, immersion injuries, contact dermatitis, exposure to wildlife, and psychodermatology. Nutritional dermatoses may also occur after any disaster, due to acute and/or chronic nutritional deficiencies, and thus warrant consideration during post-disaster care; however, a review is beyond the scope of this article. In the remainder of this article, we use the terms *floods* and *flooding* to indicate the inundation of land by water, irrespective of the character of that water (e.g. seawater, freshwater, or brackish water) or the duration of the water on the land. The terms *marine* and *aquatic* denote seawater and freshwater ecosystems, respectively.

1.2 Flood-Associated Dermatological Infections

Environmental water exposure increases the risk of skin infections and problems, even in recreational marine environments with no known source of domestic sewage contamination, as evidenced by a recent prospective, randomized controlled trial in healthy individuals swimming off the Florida coast [9]. However, flooding disasters can expose individuals to trauma from contact with flotsam and with sharp objects lying unseen in murky waters, and by clinging to trees or climbing structures in attempts at self-rescue. For example, an Australian Defence Force surgical team deployed to Papua New Guinea after a 1998 tsunami documented numerous large flap scalp lacerations caused by floating debris, as well as many open and closed fractures [10]. Furthermore, floodwaters are often contaminated by sewage and waste water. Subsequent traumatic wounds are easily exposed to pathogens, which likely explains why skin infections and disease were among the most frequently reported medical problems in survivors and responders after the 2004 Asian tsunami and Hurricane Katrina [7, 11]. Likewise, initial reports from the World Health Organization (WHO) during the first 30 days after the devastating 2010 Pakistan floods listed, among displaced survivors, 708,677 (19 %) cases of *skin diseases* out of almost 4 million total consultations, higher than either *acute diarrhea* (14 %) or *acute respiratory infections* (14 %) [12].

Ordinary bacterial pathogens, such as pyogenic *Staphylococcus* (including methicillin resistant *S. aureus*) and *Streptococcus*, are common causes of skin infections in flooding and should be expected and treated appropriately with topical and/or systemic antibiotics. However, other less common bacterial infections associated with marine (e.g. *Vibrio vulnificus*) or aquatic (e.g. *Aeromonas hydrophila*) habitats may also occur, many of which are naturally present in the soil, vegetation, and waters of certain geographic areas [8]. However, flooding disasters increase exposure to these pathogens, thus increasing infection rates. Examples include the relatively high number of *Vibrio* wound infections reported in Hurricane Katrina evacuees exposed to flood waters [13], and *Aeromonas* skin and soft tissue infections (SSTIs) reported in the traumatic wounds of Asian tsunami patients [14]. Multidrug-resistant organisms, polymicrobial infections, and other bacterial (e.g. *Burkholderia pseudomallei*), mycobacterial (e.g. *Mycobacterium abscessus*), and fungal (e.g. *Cladophialophora bantiana*) infections are also reported more frequently after flooding disasters [14–18].

Nonbacterial infections and exposures are similarly increased. For example, in Indonesia the majority of cutaneous infections after the 2004 Asian tsunami were superficial fungal infections [7], and arthropod bites (thought likely to be from nonscabetic mites) were commonly reported in Hurricane Katrina rescue workers [13]. Flooded conditions also increase the potential for fecal–oral transmission of protozoal (e.g. cryptosporidiosis, giardiasis), bacterial (e.g. cholera, typhoid), helminthic (e.g. intestinal helminths), and viral (e.g. hepatitis A and E, rotavirus) infections. Furthermore, vector-borne diseases may become epidemic as the inundation of water greatly expands the hospitable breeding range for the arthropod vectors. These uncommon, polymicrobial, and sometimes multidrug resistant organisms add to the difficulty in managing flood victims, and increase the need for a good understanding of the primary pathogens described below.

2 Saltwater Exposure

2.1 *Vibrio vulnificus*

Vibrio are facultatively anaerobic gram-negative motile rods found primarily in warm saltwater or brackish water. The best known member of the genus is the cholera pathogen, *V. cholerae*, which causes gastroenteritis as a foodborne illness. However, it is the noncholera *Vibrio* organisms that infect the skin. The most important of these is *V. vulnificus*, which can cause fulminant cellulitis and myositis. Oral exposure leads to primary septicemia manifested by watery diarrhea, fever, chills, nausea, vomiting, and abdominal pain, whereas wound infections may present as pustules, lymphangitis, or cellulitis. People with an immunologic or hepatic disorder who ingest contaminated seafood are at greatest risk of septicemia, and cutaneous *V. vulnificus* infection may present in these patients as necrotizing fasciitis with characteristic hemorrhagic bullae (Fig. 2a) and hypotensive shock [19, 20].

In the case of flood-associated disease, the percutaneous route of infection is most common after an open wound is exposed to saltwater. Most cases of vibriosis are sporadic, but there was a cluster of infections after large-scale exposure to the brackish floodwaters of Hurricane Katrina. The US Centers for Disease Control and Prevention (CDC) reported 24 cases of hurricane-associated *V. vulnificus* and *V. parahaemolyticus* wound infections, with six deaths [13].

A clinical suspicion of cutaneous vibriosis should have laboratory confirmation (Table 1) if possible, although the patient usually requires immediate supportive care, certainly if hypotensive shock is present. In addition to blood cultures, one should perform a gram stain and culture on aspirates and tissue samples from skin lesions. Various culture media enhance detection of *V. vulnificus*, some with polymixin B and/or colistin to inhibit nontarget organisms, but confirmations assays (e.g. DNA probes, polymerase chain reaction [PCR], etc.) are still recommended [21]. Frozen-section tissue biopsy can provide an early diagnosis of necrotizing fasciitis [22]. Furthermore, it may be necessary to begin parenteral doxycycline and ceftazidime therapy while waiting for laboratory confirmation, to provide supportive therapy in an intensive care setting, and/or to pursue urgent and aggressive surgical debridement of necrotizing fasciitis [23].

2.2 Mycobacterial Infections

Atypical mycobacteria are nontuberculous, mildly acid-fast bacilli that are ubiquitous in soil and water in various natural environments around the world [24]. They can cause indolent primary infections in healthy individuals, particularly when traumatic wounds are exposed to contaminated water, but they are often more virulent in immunocompromised patients. Under nondisaster circumstances, pulmonary disease, local nontender lymphadenitis, SSTIs, disseminated infection, foreign body-related infections (e.g. catheters), and chronic granulomatous infections are the typical clinical syndromes. In these situations, common culprits causing cutaneous disease in the US and Europe include *M. marinum*, *M. chelonae*, *M. abscessus*, and *M. fortuitum* [24–26].

Mycobacterium marinum is responsible for the so-called fish-tank granuloma, and is categorized as a slow-growing mycobacterium [16, 27]. Cutaneous infection by this organism initially manifests as a painless violaceous papule or subcutaneous nodule at the site of trauma 2–3 weeks after inoculation, and is often minor and unnoticed [24]. Constitutional symptoms are usually not present unless there is a pyogenic superinfection. Usually the lesions are solitary, but occasionally one finds multiple primary lesions or secondary sporotrichoid (lymphatic) spread (Fig. 2b).

Recently, a unique nontuberculous mycobacterial (NTM) infection has been identified, and is thought to be closely related to *M. marinum* [28]. Presenting as a chronic, verrucous skin infection, the disease is seemingly isolated to Pacific Islanders, particularly on Satowan (population approximately 650), one of the 607 islands in the Federated States of Micronesia. Satowan was acquired by the Japanese in 1914 as part of their growing empire, and during World War II, heavy Allied bombing of Satowan and surrounding islands formed numerous bomb craters. Shortly after the war's end, a skin infection, called *spam* by the locals after its presumed resemblance to the food product, began to appear. A local public health investigation in

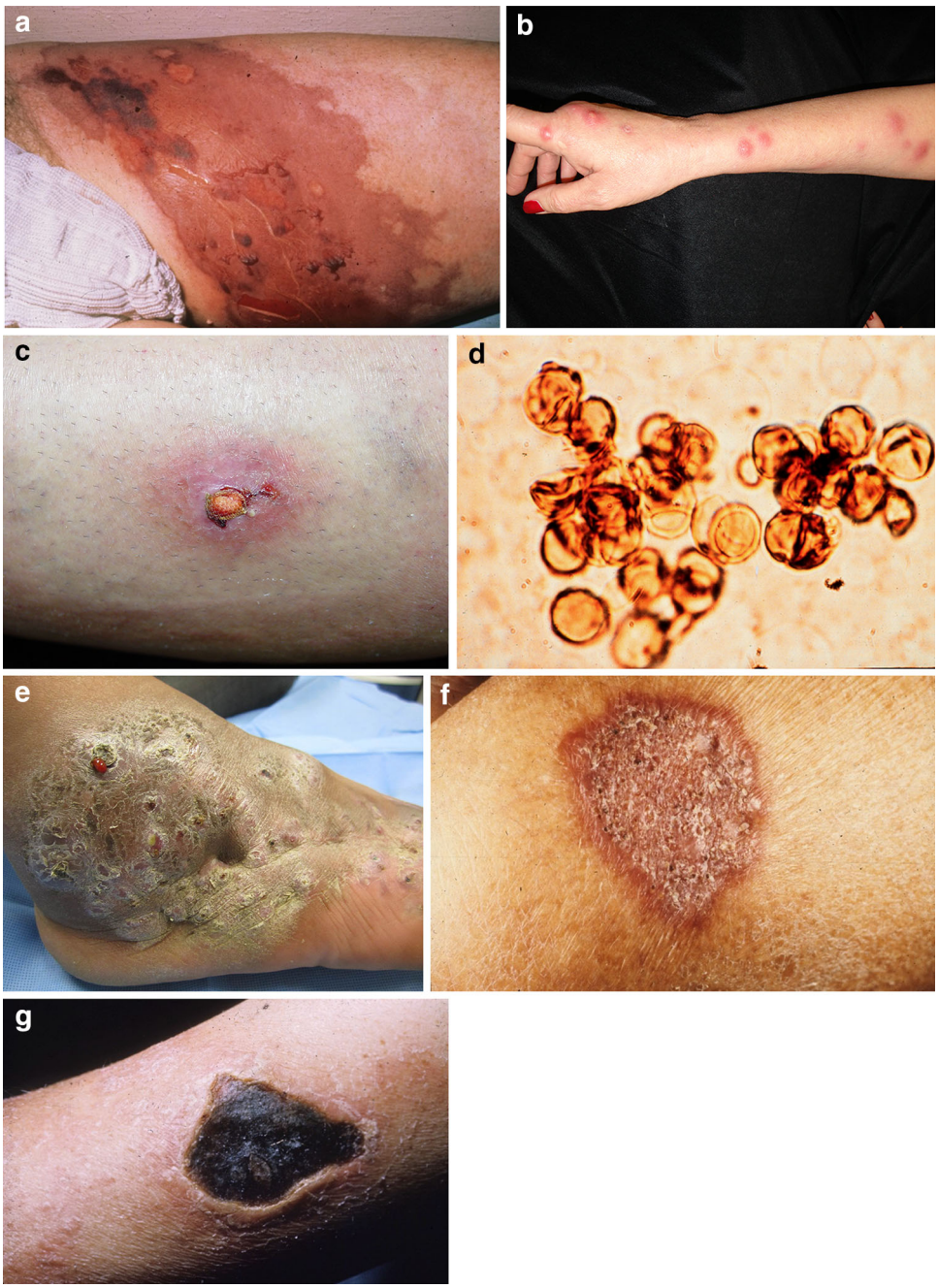

Fig. 2 **a** Hemorrhagic bullae and necrotizing fasciitis due to *Vibrio vulnificus.* **b** Sporotrichoid spread of *Mycobacterium marinum.* **c** Solitary abscess secondary to *Mycobacterium fortuitum* infection, packed with gauze. **d** Muriform (Medlar) bodies as seen in a KOH preparation. **e** Madura foot exhibiting a large plaque of draining sinuses. **f** A well-defined, solitary, verrucous lesion of blastomycosis. **g** Superficial necrosis from cutaneous mucormycosis

2004 found approximately 10 % of the Satowan population affected, but was otherwise unsuccessful in deducing the cause due to negative tissue cultures and inconclusive histopathological analysis. However, in 2006 after a 29-year-old otherwise healthy Satowan immigrant man presented to a public clinic in Portland, Oregon, with an 18-year history of enlarging verrucous plaques on his left leg and a tissue culture positive for *M. marinum*, a larger outbreak investigation was conducted [28]. Their expedition found the chronic infection (mean duration 12.5 years) to be predominantly in the legs of young men (median age 26 years). Although all mycobacterial tissue cultures and stains for mycobacterial/fungal organisms were negative, histopathological analysis found findings typical of NTM or deep fungal infections (suppurative granulomatous inflammation) and PCR detected NTM DNA with two sequenced products similar (95 and 87 % identity) to *M. marinum*. Exposure to taro farming and the stagnant, water-filled bomb craters was found to be significantly associated with infection. This case provides a good example of the relationship between water exposure and mycobacterial infections.

Table 1 Diagnostic and therapeutic highlights for flood-associated infections

Organism	Cutaneous clues	Laboratory/imaging	Microbiology	Histopathology	Culture	Advanced testing	Treatment
Vibrio vulnificus (Fig. 2a)	Hemorrhagic bulla near wound exposed to saltwater 24–48 h prior, typically lower extremity Rapidly expanding cellulitis → to necrotic ulcers, necrotizing fasciitis, and/or potentially fatal septicemia	Possible evidence of septicemia such as: CBC with smear → thrombocytopenia, ±schistocytes ↑ PT/aPTT, ↑ D-dimer, ↓ fibrinogen Evidence of end-organ damage (↑ BUN, ↑ Cr, acidotic ABG) CT > plain film may show gas in subcutaneous tissue	Gram stain → Gram-negative motile rods	Histopathology → nonpurulent bulla, hemorrhage, epidermal necrosis	Various *Vibrio*-specific media	Serology COPP PCR/RT-PCR	Doxycycline or quinolone ± third-generation cephalosporin +Surgical debridement Vaccine research in process
Nontuberculous mycobacteria (Fig. 2b, c)	Classic: slow-growing, small violaceous papulonodules with sporotrichoid spread Rapid NTM: rapid-growing nodules → abscess or cellulitis Buruli ulcer: single, firm, painless nodule slowly → wide/deep but still painless ulcer	CXR and sputum analysis if concomitant pulmonary symptoms	Acid-fast bacilli	Granulomatous infiltrate with giant cells May see acid-fast bacilli using Ziehl–Neelson stain or Auramine-Rhodamine fluorescent stain	Routine, MTBC, or Lowenstein–Jensen agars Special rapid broth systems available	Serology Liquid chromatography PCR/16S-rRNA PCR Older carbon utilization tests are slow/inadequate	Mono- or multidrug therapy based on species Macrolide, rifampin, ethambutol, minocycline, streptomycin, TMP/SMX, quinolones, INH, amikacin, etc. ±Coverage for concomitant *Staphylococcus* and *Streptococcus* infection ±Surgical debridement/excision ±Heat therapy
Aeromonas hydrophila	Rapidly forming cellulitis near wound exposed to freshwater 8–48 h prior Abscess, necrotizing fasciitis, myonecrosis, and/or septicemia ±Fishy odor	Possible evidence of septicemia (as above) CT > plain film may show gas in subcutaneous tissue	Gram stain → Gram-negative, nonsporulating facultative anaerobic rods	Diffuse suppurative infiltrate ± necrotizing fasciitis	MacConkey growth agar Cary–Blair medium for transport	Serology COPP PCR	Third-generation cephalosporin, quinolone, or carbapenem +Aminoglycoside if severe +Surgical debridement
Meloidosis (*Burkholderia pseudomallei*)	Slow-growing and persistent skin ulcer or abscess Recent or remote history of endemic exposure ±Tuberculosis-like symptoms	CXR → tuberculosis-like radiological findings	Gram stain → bipolar-stained, oxidase-positive, motile, aerobic Gram-negative rods	Diffuse suppurative infiltrate	Selective agar with gentamicin such as: Ashdown's medium Francis medium	Indirect hemagglutination antibody assay ELISA PCR	Initial: intravenous ceftazidime or carbapenem + TMP/SMX for 14 days Eradication: oral TMP/SMX ± doxycycline for at least 3 months ±Incision and drainage of abscess

Table 1 continued

Organism	Cutaneous clues	Laboratory/imaging	Microbiology	Histopathology	Culture	Advanced testing	Treatment
Leptospirosis (*Leptospira interrogans*)	Conjunctival suffusion Petechiae/purpura secondary to vascular involvement ±Widespread or pretibial erythematous papules/plaques	Hyponatremia, end-organ dysfunction (↑ LFTs, ↑ BUN/Cr)	Direct visualization of leptospires from blood, CSF, or urine Using darkfield microscopy or fluorescent antibody	Nonspecific unless special spirochete stains used (rarely performed)	Ellinghausen–McCullough–Johnson–Harris growth medium	Lepto Dipstick (IgM antibody assay) Microscopic agglutination test (gold standard) Latex agglutination Indirect immunofluorescence ELISA PCR/16S-rRNA PCR	Doxycycline, amoxicillin, or ceftriaxone Jarisch–Herxheimer reaction may occur ±Volume and potassium repletion if renal involvement ±Dialysis (rare)
Chromobacterium violaceum	Cellulitis Abscess Ecthyma gangrenosum-like lesion Septicemia	Possible evidence of septicemia (as above)	Gram stain → long, facultatively anaerobic, Gram-negative bacilli	Nonspecific	Nutrient agar → unique dark violet-metallic sheen (violacein)	None pertinent	Carbapenems, chloramphenicol + gentamycin, or quinolones ± Incision and drainage of abscess
Chromoblastomycosis (*Fonsecaea pedrosoi*, and others) (Fig. 2d)	Erythematous nodule → friable, expanding verrucous or granulomatous plaque	Radiographic imaging may be useful for determining underlying soft-tissue/bony involvement	KOH of skin scraping → muriform cells (aka sclerotic, coppery penny, or Medlar bodies)	Pseudoepitheliomatous hyperplasia, intraepidermal abscesses, muriform cells among mixed granulomatous/suppurative infiltrate May see pigmented hyphae Consider PAS or GMS	Standard mycologic media such as: Mycosel/ Sabouraud agar	PCR	Itraconazole or terbinafine ±Cryotherapy, excision, heat therapy
Blastomycosis (*Blastomyces dermatitidis*) (Fig. 2f)	Verrucous plaque with expanding 'stadium-like' borders Concomitant pulmonary and/or bone involvement possible	CXR → patchy infiltrates or normal-appearing	KOH or calcofluor of skin scraping/ sputum → broad-based budding yeast with thick double-contoured walls	Pseudoepitheliomatous hyperplasia, intraepidermal abscesses, Broad-based, single budding yeast among mixed granulomatous/suppurative infiltrate Consider PAS or GMS	Blood/Sabouraud agar	Serological tests have high cross-reactivity with *Histoplasma* Immunodiffusion EIA Radioimmunoassay PCR	Itraconazole, fluconazole, or amphotericin B ± Surgical excision
Mucormycosis (*Mucor* spp., *Rhizopus* spp., *Absidia* spp.) (Fig. 2g)	Cellulitis, facial edema Superficial-appearing, painful nodule Rapidly → ecthyma gangrenosum-like necrosis with deep involvement	Possible evidence of septicemia (as above)	KOH of dermal scrapings from base of necrotic bulla → broad, ribbon-like, nonseptate hyphae	Rapid frozen or standard H&E → broad, ribbon-like, nonseptate, and possibly angioinvasive hyphae with 90° branching in background suppurative/necrotic infiltrate Consider PAS or GMS	Blood/Sabouraud agar	PCR	Liposomal amphotericin B ± Azoles ± Aggressive debridement

ABG arterial blood gas, *aPTT* activated partial thromboplastin time, *BUN* blood urea nitrogen, *CBC* complete blood count, *COPP* colony overlay procedure for peptidases, *Cr* creatinine, *CSF* cerebrospinal fluid, *CT* computerized tomography, *CXR* chest x-ray, *EIA* enzyme immunoassay, *ELISA* enzyme-linked immunosorbent assay, *GMS* Grocott–Gomori's methenamine silver, *H&E* hematoxylin and eosin, *IgM* immunoglobulin M, *INH* isoniazid, *KOH* potassium hydroxide, *LFTs* liver function tests, *MTBC Mycobacterium tuberculosis* complex, *NTM* nontuberculous mycobacteria, *PAS* periodic acid–Schiff, *PCR* polymerase chain reaction, *PT* prothrombin time, *RT-PCR* real-time PCR, *TMP/SMX* trimethoprim-sulfamethoxazole

↑ Increased

↓ Decreased

In contrast to the slow growing *M. marinum* organism, *M. fortuitum*, *M. chelonae*, and *M. abscessus* are rapidly growing, pathogenic mycobacteria (RGM, formerly known as Runyon category IV) that were previously grouped in the *M. fortuitum* complex but are now each recognized as distinct species [29]. Several reports of patients diagnosed with these atypical mycobacterial infections followed the 2004 Asian tsunami, and they often present as a rapidly worsening solitary abscess (Fig. 2c) [16, 30]. In one report of 15 tsunami survivors who suffered traumatic injuries and subsequently developed late-onset SSTIs from RGMs, mycobacterial isolates included seven cases of *M. abscessus,* six cases of *M. fortuitum,* and one case each of *M. peregrinum* and *M. mageritense* [31]. The *M. abscessus* infections were more severe, causing multiple abscesses and requiring longer courses of antibiotics. Cases of polymicrobial NTM infections were also reported after the tsunami and included bacteria and fungi such as MRSA, *B. pseudomallei, Actinomyces neuii*, or *C. bantiana* [31, 32].

Another slow-growing mycobacterium, *M. ulcerans*, is the pathogen that causes Buruli ulcer. It is considered endemic in tropical regions of West Africa (particularly Benin, Cote d'Ivoire, and Ghana) and Asia (particularly India, Indonesia, and Papua New Guinea), and also in certain temperate regions such as southeastern Australia, where it is known as the Bairnsdale Ulcer [33]. This toxin-mediated (mycolactone), extracellular, necrotizing mycobacterial infection, found usually in immunocompetent hosts, is regarded by the WHO as an emerging infection. The environmental source is not specifically known but epidemiologic evidence links the disease with patterns of exposure to natural bodies of freshwater [34]. For example, epidemiologic analysis of a spike in the number of Buruli ulcer cases in the Kasongo Territory of the Democratic Republic of Congo found that a recent civil conflict and flooding were the most likely causes of the re-emergence of the disease [35]. Similarly, a spray irrigation system was found to be the source of a large outbreak near Melbourne, Australia, in 1997 [36]. Transmission of *M. ulcerans* is not yet fully understood; however, some evidence has implicated mosquitoes and aquatic bugs (Belostomatidae and Naucoridae) in a complex transmission process [37–39].

Laboratory confirmation (Table 1) of cutaneous mycobacterial infections is best achieved by both culture and histopathological examination of tissue biopsy specimens as the combination is more sensitive than culture alone [8]. Direct smear analysis looking for acid-fast bacilli provides easy and rapid assessment but has low sensitivity. The use of PCR should provide results more rapidly, with high sensitivity and specificity; however, it does not provide antimicrobial susceptibility data necessary to direct treatment and the test is not yet widely available [40, 41].

Treatment of NTM infections is typically more difficult because of their natural resistance to many antibiotics [32]. As a result, prolonged or relapsing infections are common. Concomitant infections with pyogenic bacteria slow the healing process and should be treated appropriately. Even slow-growing *M. marinum* cutaneous infections rarely resolve without pharmacotherapy and may require debridement or excision [42]. Although there is no consensus on the type and duration of antimicrobial treatment, monotherapy with clarithromycin has been successfully used in *M. marinum*, *M. abscessus*, and *M. fortuitum* infections [26, 31]. Nevertheless, combination therapy with two or more antibiotics is recommended to reduce the risk of developing drug resistance. Surgical debridement prior to antibiotic therapy has been found to shorten healing time by 50 % [31]. The Satowan NTM infection has proven very difficult to treat, and empirical combination therapy is recommended with rifampicin plus two of the following: ethambutol, trimethoprim/sulfamethoxazole (TMP/SMX), clarithromycin, or minocycline [28]. If cost or access to proper medical care is otherwise prohibitive, then doxycycline alone has shown some benefit.

For Buruli ulcers, surgical excision or debridement has long been the standard of care, but antibiotic therapy alone with rifampicin and streptomycin for 8 weeks is now widely accepted and recommended by the WHO [43]. The efficacy of antibiotics, particularly in early, limited disease has been supported in various studies to include a 2010 randomized controlled trial that found similar efficacy in patients who switched from streptomycin injections to oral clarithromycin after 4 weeks [44, 45]. Unfortunately, streptomycin supplies are low worldwide and thus a large post-disaster outbreak of Buruli ulcer would likely deplete local stocks.

3 Freshwater Exposure

3.1 *Aeromonas hydrophila*

A. hydrophila shares many environmental attributes with the *Vibrio* species but differs chiefly in its habitat, which is primarily freshwater and, rarely, brackish water. *A. hydrophila* is an oxidase-positive, facultatively anaerobic gram-negative bacillus and is a natural pathogen of fish, amphibians, and other aquatic animals [46]. Human disease is often acquired orally and most often presents as gastroenteritis [47] but the organism has been increasingly implicated in SSTIs [48, 49]. Potential noncatastrophic sources of cutaneous disease include exposure to

contaminated fishing tanks [50], swimming pools [51], tap water [52, 53], medical leeches [54], and even mud football [55].

As with *Vibrio*, healthy patients typically sustain minor localized infections while immunocompromised or cirrhotic patients bear the highest risk of severe SSTIs and septicemia. In SSTIs, *A. hydrophila* typically presents as cellulitis after an open-wound is exposed to contaminated freshwater. The infected wound may generate a foul or fishy odor and is often followed by abscess formation [56, 57]. Necrotizing fasciitis or myonecrosis may occur, clinically presenting with rapid onset and swift progression to severe pain, swelling, serosanguineous bullae, gas gangrene, and sepsis [20, 58]. Although *Aeromonas*-induced necrotizing fasciitis and sepsis are more common in immunocompromised patients, they may also present in otherwise healthy trauma patients [59].

A large number of *Aeromonas* infections were reported after the 2004 Asian tsunami. Among 777 patients transferred from southern Thailand to four Bangkok hospitals, 515 (66.3 %) had SSTIs and *Aeromonas* was the most common isolated organism, accounting for 145 (22.6 %) of 641 bacterial isolates [14]. Similarly, the Bangkok Hospital in Phuket, Thailand, reported that approximately 25 % of the isolates from hundreds of patients with prolonged water exposure contained *Aeromonas* [60]. This was likely due to the fact that after the saltwater tsunami wave flooded the region, freshwater inundation from surrounding reservoirs quickly followed. During Hurricane Katrina, storm surge caused the freshwater Lake Pontchartrain to overflow and inundate 80 % of New Orleans. Subsequent water sampling found high concentrations of *Aeromonas* within the city to include the environs of the New Orleans Superdome, where many displaced residents were sheltered, and a local hospital [61]. Gram stain with culture should be used to establish diagnosis (Table 1). If possible, bacterial culture should be obtained. After transporting specimens in Cary–Blair medium, most noninhibitory enteric isolation media (e.g. MacConkey agar) will produce adequate growth [49]. In addition, a simple, low cost, fluorogenic method termed the colony overlay procedure for peptidases (COPP) assay has been developed that can rapidly detect and quantify both *Vibrio* and *Aeromonas* from water samples collected after water-related disasters, although it is not yet widely available [62]. However, various commercial identification systems should be used with caution as they have been frequently shown to either misidentify the organism or to mislabel *Aeromonas* strains as *Vibrio* [49].

In severe, necrotizing cases of *A. hydrophila* SSTIs, immediate surgical debridement is paramount, followed by antibiotics. However, this may prove difficult in a mass casualty situation in which adequate debridement may be impossible due to the sheer number of patients. *A. hydrophila* produces β-lactamase and is therefore often resistant to penicillins but is generally susceptible to third-generation cephalosporins, fluoroquinolones, and aminoglycosides [49].

3.2 Melioidosis

Melioidosis is caused by *B. pseudomallei*, a gram-negative, facultative intracellular, bipolar-staining, aerobic, and motile bacillus found in freshwaters and damp soils of humid tropics. Infecting humans and animals, *B. pseudomallei* causes disease mainly in Southeast Asia and coastal areas of Australia's Northern Territory. The disease is acquired after exposure to contaminated soil or water, either directly through open skin or through inhalation of aerosolized dust. In Thailand, melioidosis is typically an occupational disease of rice farmers. Serologic surveys in endemic areas show that 5–20 % of rural inhabitants have antibodies against *B. pseudomallei*, presumably due to prior asymptomatic infections in most cases [63, 64]. *B. pseudomallei* is infectious when aerosolized, therefore the CDC regards it as a Category B bioweapon due to its potential use on humans or livestock [65].

Melioidosis typically presents as an indolent, chronic infection with lung, skin, bone, joint, liver, or spleen involvement but can present acutely with suppurative skin lesions, pneumonia, or septicemia, particularly after a near-drowning event. Immunocompromised patients are at increased risk of infection, such as those with diabetes or chronic renal disease. Melioidosis can also remain latent for many years before presenting as a persistent, unexplained fever when a previously infected but asymptomatic patient's immunocompetence wanes. Examples include several cases in American veterans of the Vietnam conflict whose disease appeared decades after extensive immersion in rice paddies [66]. Latency results from the unique ability of *B. pseudomallei* to invade various cells (mainly polymorphonuclear leukocytes and macrophages), escape endocytic vacuolization, and commandeer actin polymerization, thus enabling cell movement and cell-to-cell spread [8].

Approximately half of all melioidosis cases involve the lungs as either a primary inhalational disease or secondarily associated with bacteremic disease. Skin involvement is varied and nonspecific but can present as pustules, ecthyma gangrenosum, or necrotizing fasciitis in disseminated disease, or as subcutaneous abscesses and draining sinuses in chronic infections [67]. Some patients, particularly children, may present with acute suppurative parotitis [68]. The acute, primary pneumonic form abruptly starts with fever, chills, cough, dyspnea, and chest pain. In one-half of septic patients, no source for bacteremia can be

identified but many will have minor abrasions on their feet as well as water exposure [69]. Understandably then, this disease has an increased incidence during rainy or monsoon seasons and floods, whether seasonal or disastrous. In addition to the numerous reported cases of severe post-immersion, aspiration pneumonic melioidosis after the 2004 Asian tsunami [70–72], many cases of *B. pseudomallei* cutaneous wound infections were also reported [73–75].

Laboratory identification (Table 1) of *B. pseudomallei* is critical since acute melioidosis can present as a nonspecific, nonlocalizing, febrile illness that resembles typhoid, leptospirosis, malaria, or septicemia. Chronic melioidosis can resemble pulmonary tuberculosis or a bacterial lung abscess [76]. Bacterial wound cultures should be obtained but require the use of special selective media and may take 4–5 days to return positive [77]. Enrichment broths may accelerate the process, but other nontarget organisms will also flourish. Confirmation assays such as PCR are therefore recommended but are not widely available. Preferred treatment includes parenteral ceftazidime or a carbapenem with or without TMP/SMX or doxycycline for 14 days. Patients should then be switched to oral antibiotics, usually TMP/SMX or amoxicillin/clavulanic acid, for up to 20 weeks in severe infections, to reduce the risk of relapse [78]. Surgical drainage of abscesses should also be performed, but only after antibiotic therapy has been initiated.

3.3 Leptospirosis

Leptospirosis is a waterborne zoonosis caused by the spirochete *Leptospira interrogans* (and its dozens of serovariants), and is found worldwide. Rodents are the most important reservoirs, and infection typically arises after broken skin is exposed to freshwater or soil contaminated with urine from infected animals. *L. interrogans* thrives in warm freshwater, thus leptospirosis has its highest incidence in tropical areas. Barefoot farmers who use large mammals such as oxen to till rice fields are especially vulnerable. In the developing world, leptospirosis should be considered in those with undifferentiated fever, along with dengue, malaria, and typhoid. Outbreaks have been reported after heavy rains or floods caused sewers to overflow [79, 80]. For example, thousands of cases of leptospirosis with hundreds of subsequent deaths were reported in the Philippines after a series of typhoons caused disastrous floods in late 2009 [81]. Similar outbreaks following flooding have been reported across the world, including India, Iowa, Argentina, and Mexico [82–85]. An observational study of children in Mumbai, India, presenting with an acute febrile illness after an extremely heavy rainfall in 2005 found three clinical features significantly associated with enzyme-linked immunosorbent assay (ELISA)-confirmed leptospirosis: flood water contact, myalgias, and conjunctival suffusion [86].

Many human infections are asymptomatic and self-limited, but leptospirosis is commonly associated with two distinctive clinical forms: a mild, self-limited anicteric form, or a severe icteric form called Weil's disease. Both forms typically have two phases (septicemic/leptospiremic and immune/leptospiruric), although the phases can appear concurrently in the icteric form [87]. After an incubation period of up to 2 weeks, both the anicteric and icteric forms present with an acute leptospiremic phase that begins abruptly with headaches, fever, chills, nausea, vomiting, abdominal pain, and myalgias that can last for 5–7 days [88]. In Weil's disease, symptoms worsen and potentially progress to aseptic meningitis, uveitis, pulmonary hemorrhage, and multiorgan involvement that may cause liver or kidney failure. In the anicteric form, several relatively asymptomatic days may follow the first phase, bridging the illness to the delayed immune phase consisting of a low-grade fever and mild constitutional symptoms [87].

Physical examination in the acute phase of both forms may reveal abdominal tenderness, hepatosplenomegaly, jaundice, edema, hemorrhage, or the classic conjunctival suffusion that resembles nonexudative conjunctivitis [87]. The latter sign has been reported in 16–58 % of patients [88]. Dermatological manifestations are uncommon but include nonspecific macules, papules, urticaria, and petechiae. The *L. interrogans* serovariant *autumnalis* has been reported to produce a distinctive rash consisting of slightly raised, 1–5 cm, tender, erythematous plaques on the shins, subsiding after 4–7 days, termed *pretibial* or *Fort Bragg fever* [89, 90].

Diagnosis is confirmed (Table 1) with direct visualization of leptospires under darkfield microscopy (rarely used), serology (most common method), or PCR (ideal for early detection) [87]. Antibiotic therapy should be initiated as early as possible, and typically consists of oral doxycycline or amoxicillin in mild disease, and intravenous penicillin or ceftriaxone in severe disease. Chemoprophylaxis with doxycycline 200 mg weekly has been attempted, although a 2009 systematic review found no conclusive benefit in reducing seroconversion or clinical symptoms [91]. Sanitary measures, water purification, and rodent control are the most effective methods of prevention.

3.4 *Chromobacterium violaceum*

Chromobacterium violaceum is a facultatively anaerobic, gram-negative coccobacillus, commonly found on decaying organic matter in tropical and subtropical freshwater and soil [92]. It is generally not pathogenic, but individuals with chronic granulomatous disease or G6PD deficiency may be particularly susceptible to infection [93, 94].

Cutaneous disease typically results from the exposure of minor breaks in the skin to a contaminated source such as stagnant or muddy water. Cutaneous manifestations include localized cellulitis with pustules and nodules at the site of percutaneous inoculation or ecthyma gangrenosum-like lesions after hematogenous dissemination [92]. The infection can rapidly progress to fulminant bacteremia with multiple organ involvement and sepsis [95]. Visceral abscesses can rapidly develop in the liver, lung, and spleen. In a review of 25 case reports, Sirinavin et al. found the overall case fatality rate to be 48 %, with the fatal cases all occurring in patients with bacteremia or disseminated disease, and most not receiving effective antibiotic therapy [93]. An earlier review of 22 US cases by Ponte and Jenkins found a mortality rate of 64 % [96].

Diagnosis of *C. violaceum* infection is by blood culture (Table 1). The bacillus grows well on blood and MacConkey's agars, typically producing violacein pigment that gives the colonies a distinctive metallic-purple color. Nonpigmenting colonies rarely occur and may subsequently be misidentified as *Vibrio*, *Aeromonas,* or *B. pseudomallei* based on gram stain alone [97]. Additionally, because *C. violaceum* is ubiquitous in nature and not a common pathogen, it may be dismissed as a contaminant, thus delaying proper treatment and possibly leading to fatal consequences. Biopsy specimens show a necrotizing vasculitis, bacilli in the vessels, and minimal neutrophilic infiltrate. Treatment consists of surgical drainage of cutaneous and visceral abscesses, along with several weeks of parenteral antibiotic therapy. *C. violaceum* is usually resistant to penicillins and other β-lactam antibiotics, but carbapenems, chloramphenicol with gentamicin, or fluoroquinolones are typically effective and are thus appropriate initial treatments [98]. Early and aggressive antimicrobial therapy is pivotal in reducing the high fatality rate.

4 Fungal Infections

4.1 Chromoblastomycosis

Chromoblastomycosis, also known as chromomycosis, is a chronic fungal infection of the skin and subcutaneous tissues caused by one of several species of dark-walled, dematiaceous (pigmented) fungi [8]. Infections are typically caused by traumatic inoculation of the skin through thorn or splinter wounds from decaying plant matter. *Fonsecaea pedrosoi* is by far the most common etiological agent, responsible for greater than 90 % of infections in some studies [99]. Other organisms include *Cladophialophora carrionii*, *Phialophora verrucosa*, *Fonsecaea compactum*, *Fonsecaea monophora*, *Rhinocladiella aquaspersa,* and *Wangiella dermatitidis* [100]. Chromoblastomycosis typically occurs in tropical and subtropical regions, and cases were reported in aid workers in flooded regions of Thailand after the 2004 Asian tsunami [101].

Incubation periods vary but most cases slowly progress over several years. Infections tend to spread laterally rather than directly invading muscle or bone [102]. Clinical manifestations vary from nodular lesions to red, scaly plaques to irregular, hyperkeratotic verrucous lesions. Diagnosis can therefore be difficult as the infection may resemble other conditions such as lobomycosis, sporotrichosis, squamous cell carcinoma, podoconiosis, *M. marinum* infection, leprosy, diffuse cutaneous leishmaniasis, and tuberculosis verrucosa cutis [102, 103]. A potassium hydroxide (KOH) wet mount of skin scrapings may assist in diagnosis by revealing round, brown structures with a single or double septum and a thick cell wall. These organisms are called muriform or sclerotic cells and are also known as Medlar bodies or *copper pennies* (Fig. 2d). Tissue biopsy showing muriform cells or tissue culture may be required for definitive diagnosis (Table 1), and quantitative serologic tests exist for *F. pedrosoi* and *C. carrionii* that correlate with the extent of lesions [104]. Identifying the causal agent is advantageous because *C. carrionii* has been found to typically be more sensitive to pharmacotherapy than *F. pedrosoi* [105].

Many soil-based fungi (both dematiaceous and nondematiaceous), such as *Pseudallescheria boydii*, can also cause atypical cutaneous infections with a wide range of presentations, including the classic eumycetoma or Madura foot (Fig. 2e), sometimes many years after a flooding disaster [30, 106, 107]. An exhaustive list of organisms is beyond the scope of this review, however any nonhealing ulcer or verrucous traumatic wound in a patient exposed to soil and mud after a flooding disaster should prompt investigation for one of these subcutaneous mycoses.

Antifungals are the cornerstone of treatment, preferably with itraconazole or terbinafine, and may be combined with local cryotherapy, surgical excision, or heat therapy to improve efficacy [102, 108]. Electrodessication and curettage may spread disease and should thus be avoided. Treatment should be continued until clinical resolution of lesions, sometimes taking several months. Some newer antifungals (e.g. voriconazole, posaconazole) have shown promise in treating refractory disease [109, 110].

4.2 Blastomycosis

Blastomycosis is a fungal infection caused by *Blastomyces dermatitidis* and most often causes pulmonary disease ranging from asymptomatic infection to acute pneumonitis, similar to acute histoplasmosis, with productive cough,

fever, chills, night sweats, chest pain, shortness of breath, and occasionally hemoptysis [111]. However, cutaneous disease is its second most common manifestation and presents as a granulomatous, verrucous lesion (Fig. 2f) that can resemble squamous cell carcinoma, or as ulcerative lesions [112]. Cases are most often reported in North America, particularly the Mississippi and Ohio River valleys, and diagnostic evaluation of cutaneous disease is outlined in Table 1 [112].

Although blastomycosis is not typically associated with flooding or water exposure, moist and humid environments promote the growth of the natural, soil-growing organism, as discovered during analysis of two outbreaks along rivers in Wisconsin [113]. Further evidence exists in the case report of an otherwise healthy 26-year-old man working on a farm hit by Hurricane Katrina who presented 3 months after the hurricane with a history of worsening pneumonia and cognitive decline despite empiric antibiotics, as well as multiple, spreading, verrucous and ulcerated lesions of the face with irregular, raised borders and purulent discharge [114]. Pulmonary blastomycosis with cutaneous and CNS involvement was diagnosed after skin biopsy revealed *B. dermatitidis*. Itraconazole is the preferred treatment, however amphotericin B should be used as initial treatment for severe disease, CNS involvement, and the immunocompromised [112].

4.3 Mucormycosis

Mucormycosis is an infection caused by fungi of the genera *Mucor*, *Rhizopus*, or *Absidia*, which are commonly found in the environment. These pathogens are most known for sinus, brain, or pulmonary infections in the immunocompromised or in those with uncontrolled diabetes mellitus, but they can also present as gastrointestinal or cutaneous disease [115]. Skin infections are typically seen in those with traumatic injury to the skin and exposure to the environment, and thus may present as severe, polymicrobial infections or necrotizing fasciitis in disaster situations [116]. For example, after a 1985 volcanic eruption in Colombia caused melting of the mountain's icecap, subsequent massive flows of volcanic mud (lahar) immersed many victims and contaminated traumatic soft tissue injuries. Four hospitals in Bogotá reported 38 victims with necrotizing fasciitis of their soft tissue injuries, 8 of whom had more severe and rapidly progressing lesions and were found to have concomitant mucormycosis [117]. Mucormycosis causing necrotizing fasciitis was also reported in victims of the 2004 Asia tsunami [118, 119].

Cutaneous disease typically presents as an infection of a simple abrasion or traumatic wound that persists despite systemic, broad-spectrum antibacterial agents. If left untreated, the infection may then rapidly progress to severe, extensive tissue necrosis (Fig. 2g). Vascular invasion may lead to widespread hematogenous dissemination. Prompt diagnosis is paramount as mortality greatly increases with delayed diagnosis, even in immunocompetent individuals. For example, overall mortality among the 38 patients who presented with necrotizing fasciitis after the Colombian volcanic eruption was 47.7 %, whereas a mortality rate of 80 % was observed in the 8 patients with confirmed mucormycosis [117]. Rapid diagnosis is best performed with tissue biopsy and histopathological analysis, often on frozen section material, looking for fungal elements (Table 1). Treatment consists of aggressive surgical debridement and prompt initiation of systemic antifungal agents, typically the liposomal form of amphotericin B. In nondisseminated cutaneous mucormycosis, such treatment can greatly decrease mortality [120]. Although costly, the lipid formulations of amphotericin B are the primary therapy for mucormycosis, although certain azoles, echinocandins, cytokines, and hyperbaric oxygen may provide useful combination or adjunctive therapy in select patients [121].

4.4 Dermatophytosis

Dermatophyte infections are common in everyday practice and are thus also common infections during flooding disasters, particularly due to increased environmental exposure. For example, 10 days after the 2004 Asian tsunami, two dermatologists spent 3 weeks in an outpatient clinic and emergency room of an Indonesian military hospital, and reported 265 skin problems in 235 patients. Superficial fungal infections (particularly tinea corporis) accounted for the majority (44 %) of problems classified as *infections/infestations* [7]. Although it is unknown whether some of these fungal infections were present before the disaster, the tsunami likely increased the risk of infection because many barefoot and ill-clothed survivors were exposed to hot, humid, and water-logged conditions.

Dermatophytes infect keratin-bearing tissues, such as epidermis, hair, and nails. Infections typically present as pruritic, erythematous, scaling patches/plaques with centrifugal spread. The first step to confirm the diagnosis is a KOH wet preparation of skin scrapings looking for fungal elements, although fungal culture or tissue biopsy may also be performed. However, the latter is impractical in the disaster situation as culture results can take a month or more to return positive. A dermatophyte test medium (DTM) culture can provide quicker results. Treatment generally consists of topical antifungals, such as topical terbinafine, clotrimazole, or naftifine, although systemic medications may be indicated in extensive disease or in the immunocompromised. Topical nystatin should be avoided as it is only useful in candidal infections. In disaster

settings, the updated Interagency Emergency Health Kit 2011 published by the WHO recommends stocking miconazole cream or tablets instead of the formerly recommended gentian violet [122, 123]. Nevertheless, gentian violet or Whitfield's solution (benzoic acid and salicylic acid) may be the only remedies available in a disaster situation.

5 Parasitic Infections

After a flood, parasitic infections are often increased and can arise from cutaneous contact, ingestion, or vector-borne transmission. For example, in 1983, heavy snowmelt and subsequent flooding in Tooele, Utah, likely contaminated public drinking water with fecal–oral pathogens, thus causing an outbreak of diarrheal illness from *Giardia lamblia* infection [124]. A similar association between cryptosporidiosis and flooding has also been reported [125]. Inundated lands may expand habitats for parasites or their vectors, as demonstrated by the colonization of abandoned swimming pools by larval mosquitoes after Hurricane Katrina [126]. In 2008, Chinese researchers compiled a retrospective analysis of 22 years of data and found that rates of schistosomiasis were, on average, 2.8 times higher in the Yangtze River Valley during years characterized by flooding compared with years with normal water levels [127]. This was thought likely related to the dramatic increase in size (2.6–2.7 times normal) of potential habitat for *Oncomelania hupensis*, a small tropical freshwater snail and the intermediate host of *Schistosoma japonicum*. Similarly, after the Mozambique floods in 2000, a retrospective epidemiological analysis reported a large spike in malaria cases in the weeks following the disaster [128]. Concerned that a similar event would occur immediately after the Asian tsunami in 2004, the WHO warned of a possible increase in cases of malaria [129]. However, epidemiological data show that in Sri Lanka and most other flooded areas, malaria rates did not increase [130]. This may have been due to the large relief effort that prevented outbreaks, and also possibly because freshwater anopheline breeding sites were contaminated by the salty waters of the tsunami. Nevertheless, outbreaks of mosquito-related infections and their corresponding dermatological manifestations may occur after significant flooding, such as Dengue fever (*white islands in a sea of red*, petechiae), Chikungunya fever (morbilliform eruption with prominent postinflammatory hyperpigmentation), or West Nile fever.

Altered living situations may also play a role. Specifically, the prevalence of highly communicable parasitic diseases such as scabies would be expected to increase if large groups of refugees began to concentrate in close quarters after a disaster, as was documented during a 1997 US military humanitarian mission to Haiti, and more recently after the 2010 Pakistan floods [131, 132]. A scabies outbreak was similarly expected after the 2011 Giant East Japan Earthquake and tsunami, but was not seen, exemplifying the complex nature of parasitic outbreaks [133]. Nevertheless, floods disturb the normal ecosystem and often lead to increased exposure to parasitic organisms. Many of these parasitic infections have cutaneous manifestations, as summarized in Table 2, and should be considered in the differential of flooding victims [8, 134, 135]. Examples of scabies, cutaneous and mucocutaneous leishmaniasis, onchocerciasis, and cutaneous larva migrans are shown in Fig. 3.

6 Environment

6.1 Miliaria

We must not forget the noninfectious dermatological consequences of floods. For example, in hot, humid, and tropical climates, some patients, particularly children and the elderly, may develop miliaria. Relief workers are not immune, as demonstrated by reports from the CDC of miliaria in aid workers after Hurricane Katrina [13]. This eruption is due to sweat accumulation within eccrine sweat ducts after prolonged perspiration, with the resulting keratin obstruction in the stratum corneum manifesting as small, pruritic vesicles (miliaria crystallina) [136]. If the intraepidermal level of obstruction is deeper, an inflammatory reaction may occur, presenting as erythematous, nonfollicular, and nonconfluent vesicles and papules (Fig. 4a). This latter form, miliaria rubra, is also known as *heat rash* or *prickly heat*, and can be accompanied by intense pruritus or a stinging sensation. Miliaria rubra with pustules is termed miliaria pustulosa, and deeper obstruction at or below the dermoepidermal junction causes firm, larger papules, and is termed miliaria profunda. Treatment involves cooling the patient to reduce sweating, preferably by removal from the hot, humid environment. Symptomatic treatment of the pruritus with calamine lotion, topical corticosteroids, or oral antihistamines can be provided if needed.

6.2 Immersion Foot Syndromes

Another noninfectious consequence of prolonged exposure to wet environments is *immersion foot*, also known as *trench foot* during World War I as it was often seen in soldiers wearing wet boots and socks for long periods of time while confined to cold, damp trenches. The same process can also occur in warmer climates, particularly in

Table 2 Selected arthropod, protozoal, and helminthic parasitic infections

Infection	Epidemiology	Signs/symptoms	Workup/treatment
Scabies (*Sarcoptes scabiei*) [Fig. 3a]	Common in crowded living conditions Direct skin-to-skin transmission Global prevalence	Pruritic excoriated erythematous papules, often accentuated along areolae, umbilicus, waist band, groin/genitals, volar wrists, interdigital webspace of hands/feet, and plantar surfaces May see classic linear burrows ending at a small black dot/chevron	KOH or mineral-oil preparation of skin scrapings to look for mite, eggs, or fecal pellets (scybala) Histopathologic examination of tissue High-temperature wash of all bedding, or trash bag isolation of all linens for 7 days *Tx: permethrin, ivermectin, precipitated sulfur (neonates), benzoyl benzoate*
Cutaneous leishmaniasis [Fig. 3b]	Reservoir: canines, rodents, small mammals Vector: *Phlebotomus* or *Lutzomyia* sandfly Sandflies are smaller than mosquitoes, thus standard bed nets may be inadequate Old-World CL (Eastern hemisphere) New-World CL (Western hemisphere)	Classic limited form: painless solitary papule → dusky, violaceous nodule → wet ulcer with indurated, rolled borders Diffuse cutaneous form Be vigilant for visceral leishmaniasis: fever, weight loss, pancytopenia, hepatomegaly, more common in Old World	Histopathologic examination of tissue, slit-skin smear with Giemsa stain, culture, serology, PCR Typically self-limited, results in atrophic scar, but may have bacterial superinfection or progress to MCL *Tx: Observation only, cryotherapy, intralesional pentavalent antimony, topical paromomycin, or systemic therapy (see MCL)*
Mucocutaneous leishmaniasis 'espundia' [Fig. 3c]	Rare sequelae of CL, occurs in <5 % of CL Vector: *Phlebotomus* or *Lutzomyia* sandfly More common in New World Delayed-type hypersensitivity reaction	Slow, progressively destructive, painful nodules with ulcerations in ear, nose, throat locations Facial disfigurement and respiratory problems are late findings	Histopathologic examination of tissue, slit-skin smear with Giemsa stain, culture, serology, PCR Subsequent cutaneous trauma may reactivate disease Speciation may direct choice of tx *Tx: azoles, pentavalent antimony, amphotericin B, miltefosine, pentamidine*
American trypanosomiasis 'Chagas' disease' (*Trypanosoma cruzi*)	Predominantly Central/South America Various modes of transmission (insect, blood transfusion, sex, contaminated food) Vector: *Triatoma* spp. (reduviid bug) Insect bite, or contact of open wound or mucous membrane to insect's excrement	Acute: erythematous, pruritic, indurated nodule (chagoma) at site of bug bite 1–2 weeks after bite Swelling of eyelid (Romaña's sign) if infection is acquired through conjunctiva Acute systemic: ranges asymptomatic to fevers, malaise, lymphadenopathy, hepatosplenomegaly and, rarely, myocarditis or meningoencephalitis Chronic: cardiac, GI, and nervous system problems in 15–30 %	Peripheral blood smear, culture, antibody assays, or PCR Prevention: insecticide spray, blood-donor screening, bed nets *Tx: benznidazole, nifurtimox*
African trypanosomiasis 'sleeping sickness' (*Trypanosoma brucei*)	East Africa (*T. b. rhodesiense*)—often acute West Africa (*T. b. gambiense*)—often chronic Vector: *Glossina* spp. (tsetse fly) Insect bite, or contact of open wound or mucous membrane to insect's excrement	First stage: indurated chancre at inoculation site → heals but fever ensues with annular eruption Second stage: daytime somnolence and neurological symptoms Winterbottom's sign: posterior cervical lymphadenopathy (Western) Axillary/inguinal lymphadenopathy (Eastern)	Peripheral blood smear, culture, antibody assays, or PCR Prevention: insecticide, protective clothing/nets *Tx: pentamidine, suramin, melarsopral, eflornithine, or nifurtimox*

Table 2 continued

Infection	Epidemiology	Signs/symptoms	Workup/treatment
Malaria (*Plasmodium* spp.)	Africa, Asia, Oceania, South America Vector: *Anopheles* mosquito	Cyclical fevers, headache, sweats, insomnia, arthralgias, myalgias Skin: pallor, jaundice	Peripheral blood smear, antibody/enzyme assays Prevention: insecticide, protective clothing/nets *Tx: chloroquine, mefloquine, quinine, doxycycline, atovaquone-proguanil, artemisinins (depending on species/resistence)*
Pythiosis (*Pythium insidiosum*)	Worldwide but predominantly Thailand Formerly classified as a fungus, but now considered a parasitic oomycete or 'protist', thus closer related to *Plasmodium* than fungi	Infection typically occurs after exposure to stagnant water (e.g. swimming, agriculture); immunocompromised or those with thalassemia/hemoglobinopathies at highest risk, may be fatal Vascular (arterial), ocular (infectious keratitis), SSTIs/cellulitis	Often delayed diagnosis, culture/serology possible *Tx: prompt surgical amputation, enucleation, or excision is paramount; SSKI and various antifungals/antibiotics used with mixed results*
Cutaneous amebiasis (*Entamoeba histolytica*)	Tropical/rural Africa, Asia, Latin America, developing countries Fecal–oral transmission Extraintestinal spread to liver, brain, respiratory tract, and skin is very rare	Typically GI symptoms only (dysentery) Rare cutaneous lesions = painful, rapidly progressive, necrotic ulcers often in the anogenital region Lesions have well-demarcated, indurated margins with surrounding erythematous 'halo', occasionally with necrotic/gangrenous base	Examination of ulcer exudates/margin scrapings, biopsy, fresh smear of ulcer exudates/margin, stool examination/culture, ELISA, PCR *Tx: oral metronidazole or tinidazole ± luminal agent (paromomycin)*
Lymphatic filariasis (*Wucheria bancrofti, Brugia* spp.)	Endemic in tropical Asia, Africa, Central/South America Mosquito vectors vary geographically 90 % caused by *W. bancrofti. Brugia* occurs in geographic foci	Fevers, chronic lymphedema, hydrocele Lymphedema often painful, erythematous, and typically affects single limbs or scrotum Chronic lymphedema results in elephantiasis	Immunochromatography antigen card works well but only detects *W. bancrofti* PCR, ELISA, blood smear *Tx: DEC, albendazole, or ivermectin, ±doxycycline*
Onchocerciasis 'river blindness' (*Onchocerca volvulus*) [Fig. 3d]	Africa, Middle East, South America Vector: *Similium* blackfly Predominantly ocular/cutaneous disease	Ocular: bilateral itching, redness, pain, photophobia, blurriness, glaucoma, blindness Skin: ranges from pruritic, eczematous rash to ichthyosiform 'lizard skin' to severe skin atrophy and onchocercal depigmentation ('leopard skin') Onchocercoma: subcutaneous nodule of worm bundles	Skin snip, direct visualization of microfiliariae Mazzotti patch test, ELISA, PCR Prevention: Insecticide, protective clothing/nets *Tx: ivermectin ± doxycycline* *Risk of Mazzotti reaction if use DEC*
Loiasis 'African eye worm' (*Loa loa*)	Endemic in western/central Africa Vector: *Chrysops* deerfly	Skin: transient, pruritic angioedema of face or extremities due to hypersensitivity ('Calabar swellings') Ocular: presence of worm in conjunctiva or sclera with itching and photophobia	Direct visualization, peripheral blood smear, ELISA, PCR *Tx: DEC ± ivermectin*
Strongyloidiasis (*Strongyloides stercoralis*)	Endemic in Africa, southeast Asia, West Indies, Americas Fecal–oral or direct penetration through skin	Immunocompetent: eosinophilia, minor GI complaints Immunocompromised: GI, pulmonary symptoms, sepsis, meningitis Skin: edema, pruritus at site of penetration ('ground itch'), and rapidly progressing (5–10 cm/h), serpiginous, urticarial reaction due to larval migration ('cutaneous larva currens')	Serial stool examinations, sputum examination, ELISA, PCR, endoscopy Prevention: proper sanitation and feet protection *Tx: ivermectin and/or albendazole*

Table 2 continued

Infection	Epidemiology	Signs/symptoms	Workup/treatment
Hookworms (*Ancylostoma duodenale*, *Necator americanus*) [Fig. 3e]	*A. duodenale*: Middle East, Africa, India *N. americanus*: Americas, Africa, Asia Fecal–oral or direct penetration through skin	Anemia, weakness, transient pneumonitis (Loeffler's), epigastric pain, diarrhea, eosinophilia Skin: Anemic pallor, pruritic rash at penetration site ('ground itch'), and slower progressing (1–2 cm/day), serpiginous, urticarial reaction due to larval migration (CLM)	Fecal smear, stool examination, fecal culture, PCR *Tx: albendazole or mebendazole; $FeSO_4$ to treat anemia*
Schistosomiasis (*Schistosoma* spp.)	Africa, Asia, Middle East, South America Intermediate host: freshwater snails Direct penetration through skin	'Katayama fever' = systemic hypersensitivity reaction Chronic hepatic, intestinal, or urinary fibrosis Skin: pruritic, papular eruption at site of penetration 'Swimmer's itch' = contact dermatitis when exposed to avian and other nonhuman schistosomes	Visualization of eggs in skin biopsy, feces, or urine Serology, PCR Prevention: adequate feet protection *Tx: praziquantel ± corticosteroids*
Fascioliasis (*Fasciola* spp.)	Africa, Asia, Europe, Americas Humans infected by drinking infected water or eating aquatic plants containing parasites Parasite penetrates intestinal wall and migrates to biliary tract	Acute hepatic phase: fever, fatigue, RUQ abdominal pain Chronic biliary phase: symptoms of biliary obstruction, cholangitis, pancreatitis Skin: generalized pruritus, urticaria or, rarely, CLM-like lesions	Stool examination, serologic testing, fluid aspiration of CLM Prevention: water treatment, thorough food cleaning *Tx: triclabendazole*
Paragonimiasis (*Paragonimus* spp.)	Endemic in Asia, Africa, South America *P. westermani* is the most common species Intermediate hosts: snails, crustaceans Ingestion of undercooked crabs/crayfish	Organisms migrate to lung, causing most to present with tuberculosis-like symptoms (fever, chest pain, chronic cough with hemoptysis) Skin: urticaria, subcutaneous nodules of worm bundles, or a migratory dermatitis	Biopsy/aspiration of subcutaneous nodules, stool examination, sputum examination, CXR, immune assays Avoid raw/undercooked seafood *Tx: praziquantel or triclabendazole*
Cysticercosis (*Taenia solium*)	Endemic in Latin America, Africa, Asia Ingestion of cysts in undercooked pork	Organisms migrate to muscle, heart, and CNS Neurocysticercosis is the most common manifestation Skin: small, mobile, painless nodules due to larval cyst formation in subcutaneous tissues	Biopsy of skin nodule, radiologic imaging of brain, antibody testing, funduscopic examination Risk of seizures with treatment *Tx: albendazole or praziquantel + corticosteroids*

CNS central nervous system, *CL* cutaneous leishmaniasis, *CLM* cutaneous larva migrans, *CXR* chest X-ray, *DEC* diethylcarbamazine, *ELISA* enzyme-linked immunosorbent assay, *$FeSO_4$* iron sulfate, *GI* gastrointestinal, *KOH* potassium hydroxide, *MCL* mucocutaneous leishmaniasis, *PCR* polymerase chain reaction, *RUQ* right upper quadrant, *SSKI* Saturday solution of potassium iodide, *SSTIs* skin and soft tissue infections, *Tx* treatment

victims or responders at the epicenter of a flooding disaster. Therefore, immersion foot injuries encompass a spectrum of four distinct syndromes delineated by water temperature, but all are characterized by continuous foot pain over days to weeks [137].

Trench foot and classic immersion foot are very similar, both resulting from exposure to cold water (approximately 15 °C/60 °F) which causes direct vascular injury. However, trench foot is a more insidious process caused by days to weeks of continuously wet, but not fully immersed, feet, while classic immersion foot is a quicker process resulting from 15–24 h of prolonged and complete water immersion. Time to injury for both trench foot and immersion foot is accelerated as temperatures decrease, and both injuries can be divided into three phases: the pre-hyperemic phase of pale, swollen, and possibly cyanotic feet with numbness and pain; the rewarming hyperemic phase of erythema, increased edema, and throbbing pain; and the variable post-hyperemic phase of deeper joint pain, paresthesias, cold sensitivity, and hyperhidrosis [138]. Vascular and/or nerve

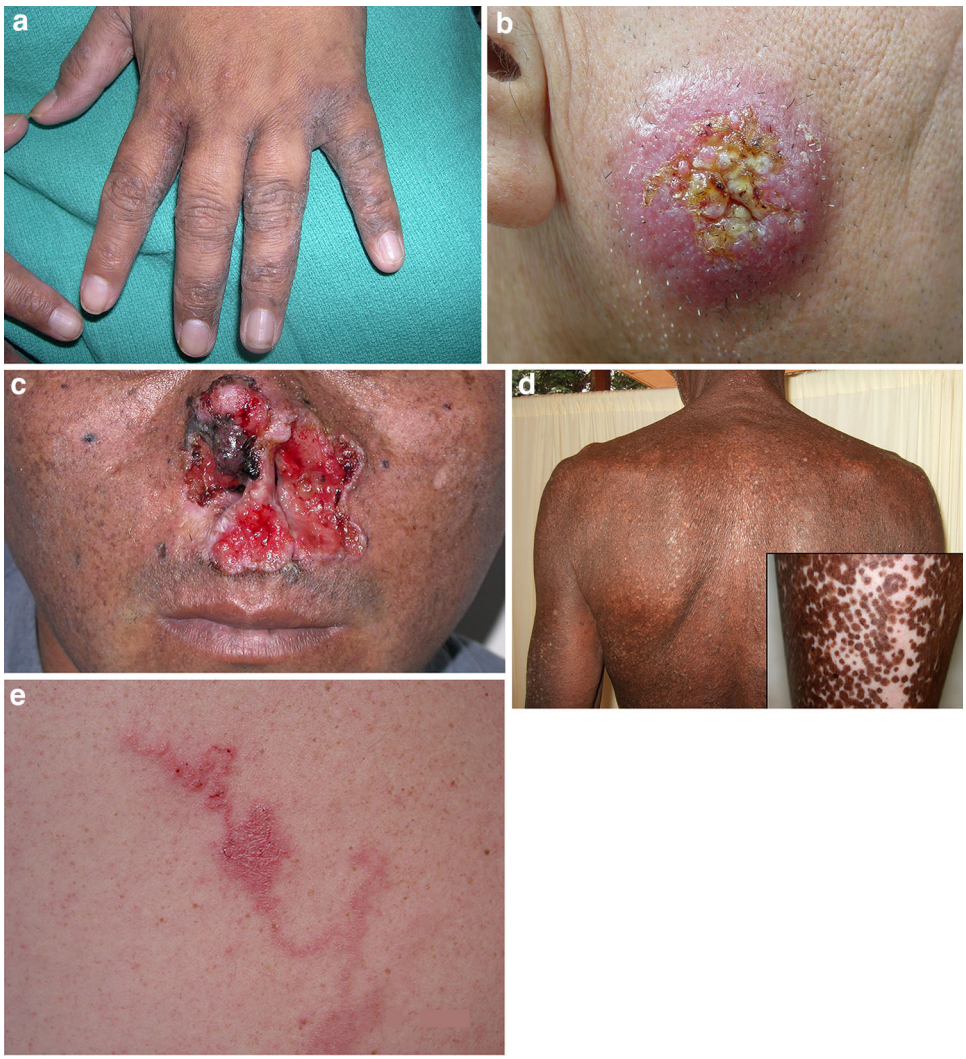

Fig. 3 **a** Erythematous papules and burrows seen in the interdigital web spaces of a patient with scabies. **b** Ulcerated, wet-appearing solitary lesion of cutaneous leishmaniasis. **c** Wet, ulcerated, mutilating appearance of mucocutaneous leishmaniasis, **d** Mottled leopard skin (*inset*) and subcutaneous onchocercomas (*left shoulder*, *right upper back*) of onchocerciasis. **e** Serpiginous and advancing appearance of cutaneous larva migrans

damage can occur, and the risk of infection is increased [139, 140]. There is likely some clinical and histopathological overlap between these entities and perniosis (Fig. 4b) or *chilblains*, as seen in reports of perniosis caused by wading through cold mountain rivers [141], but the terms *trench foot* and classic *immersion foot* imply prolonged water exposure and not just cold temperatures.

Immersion foot injuries in warmer climates are termed either *tropical immersion foot* or *warm-water immersion foot*, and both result from exposure to warmer water temperatures (22–32 °C/72–90 °F). Tropical immersion foot is characterized by several days of continuous immersion, causing a subacute dermatitis. Symptoms progress from burning pain and itching aggravated by pressure with erythema/edema (early) to numbness and paresthesias (late). Warm-water immersion foot is caused by shorter and intermittent immersion causing paresthesias and pain with ambulation, as well as swollen, wrinkled plantar surfaces. Desquamation often occurs during recovery from both forms [137].

Treatment for all types of immersion injuries consist of removal from the wet environment, allowing feet to dry, and careful rewarming if necessary. Vigilant monitoring for secondary infection, such as polymicrobial toe web infection (Fig. 4c) or pitted keratolysis (Fig. 4d), is also indicated. Proper footwear is key to prevention and has improved since the advent of jungle boots with breathable fabric and draining eyelets.

6.3 Contact Dermatitis

Floodwaters can easily become polluted with not only infectious matter but also irritant chemicals and substances. Damaged sewage and water supply systems, agricultural run-off of fertilizers and pesticides, and exposure to wild foliage such as poison ivy are only some of the potential sources of irritants and allergens. Therefore, contact dermatitis can quickly become a common complaint among survivors and rescue workers, and although not typically

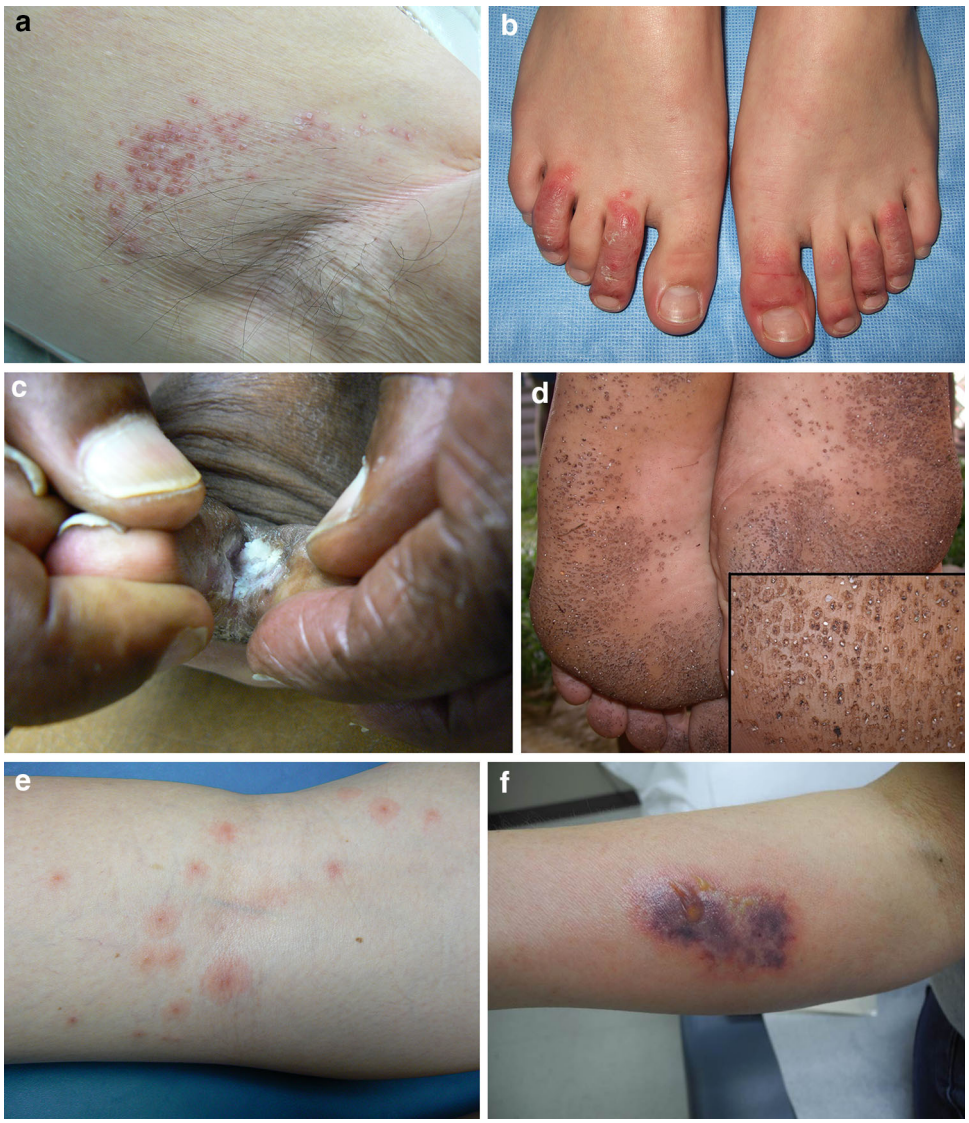

Fig. 4 **a** Erythematous vesicles and papules of miliaria rubra in the sweaty, occluded axillary space. **b** Erythematous to violaceous tender papules of perniosis on the toes of a patient exposed to cold, wet conditions. **c** Polymicrobial infection of the toes as can be seen in immersion foot syndromes. **d** Pitted keratolysis in a patient exposed to chronic, wet conditions. **e** Papular urticaria of multiple arthropod bites. **f** Necrotic erythema of a *Loxosceles reclusa* (Brown recluse) spider bite

life-threatening, it can greatly increase disaster morbidity. For example, after Hurricane Katrina, Louisiana's poison control centers documented an increase in toxic exposures to gasoline, lamp oil, and carbon monoxide [142]. In addition, 44 offshore oil facilities were damaged and cumulatively spilled over 7,000,000 gallons of crude oil, some of which entered residential areas [143]. Similarly, in their study analyzing the types of skin problems after the 2004 Asian tsunami, Lee et al. classified approximately 30 % of skin complaints into an *eczema* group, the majority (76 %) of which were reportedly caused by irritant or allergic contact dermatitis [7]. In this report, the arms and legs were most affected, unlike traumatic skin disorders, which most often involved the hands and feet. However, irritant contact dermatitis of the feet may be difficult to distinguish from fungal infections. For example, of 96 flooding victims presenting to an outpatient clinic in Thailand in 2006, *eczema* was the most common dermatosis, 16 of which were initially thought to be fungal infections after presenting with wet, macerated, and itchy interdigital web spaces [144]. However, fungal culture was positive in only two specimens, while secondary bacterial infections were present in the remaining 14 cases. KOH of skin scrapings is a simple but often very helpful method to investigate a scaly and eczematous dermatitis.

Chemical burns may also result from disastrous flooding due to infiltration of dry chemical stores, damage to factories/warehouses from severe storms, or prolonged exposure to water where inadvertent spillage has occurred. For example, large amounts of sodium hydroxide and ammonium chloride solutions were created when flooding from the 2005 Matsa typhoon infiltrated a dry alkaline chemical storehouse in Shanghai, China [145]. Over 100 workers suffered alkali chemical burns ranging from 0.5 to 25 %

total body surface area, including 68 cases of deep partial-thickness burns and 45 cases of full-thickness burns.

In general, the first approach for the majority of chemical burns is to quickly brush off any remaining dry chemicals and then begin copious irrigation with water at low pressures [146]. For acid burns, continuous water irrigation until the pH of the skin becomes neutral is sufficient, but for alkali burns, two or more hours of continuous irrigation may be required before the skin pH remains neutral 10 min post-irrigation. Certain chemicals, such as dry lime, alkali elements (sodium/potassium/lithium/magnesium), phenol, muriatic acid, and sulfuric acid, may worsen with standard irrigation or require special care, such as mineral oil, soap cleansing, or polyethylene glycol solutions prior to or after water irrigation. Therefore, an attempt should be made to identify the chemical and follow local chemical hazard guidelines or the instructions on the appropriate Material Safety Data Sheet (MSDS), if available.

6.4 Traumatic Wounds

Although dependent on the type of flooding disaster, traumatic wounds are typically the main initial cause of morbidity and mortality. For example, in Calang, the capital city of the Aceh Jaya District, Indonesia, and near the epicenter of the 2004 Asian tsunami, 70 % of the city's population of approximately 12,000 died at the time of impact [147]. After the tsunami, emergency departments (EDs) were overflowing with injured survivors, such as the Karapitiya Teaching Hospital in the Southern Province of Sri Lanka that observed admission rates due to injury increase from 20 % pre-tsunami to 89 % on the day of impact [148]. However, in some cases there may be a delay before presentation. For example, aggregate data from the emergency rooms of a six-hospital urban healthcare system (including a Level 1 trauma center) in the coastal southeastern region of Virginia actually observed a 46 % decrease from the average daily ED census on the day of landfall of Hurricane Isabel in 2003, and then a 25–35 % increase above average in the subsequent 4 days post-impact [149]. The largest increase among presenting complaints was minor trauma (+57 %).

Increased rates of minor trauma such as cuts, lacerations, and puncture wounds, some infected and many occurring in both victims and relief workers, were similarly seen in other EDs after Hurricanes Andrew, Georges, and Katrina [150–153]. Even minor abrasions or cuts on lower extremities, when chronically exposed to the stagnant water or mud following a flood, are at increased risk of infection and may subsequently evolve into the polymicrobial *tropical ulcer* (tropical phagedenic ulcer, Aden ulcer, Malabar ulcer, or jungle rot) [154]. Classically, an inflammatory papule will progress to a painless, undermined ulcer mimicking Buruli ulcer or cutaneous leishmaniasis, and malnourishment may be a predisposing factor. Therefore, those responding to disaster situations should be prepared for the cornerstones of primary wound care: cleaning, debridement, closure, and protection [155].

First, basic cleaning to carefully remove obvious debris and foreign bodies must be performed. The extent of the wound must be identified to assess for injury to underlying structures, and surgical consultation for repair should be obtained if appropriate. Any devitalized, severely damaged, or necrotic tissue must be debrided as retained devitalized tissue greatly increases the risk of infection [156]. Ample irrigation should then be performed before closure, typically with isotonic saline for uncomplicated wounds. Hydrogen peroxide is generally not recommended as it can damage exposed tissue and thus delay wound healing, but a diluted antiseptic solution may be clinically indicated in excessively dirty, grossly infected, or bite wounds [157]. Lastly, uncomplicated wounds caused by relatively clean, sharp instruments may be closed if the time of injury was less than 12–18 h prior. Deep or puncture wounds, grossly infected/contaminated wounds, animal bites, or wounds that occurred more than 18 h earlier should be allowed to heal by secondary intention [158]. In flooding disasters, major and minor wounds are often contaminated, and therefore many recommend leaving even minor wounds open [159]. Tetanus immunization may be indicated depending on the patient's immunization status. Prophylactic antibiotics have not yet been shown to significantly reduce infection rates in most wounds [160], but may be clinically indicated in certain high-risk infections such as extremity or puncture-type bite wounds, highly contaminated or grossly infected wounds, crush wounds, or wounds that cannot be adequately cleaned and debrided. Relative indications for antibiotic therapy include wounds involving tendons, bones, or joints, wounds in areas of underlying lymphedema or venous stasis, wounds with delayed treatment, and wounds in the immunocompromised [161].

Environmental electrical injuries, such as those from lightning strikes and downed powerlines, may also pose a hazardous risk to disaster victims, particularly after severe storms with heavy lighting and strong winds. Not only does water pose an electrical conduction hazard, but victims climbing tall structures to avoid rising waters may inadvertently be at increased risk of lightning strikes or powerline injury. Disaster victims and relief workers are also at risk immediately after the disaster during cleanup operations. As an example, seven cases of fatal electrocutions were reported in Puerto Rico by the CDC after Hurricane Hugo struck in 1989 [162, 163]. Although one case involved a man electrocuted while trying to remove his

television antenna prior to the storm, the remaining six cases occurred after the storm, five of which were occupational cases of electrocution in experienced workers by dangling or downed powerlines. The sixth electrocution occurred after a man contacted an exposed ground cable while chopping a downed tree. Four similar cases of electrocution were also reported in South Carolina after Hurricane Hugo [164].

Lightning strikes typically comprise only a small part of electrical injuries but they are probably underreported in the nondisaster setting [165]. Contact with exposed electrical wiring or downed powerlines are more likely to occur during severe weather events and subsequent recovery operations, as discussed above, and the extent of injury is highly dependent on the amount of current conducted through the body. Electrical injuries most often cause death by cardiac arrest due to the direct effect of the electrical current, and thus immediate cardiopulmonary resuscitation is essential. However, a portion of the electrical energy is also converted to thermal energy, often resulting in cutaneous burns. In both lightning strikes and electrical wiring injuries, superficial, partial-thickness, and/or full-thickness burns may occur. Due to an extremely short duration of discharge, a direct current, and a *flashover effect* where current flows over the skin surface, discharging into the ground, superficial burns are more common in lightning strike injuries, and typically heal rapidly [166, 167]. Burns can occur in up to 89 % of lightning-strike victims, and skin lesion morphology varies [168]. For example, linear burns occur in areas of high sweat concentration and are probably due to water vaporization, punctate burns are typically very small in size but may be full-thickness, and feathering lesions induced by the flashover effect present as branching, arborescent, and erythematous cutaneous lesions called keraunographic markings or Lichtenberg figures [169]. These are from extravasation of blood and are thus not true burns and typically fade within a few hours [170].

In contrast, low- and high-voltage electrical wiring injuries (e.g. household wiring, powerlines) are associated with increased morbidity and mortality primarily due to an increased duration of exposure and an alternating current that can result in prolonged muscular tetany and severe internal organ damage [167]. Visible cutaneous damage often occurs in electrical wiring injuries, depending on voltage, as evident in one postmortem study of 220 fatal electrical injuries with visible burns in 57 and 96 % of low- and high-voltage victims, respectively [171]. However, it is important to note that the extent of visible damage does not correlate to internal damage, particularly in low-voltage injuries, as patients with little to no cutaneous burns may still have severe muscle coagulation and internal organ damage [167].

Treatment for lightning and electrical wiring burns is no different from other thermal burns. In addition to standard wound care, aggressive fluid resuscitation, fasciotomy, escharotomy, debridement, and even amputation may be required, depending on the severity of the burn [167].

6.5 Animal Bites

Providers responding to a flooding disaster must be prepared for an increase in bites from wild and domestic animals. Rising flood levels will cause wild populations to shift to higher ground and compete with domestic animals and humans for space, perhaps increasing horizontal transmission of disease (e.g. rabies). For example, an animal bite was the third most common trauma complaint seen by medical response teams deployed to Webster, Texas, following Hurricane Ike in September 2008 [172]. However, interestingly, household pets known to the victim caused all of the dog and cat bites; it was not wild animals or misplaced pets biting strangers or rescue workers as was expected. Initial WHO situation reports from the dreadful 2010 floods in Pakistan provide further evidence of the risk of animal bites, reporting over 700 dog bites in the first 3 weeks [131]. Snake bites were also a serious concern as hundreds of cases were reported in the first few weeks and local stores of antivenin were quickly depleted.

Animal bites may become infected and are often polymicrobial, commonly from *Pasteurella*, staphylococci, streptococci, and/or anaerobic bacteria [173]. Wounds can vary from minor scratches and abrasions to deep lacerations with or without fractures, and can occasionally progress to sepsis or osteomyelitis [174]. Providers should assess for underlying tissue damage and for signs of infection, such as fever, erythema, swelling, tenderness, and purulent drainage. Aerobic and anaerobic blood cultures should be obtained if evidence of systemic infection exists, and plain radiographs are indicated if bite wounds are deep, or bony involvement is thought likely. Superficial wound cultures are not typically helpful unless obvious evidence of infection exists. Standard wound care as described above should be performed, with particular attention to adequate debridement, cleansing the wound with a mild antiseptic solution, and consideration of antibiotic therapy in deep puncture wounds, obvious signs of infection, wounds with crush injury, wounds on the hands or close to bones/joints, wounds requiring surgical repair, and the immunocompromised [175]. If antibiotic therapy is to be given, the first dose should be given as soon as possible and should provide empiric coverage of gram-negative and anaerobic organisms (e.g. amoxicillin-clavulanate). Most wounds should be allowed to heal by secondary intention; however, primary wound closure or delayed primary closure may be feasible options in certain

circumstances [176]. Tetanus prophylaxis should be given according to the patient's immunization status for any bite wound breaking the skin surface. Rabies prophylaxis may also be indicated in bites from unvaccinated pets, wild animals, and in areas where there is a high prevalence of rabies.

The management of snake bites varies worldwide, and thus knowing the indigenous species and bringing the appropriate antivenin, if available, is paramount. A snake field guide was published in 2006 specifically for disaster-response personnel deploying into hurricane-prone regions of North America [177]. Envenomation may cause neurotoxicity, shock, rhabdomyolysis, renal failure, coagulopathy, or local tissue necrosis, and thus treatment strategy is highly dependent on the type of venom. However, general field guidelines include removing the patient from the snake's territory, keeping the patient warm and calm, immobilizing the bite injury below the level of the heart if possible, identifying the snake without putting the patient or oneself in danger, and evacuating the patient as soon as possible to the nearest medical facility [178]. Antivenin may be available but can cause allergic reactions such as serum sickness or anaphylaxis, and thus should be used carefully [179].

6.6 Insect Bites

Insect bites and stings may also increase after severe storms and flooding disasters, and can torment both victims and responders [180]. For example, cumulative data from seven North Carolina EDs treating patients after Hurricane Hugo show that of 1833 patients treated for injuries, 428 (21 %) were due to insect stings, and a significant portion (26 %) of insect-sting patients experienced a generalized reaction (e.g. hives and/or wheezing) [181]. Similarly, 43 % of 136 construction workers repairing buildings damaged by Hurricanes Katrina and Rita were diagnosed with papular urticaria by a dermatologist, and a significant majority (78 %) were noted to be housed in 4 of 11 huts, the only four that had flooded during the hurricanes and likely harbored mites as the source of papular urticaria [182].

Many insects may lose their natural hosts, such as rodents or birds who move to other areas by flooding, and therefore seek alternative hosts such as humans [183]. Hurricanes and flooding may also reduce natural predators of insects, subsequently allowing their populations to overgrow. This was the case in Cozumel, Mexico, in 1989 after Hurricane Gilbert killed the natural predators (wasps and bees) of a local moth (*Hylesia alinda*) [184]. This moth has nettling hairs on its abdomen that contain a histamine-like substance, and caused a puzzling outbreak of a non-specific dermatitis in hundreds of hotel employees.

Insects themselves may also move to avoid rising floodwaters. For example, staphylinid (rove) beetles (*Paederus* spp.) are a type of *blister beetle* that secrete paederin, an irritating vesicant often released when the insect is swatted or crushed onto the skin. This vesicant causes an irritant contact dermatitis called Paederus dermatitis, linear dermatitis, blister beetle dermatitis, Nairobi fly dermatitis, or Nairobi eye (conjunctivitis), depending on the region. During rains or flooding, these insects seek dry ground, thus explaining why an Egyptian factory was attacked by rove beetles after a sudden flood in 1994, resulting in cases of dermatitis and conjunctivitis in approximately 40 workers [185]. Although typically seen in Africa and the Middle East, a rove beetle outbreak was also reported in a US military unit training in the Arizona desert during heavy rain and flooding [186].

In general, arthropod bites can cause local inflammatory reactions, hypersensitivity reactions such as papular urticaria (Fig. 4e), painful necrosis after certain spider bites (Fig. 4f), or systemic allergic reactions. Reaction type varies depending on the offending arthropod, and an exhaustive list cannot be cited here. Specific treatments also vary but, in general, insect bites should be washed with soap and water, and then optional cooling packs or topical lotions (e.g. calamine) applied to soothe irritation and pruritus. Topical antihistamines or steroids are not routinely recommended, although they may provide some relief, and systemic antihistamines may help ameliorate severe pruritus [187]. Temporary surveillance for significant allergic reactions or anaphylaxis is always warranted and, if present, subcutaneous epinephrine injection and airway management may be necessary [188]. Insects can also transmit a myriad of disease-causing pathogens, including bacteria, viruses, protozoa, and helminth parasites. Therefore, the use of protective or permethrin-treated clothing and fabrics, insect repellent, and physical barriers (e.g. bed nets) is vital to adequate prevention. Support of environmental specialists and entomologists is often necessary.

7 Psychodermatology

In a disaster situation, saving life and limb is paramount, but the secondary traumatic effects of losing family, friends, shelter, possessions, land, and livelihood can have long-term and deleterious psychoemotional consequences. For example, in a cohort of 17 people who were severely injured in the Asian tsunami and airlifted to a German medical center, each patient had at least one relative killed, the children of two mothers perished, and the majority of patients suffered severe distress consisting of nightmares, sleep difficulties, flashbacks, intrusive thoughts, guilt, and

anxiety [189]. However, not well understood is the link between these psychoemotional problems and dermatological disorders. Certainly in the disaster setting, any existing medical condition can be exacerbated (possibly exhibiting skin manifestations) simply due to loss of access to routine medical care, loss or contamination of daily medications, and poor sanitation. This was demonstrated in a recent report outlining the significant impact of a flooding disaster in West Africa on people living with HIV [190]. However, some purport that the increased risk of cutaneous disease during a water-associated disaster results not only from water and traumatic exposure but also from psychological and physical stress [191]. Examples of possible psychodermatological disorders include stress-related exacerbation of atopic dermatitis, seborrheic dermatitis, psoriasis, and alopecia areata, as well as induction of delusional infestation, neurotic excoriation, factitial dermatitis, or trichotillomania [192].

Due to the inevitable stress and anxiety among disaster survivors, and the potential for the induction or exacerbation of dermatological disorders, an effort should be made to provide a multidisciplinary approach consisting of prompt, appropriate medical care as well as mental health intervention.

8 Conclusions

Over the last few decades, major flooding events seem to be occurring with increased frequency and severity, and many of the deleterious effects have dermatological manifestations or consequences. This extensive review may be useful as a basic field manual for those called to respond to the victims of flooding disasters.

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