Parasitic infestations affect much of the world’s population, particularly developing countries. Helminths (parasitic worms) are one of the most common parasitic infections and contribute to multiple socioeconomic problems and human diseases. Helminths are classified into 2 distinct phyla: roundworms (nematode) and flatworms (platyhelminthes). Roundworms have a cylindrical unsegmented body with a body cavity, digestive tract with openings on both ends, and 2 separate sexes. Flatworms are characterized by a flat body, lack of specialized circulatory or respiratory organs, typically a single opening digestive track, and predominately hermaphroditic species. The flatworms can be further divided into trematodes (or flukes), which are unsegmented and cestodes (or tapeworms), which have segmented bodies. Parasitic helminths can be acquired via a variety of mechanisms, including contaminated water, undercooked meat, and through direct skin contact like walking barefoot. The manifestations of human disease range from self-limited pruritic rashes to life-threatening multi-system infestations.

Nematodes
See the article entitled “Tropical dermatology: cutaneous larva migrans, gnathostomiasis, cutaneous amebiasis and trombiculiasis” for a discussion of gnathostomiasis and cutaneous larva migrans.

Strongyloidiasis
Strongyloidiasis is caused by the helminth Strongyloides stercoralis, which is a widespread, soil-transmitted intestinal nematode common in tropical and subtropical countries. Infection occurs via filariform larvae penetration into the skin. The organism travels by the venous system to the lungs, then ascends the bronchi to the trachea, it is then coughed up and swallowed by the host, and finally resides in the small intestine.

Larva currens (Figures 1 and 2) is the classic skin finding of strongyloidiasis. It is characterized by a serpiginous urticarial rash that moves rapidly (up to 10 cm/hour) through the skin. Larva currens is typically found on the buttocks, thighs, and lower extremities and lasts hours to a few days. Cutaneous findings may be sporadic with symptom-free periods of weeks to months leading to a delay in diagnosis and occasionally referral to psychiatry. Other common presentations include: chronic urticaria (lasting 1-2 days), morbilliform eruptions, prurigo nodularis or lichen simplex chronicus.

Rare, disseminated cases occur in the setting of immunosuppression and can have fatal results via sepsis, meningitis, and acute respiratory distress syndrome (ARDS). Disseminated strongyloidiasis is increasingly associated with systemic corticosteroids therapy.
Delay in diagnoses and treatment can have fatal results. Treatment for at-risk patients on immunosuppressives, he developed acute respiratory failure and septic shock leading to death. A few days before his death, he developed petechiae and purpura on his abdomen and thighs that revealed interstitial strongyloides larvae on biopsy. 

Treatment options for strongyloidiasis include: oral thiabendazole (25mg/kg BID), ivermectin (200mcg/kg/day), and albenza (400mg once or twice daily). Oral regimens are short in duration, ranging from 1-5 days and 7-14 days for disseminated cases. Empiric treatment for at-risk patients on immunosuppressive therapy includes ivermectin daily (200 mcg/kg). 

**Onchocerciasis**

Onchocerciasis is caused by the tissue-dwelling nematode *Oncho cercus volvulus*, which is transmitted by the blackfly. It is commonly found in sub-Saharan Africa, Central and South America, and Yemen. Classic cutaneous manifestations include: “craw-craw” (acute papular pruritic dermatitis), onchocercoma containing the adult parasitic worm (Figure 3), “elephant or lizard skin” (lichenification and hyperpigmentation), “leopard skin” (atrophy and loss of pigment that spares the perifollicular skin), “hanging groin” (elephantiasis from chronic lymphatic obstruction), and sowda reaction (hyperreactive, localized and asymmetric form characterized by pruritic, hyperpigmented, and hyperkeratotic plaques). 

Ocular manifestations are due to microfilariae invasion into the conjunctivae, cornea, and anterior/posterior chambers. Clinical manifestations include: conjunctivitis, sclerosing keratitis, uveitis, optic atrophy, glaucoma, and blindness. Disease-induced blindness is known as “river blindness” and according to the World Health Organization, is the world’s second leading infectious cause of blindness. 

Nodding syndrome (NS) is an unusual tropical disorder that affects children and adolescents, particularly in South Sudan, southern Tanzania, and northern Uganda. It is characterized by occasional, involuntary nodding of the head, which is thought to be a form of epilepsy. Disease progression after development of head nodding is rapid and consists of seizures, mental and growth retardation, malnutrition, and increased mortality. Existing data suggests an association with *Onchocerca volvulus*, but definitive etiology has yet to be determined. Direct invasion of *O. volvulus* into the central nervous system, immunological response to the parasite, or pruritus driven insomnia resulting in an increased susceptibility to seizure, are proposed mechanisms. While there is no cure for nodding syndrome, current recommendations consist of treatment with ivermectin and anticonvulsants.

Treatment options for onchocerciasis include: ivermectin 150mcg/kg single dose every 3 to 12 months. Doxycycline 100-200mg daily for 6 weeks has been shown to facilitate slower decline in microfilariae levels by targeting *Wolbachia* endosymbionts, thus reducing the risk of the inflammatory Mazzotti reaction seen in patients with a high parasite burden and rapid death of microfilariae.

In the effort to eliminate onchocerciasis from Africa, there has been a search for a reliable point-of-care diagnostic test that can distinguish between active and past infection, as well as gauge treatment response. Globish et al, from Scripps Research Institute, discovered NATOG (N-acetyltyramine-O, β-glucuronide), a unique host-specific biomarker of *O. volvulus*. Using liquid chromatography, NATOG was found in the urine of infected patients but was lacking in uninfected controls. Additionally, the concentration of NATOG was greatly reduced in infected patients that were treated for *O. volvulus* infection with doxycycline. This biomarker may assist in the effort to monitor disease progression and the effort to eliminate onchocerciasis from endemic areas.

**Dirofilariasis**

Dirofilariasis is a rare human disease caused by nematodes of the genus *Dirofilaria*. There are about 40 different species of *Dirofilaria*, which typically infect cats, dogs, foxes, and raccoons. There are only a few species that cause human disease, and they include: *Dirofilaria immitis* (dogs), *Dirofilaria repens* (dogs and cats), *Dirofilaria striata* (boccsats), *Dirofilaria tenuis* (raccoons and opossums), and *Dirofilaria ursi* (bears). 

Human disease is typically transmitted via bites from dirofilarial-carrying mosquitoes (*Culex, Aedes, Anopheles*). Recent reports have shown an increased number of human infections, leading some to consider dirofilariasis as an emerging zoonosis. Endemic regions include: Italy, Eastern and Southern Europe, Asia Minor, Central Asia, India, and Sri Lanka. Most of these cases in the United States occur in Florida, Texas, and Georgia. 

Most cases of human infection are asymptomatic. However, when symptomatic, dirofilariasis is characterized by subcutaneous nodules at the site of inoculation or lung parenchymal disease. Subcutaneous nodules near the eye are most often caused by *Dirofilaria repens*. Parks et al, presented a case report of migratory mildly tender subcutaneous nodules located on the lower extremity, abdomen, chest, and back. The migratory nodules lasted for 1-2 days before involution. Biopsy of one of the poorly defined nodules near the eye revealed the presence of filarial nematodes. 

"FIGURE 3. Onchocercoma. Courtesy of Karen Warschaw MD and Wilford Hall Medical Center teaching file."
nODULES showed *Dirofilaria tenuis*. Self-limited arthritis has also been reported with *D. tenuis* infection.22 Excision is the mainstay for diagnosis and treatment. Anti-helminthic medications are generally not required, although may play a role in treating dirofilarial rheumatism.26

**Trematodes**

**Schistosomiasis and swimmer’s itch**

Schistosomiasis is a tropical parasitic disease, caused by blood-dwelling trematodes (flukes). Schistosome cercariae (secondary larvae) are released by fresh water snails, their intermediate hosts, which then penetrate the skin of their definitive host (humans, birds, mammals) via proteolytic enzymes.29 The larvae then enter the blood stream, travel through the lungs, and then to the portal vascular system where they mature into adult schistosomes.30 After mating they migrate to the small blood vessels of specific destinations—*S. haematobium* infect the urinary system, while *S. japonicum* and *S. mansoni* infect the gastrointestinal tract. Female worms produce hundreds to thousands of eggs per day, which penetrate the bladder or rectum, and are excreted via the urine or feces.31 The eggs of each species have a characteristic size, shape, and spine—*S. haematobium* (oval, thin refractile wall, and delicate apical spine),32 *S. japonicum* (small round, thick refractile wall, and typically no visible spine),30 and *S. mansoni* (thick refractile wall and thick lateral spine).33

Schistosomiasis is mainly distributed in the tropical regions of Sub-Saharan Africa, Asia, and South America.34 The World Health Organization estimates that at least 90% of cases requiring treatment occur in Africa.35 Recently, the genus has been nearly eradicated from Japan and the Caribbean islands. In the US, outbreaks often occur in the north central states.

Swimmer’s itch (cercarial dermatitis) is typically caused by schistosome species with non-human hosts, such as birds and mammals (eg, *Trichobilharzia, Gigantobilharzia*, others). As a result, humans are dead-end hosts and the cercariae die after penetration into human skin, leading to a localized and self-limited cutaneous eruption.36 Swimmer’s itch is characterized by mild itch and a macular eruption about one to several hours after contact. Intensely pruritic papules and urticaria develop about 10 to 15 hours later. Symptoms affect only exposed skin and resolve within a few days to a week after water exposure.37

Other cutaneous manifestations include: Katayama fever (systemic hypersensitivity reaction several weeks post-infection, characterized by a flu-like syndrome, fever, headache, peripheral blood eosinophilia, pulmonary infiltrates, and urticaria),38 perineal and genital lesions (ova deposits within the skin and blood vessels manifesting as papular or verrucoid lesions),39 and ectopic cutaneous lesions (eg, adult worms migrating to the paraumbilical veins, presenting as abdominal papular lesions).40

Most of the complications associated with schistosomiasis are from chronic infection of eggs “trapped” within specific organs.41 The subsequent inflammation and fibrosis can lead to bloody diarrhea, abdominal pain, fatigue, anorexia, hematuria, dysuria, bladder infections, hydronephrosis, hepatosplenomegaly, portal hypertension, esophageal bleeding, and ascites.31

Treatment for schistosomiasis is a single dose of 40-60mg/kg of praziquantel. This is typically given in two or three oral doses of 20 mg/kg, and sometimes is repeated for 2 to 3 days.31 Efforts to control and prevent schistosomiasis have shifted away from environmental chemicals targeting snail populations, as these methods were often expensive and inefficient. Rather, the emphasis has shifted to mass treatment without individual diagnosis for at-risk communities.42

**Cestodes**

**Sparganosis**

Sparganosis is caused by infection of the larval stage of a pseudophyllidean tapeworm. The adult tapeworm lives in dog and cat intestines. The majority of human infections are due to *Spirocerca mansonioides*, but the disease can also be caused by *Sparganum proliferum*.43 Humans are infected most commonly through drinking contaminated water or ingesting infected intermediate hosts like snakes, fish, or amphibians. Infection also occurs in certain cultures, particularly in Asian countries, where intermediate hosts are made into pouticules and applied as treatment to wounds.44 Sparganosis is worldwide, but most common in Southeast Asia, China, and Japan.43

The most common clinical feature is a slow-growing, sometimes tender and migratory, subcutaneous nodule(s) that measures 1-3 cm in size. Case reports suggest that larvae can survive in human skin for up to 20 years.45,46 Patients can also have pruritus and urticaria that correlate to the release of toxins of a migrating parasite from the intestine to other tissues.47 Nonomura et al, described a case of sparganosis presenting as a periumbilical folliculitis with surrounding erythema.48 A rare form of fatal sparganosis, caused by *Sparganum proliferum*, is characterized by dissemination of parasites into multiple organs, including the skin (ulcerating cutaneous lesions), bone, brain, and other visceral organs.49

Treatment of sparganosis consists of complete excision. Medical therapy has typically been shown to be ineffective.46-50

**Cysticercosis**

Cysticercosis is caused by infection of the larval stage of *Taenia solium* (pork tapeworm). The adult form of *T. solium* resides in the intestinal tracts of pig and humans and is acquired by consuming raw or undercooked contaminated pork, or though consumption of food or water contaminated with eggs and proglottides of the parasite.50

*T. solium* living in human intestines causes minimal clinical symptoms. However, humans can become intermediate hosts, when larvae penetrate intestinal mucosa and form cysts in distant tissue (cysticerci). Cysticerci can develop in almost any organ, including: the heart, eyes, brain, lungs, liver, kidneys, skeletal muscle, and skin. Cysts in the central nervous system can cause serious sequelae including meningitis, hydrocephalus, and seizures. Neurocysticercosis is thought to be one of the leading causes of preventable epilepsy in the world.52,53 Cutaneous findings result from cysticerci in the muscle and subcutaneous tissue. These cysticerci present as single or multiple mobile, firm, subcutaneous nodules, with normal overlying skin and an average diameter of 1-2 cm.53,54 These nodules often escape clinical detection unless there is a heavy disease burden.55 They can range in number from few to over 1000 and are often asymptomatic, but can be painful in up to 20% of patients.56 Cutaneous cysticercosis has been reported to occur in about 54% of cases of cysticercosis, sometimes preceding neurologic involvement and thus can be instrumental in early diagnosis.53,57
Cysticercosis is prominent in poor areas of the world associated with pork raising and consumption, including Asia, Africa, and Latin America. Treatment consists of surgical excision of inactive and symptomatic cysterci, oral anti-helminths for active disease (albendazole 15mg/kg/day x 8 days, praziquantel 50mg/kg/day x 15 days), and interventions to stop transmission of the parasite (ie, improved sanitation, vaccinations for pigs, and meat inspection). Studies suggest that albendazole is more effective than praziquantel in treating neurocysticercosis.

Echinococcosis

Echinococcosis is caused by infection of the larval stage of tapeworms from the genus Echinococcus. E. granulosus (cystic echinococcosis) is the most common cause, but E. multilocularis (alveolar echinococcosis) and E. oligarthrus can also cause echinococcosis. The adult form of E. granulosus resides in the intestines of canids (dogs, wolves, foxes). Intermediate hosts (such as humans, sheep, and cattle) become infected by consuming or handling food or fur contaminated with infected feces. Echinococcosis occurs in sheep and cattle-raising areas, including South America, Asia, East Africa, Middle East, Lebanon, and Greece.

Clinical manifestations are typically due to mass affect from slow-growing cysts residing in any tissue and the disease can be fatal. The most common tissues affected are the liver (50%-75%) and lungs (20%). Bone and skin involvement is estimated at ~2% each. Enlarging cysts can cause jaundice, portal hypertension, cholangitis, hemoptysis, and anaphylaxis (from cyst rupture). Cutaneous signs are rare, but can present as firm, non-tender, subcutaneous nodules. Urticaria and pruritus can result from a hypersensitivity reaction to a ruptured cyst. Bresson-Hadni et al, described two cases of alveolar echinococcosis presenting with supraumbilical cutaneous nodules and/or painless fistulas. These cutaneous findings were caused by extension of larvae from primary liver lesions via the falciform ligament.

Treatment is primarily via surgical excision of cysts that are symptomatic or likely to rupture. Medical treatment is prolonged albendazole (10 mg/kg/day) for 8 weeks. Improved survival rates in cases of alveolar echinococcosis have been demonstrated when using albendazole up to 2 years following surgery.

References

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