Parasitic Diseases With Cutaneous Manifestations

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Parasitic diseases result in a significant global health burden. While often thought to be isolated to returning travelers, parasitic diseases can also be acquired locally in the United States. Therefore, clinicians must be aware of the cutaneous manifestations of parasitic diseases to allow for prompt recognition, effective management, and subsequent mitigation of complications. This commentary also reviews pharmacologic treatment options for several common diseases.

The burden of parasitic disease impacts individuals worldwide. Within the United States, parasitic disease is usually associated with travel or immigration, but infestations may also be acquired locally (autochthonously). Because one-third of travelers present with cutaneous disease as late as 1 month after returning home, the temporal association with travel may be obscured [1].

This commentary will focus on the most common parasitic diseases with cutaneous manifestations encountered within the United States. Although other parasites can present with dermatologic findings, our discussion will cover pediculosis, scabies, demodicosis, cutaneous larva migrans, cutaneous schistosomiasis, tungiasis, myiasis, leishmaniasis, and trypanosomiasis.

Pediculosis

Human lice infestations result from Pediculus humanus capitis (head lice), Pediculus humanus humanus (body lice), and Phthirus pubis (pubic lice). Prolonged interpersonal contact is responsible for most transmission, but fomites contribute significantly to body lice acquisition. The typical presentation includes excoriated erythematous papules, pruritus, and regional lymphadenopathy. Potential complications include impetigo and post-streptococcal glomerulonephritis [2].

Head lice infestations are common regardless of geography or socioeconomic status, and they cost the US population $1 billion annually [2, 3]. Misdiagnosis leads to 12–24 million missed school days and $4–$8 million of lost parental earnings annually [4]. Head lice (2–3 mm in size) frequently hide in the retroauricular and occipital scalp but combing to remove nits may have limited efficacy beyond decreasing social stigmatization, but combing is commonly recommended [2-6].

Despite the development of some drug resistance, permethrin 1% and synergized pyrethrins (pyrethrins plus piperonyl butoxide) are first-line agents for head lice [3, 5]. In refractory cases, topical benzyl alcohol 5%, spinosad 0.9%, ivermectin 0.5%, or US formulated malathion 0.5% are recommended treatments [2, 3, 7]. Lindane 1% is not recommended due to neurotoxicity and resistance [4, 5]. Promising new therapies include dimethicone, isopropyl myristate, and Louse-Buster desiccation [3, 4]. Nonovicidal treatments require readministration after eggs hatch at 7–10 days, and ovicidal treatments (malathion, spinosad, and ivermectin) should also be repeated if live lice are observed [3-5].

Home remedies for lice are largely ineffective, but environmental modifications such as vacuuming, laundering (at a temperature ≥149°F), 2-week fomite isolation, and close-contact avoidance may prevent reinfection [3-5]. Children can return to school after the first treatment because of the low risk of classroom transmission [3, 4]. School screenings and “no-nit” policies are poor predictors of infection and therefore are not recommended [2, 3].

Body lice lay eggs in the seams of clothing and frequently infest prisoners, refugees, and homeless individuals. The main treatment is laundering (at temperatures ≥130°F) and contact avoidance. Complications include severe diseases associated with Borrelia recurrentis, Bartonella quintana, and Rickettsia prowazekii transmission [2].

Transmission of pubic lice occurs mainly with sexual contact, and affected individuals often have concurrent sexually transmitted infections [2]. Many infestations (60%) involve hair-bearing areas beyond the genitalia [6]. Pediculosis ciliaris is an infestation of the eyelid margin with accompanying lice are also found [3, 6]. Eggs and nits are firmly attached, whereas pseudonits (seen with scaling scalp disorders) are relatively mobile [2, 3]. Combining to remove nits may have limited efficacy beyond decreasing social stigmatization, but combing is commonly recommended [2-6].

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conjunctivitis and edema, which may lead to corneal epithelial keratitis [2]. Management of pubic lice includes pubic shaving, head lice medications, temporarily limiting sexual contact, and ophthalmic grade petroleum jelly for pediculosis ciliaris [2].

**Scabies**

The scabies mite (*Sarcoptes scabiei var. hominis*) is responsible for 1.5 million disability-adjusted life-years worldwide, as a result of pruritus, insomnia, school and work absences, and psychological distress [2, 7]. Transmission results from prolonged interpersonal contact or, less commonly, fomites [2]. Elderly individuals, persons with disabilities, and homeless individuals within impoverished and overcrowded communities are particularly at risk [2].

A typical infestation involves 5–15 female mites living within epidermal burrows that induce a hypersensitivity reaction 3–4 weeks after the initial exposure or a few days after re-exposure [2, 8]. Usually the finger webs, wrists, or genitalia are severely pruritic and show erythematous, waxy burrows with a terminal black dot (the mite) [2, 8]. Immunocompromised patients may present with crusted scabies, in which there are thick scaly lesions and millions of mites (See Figure 1) [8]. A mineral oil preparation can confirm the diagnosis, but the absence of microscopic findings should not delay empirical treatment if a compatible exposure history or typical burrows are present [2, 8]. Complications can include bacterial infection, post-streptococcal glomerulonephritis, crusted scabies-associated sepsis, and post-scabetic pruritus, which may persist for 2–4 weeks [2, 6, 7].

For treatment of scabies, the drug of choice is topical permethrin 5% with reapplication after 7 days to kill newly hatched mites that emerge 2–3 days after egg deposition [2, 6]. Alternate treatments include oral ivermectin, topical ivermectin 1%, synergized pyrethrins, precipitated sulfur, benzyl benzoate 10%, and crotamiton 10% [2, 6, 7, 9]. Lindane 1% is less effective and neurotoxic [9]. Treatment of crusted scabies requires oral ivermectin with either topical permethrin 5% or benzyl benzoate 25%. (Keratolytic creams such as salicylic acid may be added to remove scaling.) [2]. Oral moxidectin is a new drug related to ivermectin that shows promise [7]. Fortunately, scabicide resistance is not significant [2, 7]. Isolation within the hospital is only recommended for crusted scabies [2]. Clothing should be laundered or bagged for 5–7 days to prevent reinfestation [6].

**Demodicosis**

*Demodex* mites asymptptomatically inhabit the pilosebaceous units of most adults (80%–100%), but an increased mite density (>5 mites/cm²) may induce pityriasis folliculorum, pustular folliculitis, periorificial dermatitis, papulopustular rosacea, and papulopustular scalp eruptions (See Figure 2) [10, 11]. Consequently, a *Demodex* mite infestation should be considered when a patient has refractory dermatologic eruptions. Oral ivermectin is the treatment of choice for *Demodex* mites, but alternatives include oral metronidazole and topical formulations of sulfur, permethrin 5%, benzyl benzoate 10%, metronidazole 0.75%, ivermectin 1%, and crotamiton [7, 10-12].

**Cutaneous Larva Migrans**

Cutaneous larva migrans (CLM) is a pruritic serpiginous eruption caused by larvae migrating slowly (1–2 cm per day) within the skin (See Figure 3) [1, 6]. While human hookworms (*Ancylostoma duodenale* and *Necator americanus*) may cause CLM prior to gastrointestinal infestation, most CLM cases are caused by animal hookworms (*Ancylostoma caninum* and...
A. braziliense) that cannot penetrate the epidermal basement membrane and therefore induce a self-limited eruption that resolves within weeks to months without treatment [1, 6]. CLM is most frequently contracted after walking barefoot in contaminated soil or sand in warm climates (including the Southeastern United States), but lesion onset may be delayed by 4 weeks after exposure [1, 6]. Complications can include pneumonia, cellulitis, and abscesses [1].

Technically, only symptomatic treatment is required for confirmed animal hookworm cases, but curative treatment relieves symptoms and mitigates the risk of an unidentified Necator americanus infestation [6]. Oral albendazole and ivermectin are the drugs of choice, but topical thiabendazole is also effective [1]. CLM should be distinguished from larva currens secondary to Strongyloides stercoralis (endemic within the Southeastern United States), which migrates faster (at a rate of a few centimeters per hour) and may cause serious systemic disease, especially in immunocompromised patients [1, 6, 13].

**Cutaneous Schistosomiasis**

Cutaneous schistosomiasis occurs worldwide and typically presents with pruritic erythematous papular, urticarial, or purpuric lesions after the larvae penetrate exposed skin. Human schistosome infections (Schistosoma mansoni, S. haematobium, and S. japonicum) infrequently present with skin involvement, but animal schistosomes (Trichobilharzia stagnicolae, T. physellae, and Gigantobilharzia spp.) can penetrate exposed human skin and induce a transient dermatitis called “swimmer’s itch” (cercarial dermatitis) that resolves after 7-10 days. Animal schistosomes are present worldwide. Treatment is symptomatic except in the unusual case when cutaneous human schistosomiasis is suspected, in which case the patient is treated with praziquantel [6].

**Tungiasis**

Tungiasis is acquired while walking barefoot on beaches or sandy soil during travel to Latin America and sub-Saharan Africa. Typically, a female sand flea (Tunga penetrans) asymptptomatically burrows into the epidermis of the foot. Subsequently, a pruritic, painful, and white papular lesion with a central dark discoloration develops over a period of 2 weeks. The potential complications of gangrene, tetanus, and osteomyelitis necessitate sterile removal of the flea and possible administration of antibiotics and the tetanus vaccine [1].

**Myiasis**

Myiasis results from fly larvae infestation of the skin, and it presents in several forms—furuncular, wound, and migratory—depending on the species of fly [1]. Travel to tropical regions is usually responsible for myiasis, but autochthonous infestations are reported in the Southern United States [1, 6].

Furuncular myiasis presents as a nodule with a central respiratory pore and symptoms of formication, pruritus, sharp pain, and discharge [14]. Dermatobia hominis are acquired from travel to Central America and South America (See Figure 4), whereas Cuterebra spp. are responsible for most autochthonous infestations [14]. Complications include bacterial infection, tetanus, and extensive destruction of surrounding tissue [14].

Wound myiasis is caused by many fly species worldwide, but Lucilia sericata and Phormia regina are responsible for most autochthonous infestations [14]. The presence of open wounds, peripheral vascular disease, alcoholism, or immunosuppression increases the risk of infestation, which may present with fever, chills, hypereosinophilia, or secondary infection [1, 14].

**FIGURE 2.** Demodex Folliculitis of the Face and Scalp

**FIGURE 3.** Cutaneous Larva Migrans of the Foot
Migratory myiasis presents with pruritus and a moving erythematous serpentine lesion secondary to a self-limited subcutaneous or lower epidermal infestation by *Hypoderma* and *Gasterophilus* spp., respectively. Acquisition of the larvae is associated with exposure to cattle or horses, respectively. In contrast to CLM, migratory myiasis lasts longer (months) and involves a smaller migratory area. Complications may include ascites, hemopericardium, meningitis, and intracerebral invasion [14].

Furuncular and wound myiasis should be treated by occlusion with petroleum jelly, nail polish, paraffin, or bacon for 24 hours to induce oxygen deprivation and promote larval self-extraction or death [14]. Manual squeezing of furuncular myiasis may be effective for some species, but *Dermatobia hominis* have bidirectional spines that prevent forceful removal [14]. Consequently, surgery is appropriate for furuncular and migratory myiasis [1]. In addition to irrigation, wound myiasis can be managed with topical or oral ivermectin [1].

**Leishmaniasis**

Leishmaniasis is second only to malaria in terms of the number of annual parasite-related deaths (20,000–30,000) [15]. Cutaneous, mucocutaneous, and visceral leishmaniasis is transmitted to humans by the *Phlebotomus* and *Lutzomyia* sandflies [1, 6]. Cutaneous leishmaniasis (CL), the most common type of leishmaniasis, is mainly acquired during travel to tropical and subtropical areas, but autochthonous cases have been reported, particularly in Texas [1, 15, 16]. Clinically, CL presents as a papule at the inoculation site that enlarges into a nodule or plaque and painlessly ulcerates; the ulcer may persist for months to years without treatment (See Figure 5) [1, 6, 16]. Depending on the species, CL may also progress to mucocutaneous leishmaniasis (ML) [1, 6].

CL is typically caused by species not associated with progression to ML, and therefore it can be effectively and equivalently managed with localized or topical paromomycin, imiquimod, or intralesional pentavalent antimonials (sodium stibogluconate and meglumine antimoniate) [15]. If the species is unknown or is associated with ML, systemic therapy is indicated [15]. Most countries consider pentavalent antimonials to be first-line systemic therapy, but liposomal amphotericin B is commonly used in the United States [15, 17, 18]. Second-line systemic options include amphotericin B deoxycholate, liposomal amphotericin B, oral miltefosine, and pentamidine (if the patient does not respond to the first-line therapy or is intolerant to antimonials) [15, 17, 18]. Other potential therapies include cryotherapy, thermotherapy, daylight-activated photodynamic therapy, (R)-PA-824, and various imidazoles (including ketoconazole, fluconazole, and fexinidazole) [17, 18].

**Trypanosomiasis**

American trypanosomiasis (*Trypanosoma cruzi*), or Chagas disease, is endemic to Latin America and likely afflicts 10 million people. Transmission typically results from the introduction of fecal matter from infected Triatomines into the bloodstream. However, transmission may also occur via transplacental transfer, blood transfusions, organ transplantations, and food. While travel is responsible for most infestations, autochthonous cases have been reported in the Southern United States. The acute presentation includes a chagoma, an indurated violaceous nodule with central edema that may resolve within weeks, and schizotripanides, a transient morbilliform or urticarial rash. Therapies for the acute infestation include benznidazole as a first-line therapy, and nifurtimox. During the chronic phase, patients may develop cardiac, gastrointestinal, or neurological sequelae. Because 60%–70% of patients remain asymptomatic, treatment of chronic American trypanosomiasis is controversial [19].
African trypanosomiasis (Trypanosoma brucei gambiense and T. brucei rhodesiense) is transmitted to travelers by the tsetse fly (Glossina spp.) [20]. The acute presentation includes a circumscribed, indurated, and violaceous nodule that desquamates after 2–3 weeks, leaving behind a central eschar [20]. Other acute findings include localized lymphadenitis and trypanids, a transient erythematous targetoid macular or urticarial rash [20]. Awareness of these cutaneous manifestations may allow for early treatment and prevention of central nervous system involvement [20]. Treatment of the acute phase involves use of pentamidine (for T. b. gambiense) or suramin (for T. b. rhodesiense), whereas individuals with central nervous system involvement require treatment with melarsoprol, dimercaprol, or eflornithine [21]. Fexinidazole is a promising medication, but it requires further study [21].

Conclusion

Parasitic infestation of the skin is a common presentation to dermatologists as well as to primary care physicians. The disease burden can cause a debilitating itch or complications from secondary infection. Accurate diagnosis can be facilitated by microscopic examination of skin scrapings, microscopic examination of hair, or in some cases biopsy. Prompt identification and management of both the infestation as well as related symptoms can provide rapid clinical improvement and return to a normal lifestyle.

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