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Changing Paradigms in Parasitic Infections: Common Dermatological Helminthic Infections And Cutaneous Myiasis

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Abstract. *Parasitic diseases caused by helminths, or worms, account for billions of human infections worldwide. Although most human pathogens caused by these organisms are infrequent in the United States, skin manifestations of parasitic diseases are being seen much more frequently in dermatologists' offices due to the increase in immigration and travel. Helminths are notable for their complicated lifecycles often including consecutive developmental phases, in separate hosts or in a free-living state. These parasitic organisms are usually macroscopic, multicellular organisms, and do not breed within their mammalian hosts. Notably, many helminthic diseases present with dermatologic signs and symptoms including skin nodules, cysts, migratory skin lesions, and pruritus. In this section, we discuss cutaneous myiasis as well as the four most common nematode dermatologic conditions seen in the United States.*

Most parasitic diseases are infrequent in the United States and are often considered exotic and unusual afflictions. They are, however, a major cause of morbidity and mortality worldwide, especially in the tropics and subtropics. With the increase of globalization and global warming, the potential increase of these diseases is inevitable. Although diseases such as ascariasis, cysticercosis, enterobiasis, hookworm disease, lymphatic filariasis, onchocerciasis, and schistosomiasis may only be vaguely remembered from medical school classes, skin manifestations of parasitic diseases are being seen much more frequently in dermatologists' offices due to the increase in immigration and travel. For many helminthic diseases, important diagnostic clues can be provided by skin examination. Cutaneous nodules and cysts, migratory skin lesions, and pruritus are all common signs of parasitic infections.[1]

Depending on the number and species of parasite, the immunologic response to the host, and the organs affected, a wide range of pathologic changes can occur with parasitic infections. Mechanical and irritant effects of the parasite can injure the host either by toxic products produced by the parasite or by means of the host immune response to the parasite.

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In this article, we present in depth the most commonly seen cutaneous manifestation of helminthic diseases seen in the United States. These include three roundworm infections, cutaneous larva migrans (CLM), strongyloidiasis (larva currens), and enterobiasis (pinworms), as well as cercarial dermatitis (swimmer's itch) from the phylum Platyhelminthes (flatworms). Additionally, a discussion of cutaneous myiasis, another parasitic disease that also can cause a migratory creeping eruption, is discussed.

Cutaneous Larva Migrans

Cutaneous larva migrans (CLM) is the general term applied to an infestation caused by various nematodes that produce characteristic migratory, pruritic, and serpiginous lesions or burrows. CLM has numerous synonyms including "creeping eruption," "creeping verminous dermatitis," "sandworm eruption," "plumber's itch," and "duck hunter's itch."

The condition is caused by the larvae of hookworms that are not normal parasites of humans, yet capable of penetrating human skin on exposure.[1-11] *Ancylostoma braziliense*, the dog and cat hookworm, is the most common cause, but other nematode species can also result in a creeping eruption. Other types of injurious nematodes includes *A. tubaeforme, A. caninum, A. ceylonicum, Uncinaria stenocephala, Bunostomum phlebotomum, Gnathostoma spinigerum, Dirofilara conjunctivae, Capillaria* species, *Strongyloides myopatami, S. papillosus*, and *S. wester*.[3] *Strongyloides stercoralis* may cause CLM; however, it is more commonly associated with larva

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Table 1. Causes of cutaneous larva migrans (CLM)[3]

Species	*Primary Host*	*Clinical Features*
Ancylostoma braziliense, A. tubaeforme	dog & cat hookworm	Thread-like, slightly raised, pruritic, migratory burrow; may persist for 3 months or more; moves 1–2 cm/day.
Ancylostoma caninum, A. ceylanicum	dog hookworm	Papular, rarely linear lesions; atypical CLM; disappears in 2 weeks.
Uncinaria stenocephala	European dog hookworm	Similar clinical appearance to *Ancylostoma braziliense.*
Bunostomum phlebotomum	cattle hookworm	Papules with a few mm migration; disappears in 2 weeks.
Strongyloides stercoralis (cause of larva currens)	human (strongylid)	Urticarial band in perianal or buttocks area; rapid migration up to 10 cm/h; chronic and intermittent for years.
Strongyloides animal species	Sheep, goat, cattle, horse, raccoon & nutria parasite (Strongylid)	Macule papular serpiginous lesions.
Capillaria species	Rodents, cats, dogs, foxes, poultry (whipworm)	Linear track; extremely severe pruritus.
Gnathostoma species	Cat, dog, pig & wild feline nematodes	Intermittent episodes of red, edematous, subcutaneous nodules; limited migration of individual lesions; may be recurrent for years.

currens that will be discussed later in detail (see Table 1).

Although most commonly found in tropical and subtropical areas, CLM has a worldwide distribution, including the Caribbean, Southeast United States, Central and South America, Africa, Scotland, England, and Southeast Asia.[3,8-11] In the United States, most cases are seen in vacationers returning from these endemic areas; however, CLM is found in the coastal areas of the United States from New Jersey to Texas, with the highest incidence in Florida.[7]

Cutaneous larva migrans is acquired by skin contact with the infective larvae that reside in the sand or soil. The larvae penetrate the skin but are restricted to the epidermis, where they wander aimlessly, unable to penetrate the basement membrane or complete their life cycle. In short, humans are a dead-end host for the larvae. The most common sites of acquisition are beaches, sandboxes, sand piles at construction sites, crawl spaces under houses, and contact with soil contaminated with animal feces. In Florida, dogs are prohibited from roaming on most beaches to prevent transmission of nematode larvae.[3]

The incubation period may vary with the species of hookworm, but the onset of symptoms usually ranges from 1 to 6 days after exposure. It is possible, however, that the larvae may rest or remain dormant in the skin for several weeks or months before migrating. The eruption is self-limited, normally lasting between and 8 weeks, coinciding with the death of the larvae organism. Rarely, a case may last over a year; however, there is some question whether or not reinfestaion may play a role in these unusual cases.

Clinically, CLM begins as a transient reddish papule appearing within a few hours after larval penetration. With time, the characteristic erythematous, slightly raised, and vesicular lesions, 2 to 4 mm wide, appear in a serpiginous track and reach 15 to 20 cm in length. The migratory nature of the lesion varies, as the various hookworm larvae can travel between a few millimeters to two centimeters per day. These cutaneous tracks can be singular or multiple. Less classic presentations of CLM are folliculitis and eczematouslike eruption.[9,10] Patients normally complain of intense pruritus, and pain also is frequently associated as well. The larva is found in normal skin 1 to 2 centimeters beyond the track. Anatomically, the most common site for creeping eruption is the feet, followed by the hands, arms, buttocks, and genitalia. Secondary bacterial infections may result in fever and usually yield *Streptococcus pyogenes*. Severe edema and cellulitis often mask the tracks, making diagnosis more difficult[3] (Fig 1, clinical picture of feet). The diagnosis of CLM is based primarily upon the characteristic clinical findings with a known epidemiologic exposure; however, eosinophilia is also a common clue. Epiluminescence microscopy has proved beneficial in surface identification.[12] The differential diagnosis includes scabies, tinea, cellulitis, contact dermatitis, erythema chronicum migrans, migratory myiasis, loiasis, and jellyfish or Portuguese man-of-war stings.

There are several treatments for cutaneous larva migrans, although two therapies are preferred. The first recommended treatment for CLM is oral ivermectin in a single dose of 200 μg/kg. (See Fig 1, A and B). This drug is available in the United States and many countries worldwide. It has an extensive safety profile and is well tolerated, with reported cure rates for CLM between 81% and 100%.[13-16] The second suggested treatment is topical thiabendazole solution or ointment at 10% to 15% applied topically 2 to 4 times daily for 2 weeks. This treatment may possibly be equally effective as a single oral dose of ivermectin, but compliance is an issue. Also, this preparation is not commercially available and requires compounding. Alternative treatments include systemic thiabendazole at 25 mg/kg a day for 2 days with cure rates of 68% to 84%, but is less well

tolerated than ivermectin given its high incidence of side effects, which include dizziness, nausea, and vomiting.[14] Additionally, oral albendazole at 400 mg a day for 3 days could be considered in the appropriate patient, with cure rates between 46% and 100%.[14,17] Older treatment with ethyl chloride spray, carbon dioxide slush, and liquid nitrogen are traumatic and unreliable.

Strongyloidiasis

Larva currens, "running or racing larvae," is a cutaneous manifestation caused by the intestinal infection or autoinfection with the human nematode, *Strongyloides stercoralis*, and very rarely *S. fulleborn*.[1-3] This parasitic nematode is found in feces-contaminated soil, primarily where the ground is moist, subtropical, or tropical. The organism may be free-living and infection occurs through skin penetration by filariform larvae that measure 500 × 16 μm. The migration of the larvae are much more rapid than in larva migrans, traveling up to 10 cm per hour.[3] The larvae enter the bloodstream of the host, at which time the rash subsides. The larvae then exit via the lungs, and ascend to the glottis to be swallowed. Maturation and reproduction take place in the upper small bowel, and rhabditiform larvae (noninfectious) hatch from ova within the gastrointestinal tract. These larvae may then transform into the infectious filariform, which are capable of reinfecting the host by either penetrating the intestinal mucosa (internal autoinfection) or the skin (external autoinfection).

The cutaneous lesions are commonly seen in the perianal and perineal areas; however, occasionally the abdomen, buttocks, and thighs are involved. Eosinophilia, gastrointestinal complaints, and Gram-negative bacteremia are commonly present. Since this parasite can complete its life cycle in humans, larva currens can exist for a long time, leaving the host quite debilitated.[2,3] A high mortality of about 86% is seen in these patients.[3] Other conditions such as immunocompromised states, especially HIV/AIDS, chronic steroid use, leprosy, burns, achlorhydria, nephrosos, and institutionalization predispose patients to chronic infections and hyperinfection.

Identifying *S. stercoralis* ova or larvae in the stool is diagnostic. Multiple fecal samples may be necessary, since the excretion of the larvae tends to be sporatic. The enzyme-linked immunosorbent assay (ELISA) and immunofluorescent antibody assays may be useful in nonendemic situations. Biopsies are only useful in hyperinfection, when larvae are abundant.

Oral ivermectin, (Stromectol®), has been approved by the United States Food and Drug Administration (FDA) for the treatment of strongyloidiasis and onchocerciasis. Ivermectin is safe and effective in patients with HIV/AIDS. Oral ivermectin treatment for other parasitic and ectoparasitic diseases in the United States is commonly used "off label." Oral thiabendazole, 25 mg/kg bid for 2 to 5 days or a 2 day-course repeated 1 week later, is the next choice of therapy. Mebendazole, 100 mg bid for 4 days, and albendazole, 400 mg daily for 4 days, can also be used but cannot compare to ivermectin in efficacy or patient compliance.

Enterobiasis (Pin Worms)

The most common helminth infection in the United States and Western Europe, especially among school-age children, is enterobiasis, infection with pinworms. Its distribution is worldwide, and it has no association with any particular socioeconomic level, race, or culture. Synonyms include pinworm or "seatworm infection," oxyuriasis, and "threadworm." Over 10 years ago, the estimated prevalence of this condition in the United States was reported to be as high as 42 million, but is probably much higher than that now.[18] Prevalence rates in children are reportedly as high as 61% in India,[19] 50% in England,[20] 39% in Thailand,[21] 37% in Sweden,[22] and 29% in Denmark.[23] Finger sucking increases the incidence and relapse rates for this infestation.[24] Man is the only host for the causative nematode, *Enterobius vermicularis*.

Enterobiasis is usually acquired by the ingestion of infective eggs by direct anus-to-mouth transfer by the fingers. This can occur with nail biting, poor hygiene, or inadequate handwashing.[25] Alternatively, one can become infested from contaminated bed linens, clothing, or bathroom fixtures, or inhalation and ingestion of dust containing airborne eggs that are dislodged from bed linens and clothes. Viable eggs and larvae of *Enterobius vermicularis* have been reported in the sludge of sewage treatment plants.[26]

After ingestion of ova, the larvae hatch in the duodenum and develop in the cecum through two molts into adulthood within 1 to 2 months. Adult pinworms attach to the mucosa of the cecum, appendix, and nearby bowel, living up to 13 weeks. The female *E. vermicularis* are threadlike, white worms, 5 to 13 mm in length and 0.5 mm in width. They migrate nightly to the perianal area to deposit their eggs, which remain viable for up to 2 weeks. As a consequence of this nocturnal migration, some worms find their way into adjacent orifices, most commonly the female genitourinary tract, where they may produce symptoms such as vulvovaginitis and postmenopausal bleeding.

A single female pinworm may lay up to 16,000 eggs.[27] The eggs are ovoid, 55 × 25 mm, flattened on one side, and embryonate in 6 hours. These eggs can remain viable for 20 days in a moist environment. By a process called retroinfection, some of the pinworm larvae that hatch on the anus may return to the gastrointestinal tract of the original host, causing a heavy parasite load, as well as insuring continued infestation. It is

very common for several family members to be infested with pinworms at the same time. All close household contacts, even if asymptomatic, should be treated to prevent possible reinfection.

The incubation period before symptoms appear from enterobiasis is weeks to months after ingestion of infective eggs. Four to six weeks are necessary before eggs can be found in the perianal and perineal areas. The worm itself lives for a few months in humans; however, autoinfection by anus-to-mouth due to lack of hand washing hygiene allows the infestation to continue indefinitely, and reinfections are common. One third of infested individuals are totally asymptomatic.[25]

The most common presentation of pinworms is perianal or perineal pruritus. The itching is usually most intense at night. The skin can be excoriated and can become secondarily infected. Other symptoms attributed to enterobiasis are anorexia, abdominal pain, irritability, restlessness, insomnia, dysuria, enuresis, and in females, vulvovaginitis and vaginal discharge. For example, 36% of young girls with urinary tract infections have been found to additionally have pinworms.[28] Infrequently, ectopic infection results in vaginitis, endometritis, salpingitis, epididymitis, urethritis, pelvic inflammatory disease, or pyelitis.[29,30] Infection with *Enterobius vermicularis* has caused perforation of the ileum, tubovarian abscesses with cytologic detection of eggs and worms, eosinophilic ileocolitis, and in some cases, enterocutaneous fistulas.[31-34] A case of pinworms causing a subcutaneous abscess was confirmed by fine-needle aspiration.[35] Helminth infestations are seen in over 27% of acute appendectomies, suggesting the need of helminthological examination and etiotropic treatments in these patients.[36] After laparoscopic appendectomy, careful evaluation of the appendix stump and careful thermal desiccation and removal of pinworms, if present, should be considered when using the endoloop technique.[37]

The diagnosis is usually made by clinical history, although identification of the *E. vermicularis* eggs can be obtained from the perianal area. This is achieved by applying a clear adhesive cellophane tape to the anal area in the early morning before defecation or bathing. The eggs, which are naturally transparent and colorless, stain deep blue with Lacto-phenol cotton blue, assisting in detection and identification.[38] The threadlike, light-yellowish white adults with a long, thin sharply pointed tail, may be seen in the stool or on toilet paper. Rarely are ova or larvae detected in the stool or urine. Eosinophilia only occurs in invasive disease; otherwise, all laboratory studies are normal.

The differential diagnosis of rectal symptoms includes irritant dermatitis, allergic dermatitis, idiopathic pruritus ani, tinea, bacterial infection, lichen simplex chronicus, and strongyloidiasis.

The drug of choice is mebendazole 100 mg in a single dose and repeated in 1 week. It is safe and effective, with a cure rate of 96%.[25] Other therapies effective for pinworms include a single dose of pyrantel pamoate, 11 mg/kg up to a maximum of 1 g, repeated in 1 week. Second-line drugs include: piperazine citrate, from 65 mg/kg to a maximum of 2.5 gm for 7 days; a single dose of pyrvinium pamoate, 5 mg/kg with maximum dosing at 350 mg; and albendazole given as at 400 mg in a single dose. With all treatment programs, reinfection is frequent, and treatment of the entire household is suggested whether or not symptoms are present.

Cercarial Dermatitis or "Swimmer's Itch"

Cercarial dermatitis, commonly known as "swimmer's itch", is a distinctive papular eruption which occurs when the skin of humans is penetrated by cercariae of nonhuman schistosomes (trematodes).[3] Other common names for this condition are "clam digger's dermatitis," "schistosome dermatitis," and "sedge pool itch." The normal hosts of these cercariae are birds, rodents, and other small mammals. There are approximately 20 species of these flukes worldwide that have been implicated in this human disease from various species including *Schistosoma, Ornithobilharzia, Gigantobilharzia, Austrobilharzia, Trichobilharzia,* and *Orientobilharzia*.[3]

The life cycle of the schistosome requires an intermediate mollusk host and a fresh water aquatic environment for disease transmission. It begins with eggs produced by the adult, being shed with the animal feces of the host into the environment. These eggs hatch on reaching water, and release larvae. These larvae seek their intermediate snail host, to which they penetrate and mature as sporocysts in their digestive tract. After 4 to 6 weeks they emerge from snails as cercariae. At this time, they must penetrate the skin of a bird or small mammal to proceed with further maturation. Man is an accidental host. Besides thermotactile stimulation, these organisms follow nonselective chemo attractants such as cholesterol and ceramide, which facilitate the sticking of the sucker of the flatworm to the host's skin.[39] The cercariae often begin penetration of intact human skin as the skin dries after leaving the water. The nonhuman schistosomes incite a greater inflammatory reaction from penetration, although this varies among individuals.[3] A "prickling sensation" occurs minutes after exposure to infested water and may last for several hours. This sensitization develops to enzymes produced by the cercariae and is probably the cause of the inflammatory reaction seen clinically. Since humans are an accidental host and the larvae cannot complete their life cycle, the cercariae die in the human epidermis within 24-48 hours. The clinical disease is limited to the exposed surfaces of the body.

Cercerial dermatitis is a potential hazard worldwide, wherever people share an aquatic environment with

vertebrates and mollusks harboring schistosomes. In some regions such as in numerous lakes in Michigan, Wisconsin, and Minnesota, it is endemic. The development of swimmer's itch is significantly associated with exposure to lake water in the area of the lake with the highest algae content and shallowest depth.[40] The peak release of cercariae from snails occurs from May to October in lakes in the northern hemisphere.[41] More cercariae tend to emerge in the morning hours, and are more concentrated in the shallow water regions of lakes. Additionally, warmer temperatures encourage a higher release of cercariae. Cercarial dermatitis is usually associated with fresh water lakes, ponds, streams, irrigation ditches, snail infested wells, and rice paddies, but the condition also can be seen in brackish water inhabited by the appropriate mollusk hosts. Swimmer's itch has also been contracted via contact with cleaning aquariums.[42,43] The disease is most often seen in summer vacationers; however it is often commonly seen in duck hunters, fishermen, and in people with occupations that require exposure to water, such as clam diggers and rice farmers.

Clinical features develop about an hour after exposure to cercariae-infested water and present with pruritic erythematous macules, which can last for several hours. Papules, papulovesicles, purpura, and wheals often ensue 10 to 15 hours later, associated with accentuated pruritus. Thereafter, the classic signs of discrete, highly pruritic papules surrounded by a zone of erythema are noted. Classically, the distribution of the eruption spares parts of the body covered with clothing and involves only skin surfaces directly exposed to water. Edema, secondary bacterial infections, lymphangitis, and regional adenopathy develop in some individuals, as may eosinophilia and systemic symptoms of generalized urticaria, nausea, and vomiting. Humans reveal a varied degree of sensitization to cercariae, with repeated exposures to the organism normally producing more severe cutaneous reactions.[3] Postinflammatory hyperpigmentation is a common sequelae.

The diagnosis of this condition is made from characteristic history and clinical findings with exposure to the appropriate epidemiologic setting. Although some work has been done using serologic tests to confirm the diagnosis[44] and DNA probes for the exact identification of larvae have been developed,[45] these tests are not routinely available. Obviously, documenting cercariae in the incriminated waters supports the diagnosis.

A differential of swimmer's itch includes seabather's eruption, plant dermatitis, insect bites, folliculitis, contact dermatitis secondary to marine plants, varicella, viral examthems, human schistosomiasis, hydroids, and corals, as well as the various causes of urticaria.

The condition is self-limited and treatment is oriented toward the relief of symptoms. Oral antihistamines, topical steroids, and topical antipruritic agents may be useful in relieving pruritus and discomfort. An occasional patient may require a short course of oral steroids. If contact with infested waters occurs, toweling off vigorously and showering promptly after such exposure may greatly limit penetration of the cercariae. As a preventive measure, wearing tightly woven clothing or applying of a greasy thick coating of unguent offers some protection.

Copper salts, niclosamide, formaldehyde, and other helminthicides and molluscacides have been suggested to control snail populations in ponds and small lakes; however, concerns as to adverse effects such treatments would have on the environment have greatly limited their usage. Another effective method to control swimmer's itch is to disturb the epilithic habitat of snails using a boat-mounted rototiller or tractor and rake in areas of high snail concentration in shallow areas of the lake during the breeding and early developmental phase of the snail.[46]

The authors query whether infective larvae may migrate in the body via the circulatory system. The possible residence of these parasites within other organs of humans is a controversial topic.[47] Data from other animal models indicate that cercariae transform into schistosomula and migrate within the mammalian host, especially to the lungs.[48] These worms can survive for days within mammalian organs and feed on red blood cells.[48] In short, the life cycle and adaptations of these cercariae to various vertebrates including humans still requires more study to better disclose this hidden area of parasitology.

Seabather's Eruption

Because seabather's eruption is often confused with swimmer's itch, we mention it here, although it more appropriately should be categorized among stinging marine invertebrates rather than parasitic infections.[3] (See Table 2). Seabather's eruption, also known as marine dermatitis and incorrectly termed "sea lice," is a highly pruritic cutaneous eruption that occurs primarily under swimwear after bathing in the ocean. Most reported cases are from the Southeast coast of Florida, Cuba, Mexico, and in the Caribbean[49-53]; however, outbreaks have occurred 1000 miles north in Long Island.[54] Outbreaks occur between March and August, with a peak incidence in May, usually falling around Memorial Day weekend, South Florida's most popular beach weekend of the year.[52] Conservative estimates reveal that over 10,000 persons developed seabather's eruption during a 12-month period in Florida. Interviewing 735 beach swimmers in Palm Beach County, Florida revealed a 16% incidence of seabather's eruption.[49]

Although this highly pruritic condition was first reported in 1949 by a Miami dermatologist,[55] it took

*Table 2. Characteristics of cercarial dermatitis, seabather's eruption, and "sea lice"**

	Cercarial Dermatitis "Swimmer's Itch"	*Seabather's Eruption Northern*	*Seabather's Eruption Southern*	*"Sea Lice"*
Location	Worldwide distribution, highly endemic in Canada & Great Lakes, & U.S. Northern mid-west lakes	North Atlantic coast from Massachusetts to South Carolina	Southern Atlantic coast, especially S. Florida & the Caribbean	Worldwide distribution
Type of water	Fresh (predominantly), occasionally salt water tributaries	Salt (ocean)	Salt (ocean)	Salt (ocean)
Affects	Humans	Humans	Humans	Fish
Skin involved	Exposed	Covered	Covered	N/A
Etiology	*Schistosome cercariae,* usually of birds, rodents, or ungulates	Cnidarian larvae of the sea anemone, *Edwardsiella lineata*	Cnidarian larvae of the thimble jellyfish, *Linuche unguiculata*	Parasitic crustaceans

* *Adapted from Wong, Meinking, Rosen, et al. (ref. 52).*

almost 45 more years before the cause was found.[52] In the spring and summer of 1992, people swimming in the ocean in South Florida and the Caribbean had the most severe cases and highest incidence of Seabather's eruption ever documented.[52] Organisms collected from ocean water infested with Cnidarian larvae were grown to maturity at the University of Miami School of Marine and Atmospheric Science. A month after Hurricane Andrew hit South Florida, the larva had matured into very small thimble jellyfish.[52] There has been a lot of interest and discussion as to whether or not the high incidence of Cnidarian larvae in the spring and summer of some years might correlate to hurricane activity later in the year. The University of Miami researchers continue to follow the Cnidarian larvae activity each year for a possible pattern.

It is now known that Seabather's eruption is caused by the stinging nematocysts of the larvae of the phylum Cnidaria (formerly Coelenterate), which includes jellyfish, Portuguese man-of-war, sea anemones, hydroids, and fire coral.[52] The thimble jellyfish, *Linuche unquiculata*, has been found to be the cause in waters off the coast of Florida and in the Caribbean.[50-52] Cases of seabather's eruption reported off the coast of Long Island and in the North Atlantic coastal states occur from August through October and are caused by the larval form of the sea anemone, *Edwardsiella lineata*, which has not been found any further South than the Carolinas.[52,54]

Cnidarian larvae are pinhead-sized at 0.5 mm, and range from black to greenish brown. They are usually float about 6 inches below the surface or close to the surface of the water and get trapped inside of the swimming suit while the person is in the ocean. Any external pressure on the larvae, or changes in osmotic pressure caused by evaporation, or exposure to fresh water provokes the release of the coiled nematocysts, which fire irritating toxins into the skin. Examples of eliciting conditions include mechanical pressure from clothing or weight belts and other dive gear, lying on a surfboard or sitting on the beach in a wet bathing suit, or from osmotic variances occurring upon drying of the swimwear, or upon rinsing off with freshwater while continuing to wear the bathing suit.

Exposure to Cnidarian larvae is associated with stinging or prickling under swimwear upon exiting the ocean. Lesions appear within a few hours of exposure and consist of pruritic erythematous papules and wheals, often developing into pustules or vesicles. Affected individuals often complain of itching, burning, and pain. Resolution is spontaneous after a few days during which a slight serous crust appearing as a central punctum is noted. A delayed hypersensitivity reaction may occur after 10 days and can be more severe, extending to exposed areas of the body not previously affected.[52] Especially in children, systemic symptoms may accompany the rash including fever (101°–104°F), nausea, vomiting, diarrhea, headache, weakness, muscle spasms, and malaise.[52]

While some of the stinging sensation may be due to nematocyst activation, immunologic hypersensitivity occurs, as ELISA tests on serum from affected persons show specific reactivity to the organism and positive patch tests to antigens have demonstrated cell-mediated immunity involvement.[50,52,56] Interestingly, some persons appear to be immune to seabather's eruption despite frequent exposure.[52] Blocking antibodies may be the source of this apparent immunity.[56]

Unlike swimmer's itch, which is found on the exposed areas of the body, seabather's eruption is found on the unexposed parts of the body that are covered by the bathing suit, T-shirt, shorts, or shirt. Swimmer's itch can occur after exposure to fresh or brackish water, whereas sea bather's eruption only develops from salt-water exposure. Swimmer's itch is more often found in the Northern United States and Canada; sea bather's eruption is concentrated in Florida and the Caribbean. Cercarial forms of schistosomes cause swimmer's itch,

whereas larval forms of marine coelenterates produce seabather's eruption. (See Table 2)

The differential diagnosis for seabather's eruption is identical to swimmer's itch and includes insect bites, folliculitis, contact dermatitis secondary to marine plants, varicella, viral examthems, human schistosomiais, hydroids, and corals, as well as the various causes of urticaria.

The cutaneous reaction from seabather's eruption resolves spontaneously; treatment of seabather's eruption is symptomatic with antipruritic lotions, high potency topical steroids, antihistamines, and oral steroids if indicated.[3,52]

Preventive measures include removing swimwear before showering and not wearing T-shirts in the water. Scantier swimwear would have advantages. Whole body Lycra swimsuits and wetsuits with restrictive cuffs can be protective. Additionally, bathing suits should be washed in fresh water and detergent and dried before reuse; otherwise, unreleased nematocysts will fire, producing the eruption without additional exposure to ocean water treatment.

Numerous agents have been used in attempts to inactivate nematocysts, including isopropyl alcohol, vinegar, papain, and baking soda.[52]

Myiasis

Myiasis is an infestation of the skin of mammals and humans by developing larvae (maggots) of a variety of fly species within the arthropod order of Diptera. The three main clinical types are wound, migratory, and furuncular (or follicular) myiasis. Larvae inhabit skin wounds or burrow into the dermis, causing boil-like lesions. Historically, fly larvae, and more specifically maggots, were central to Francisco Redi's experiment rejecting the theory of spontaneous generation. Maggots have been used therapeutically for surgical debridement since Civil War times for the antibacterial properties of their midgut flora.

Myiasis is a worldwide infestation with seasonal variation the prevalence of which is related to latitude and the life cycle of the various species of flies. Higher incidences are in the tropics and subtropics of Africa and the Americas. The disease-producing flies prefer a warm and humid environment, thus myiasis is restricted to the summer months in temperate zones, while being year-round in the tropics. A recent outbreak in an American hospital was accredited to a massive mouse infestation during the summer months.[57]

Myiasis can be caused by several species of arthropods of the order Diptera, the two-winged true fly, which for a period of time, feed on the dead or living tissue of the host, liquid body substances, or ingested food. Two approaches to classification of the causative flies are by the various taxonomic families or by etiologic differences in behavior of the various species of flies. The most common flies worldwide causing human infestation are *Dermatobia hominis* and *Cordylobia anthropohaga*.

Route of transmission to human hosts of fly larvae differs among the various varieties of flies. For example, *Dermatobia hominis* (the human botfly) lays her eggs onto mosquitos that in turn deposit them onto a warm-blooded mammal. *Cordylobia anthropophaga* (the tumbu fly) deposits her eggs on moist clothing, soiled blankets, and in sand. The larvae can live 15 days without feeding, but once it makes contact with a host, it penetrates the skin, which initiates further maturation. In endemic areas, people usually iron their clothes and horse blankets after being hung out to dry to kill the fly eggs. In wound myiasis, any open wound or orifice may attract flies to deposit their eggs in the immediate vicinity. Any body area could be infested, with the most serious potential sequelae occurring when the infestation involves the nasal cavity and sinuses. The incubation period for larvae to maturate into adulthood depends on the fly and can range from 1 to 12 weeks.

Furuncular cutaneous myiasisis, which is caused by both the human botfly and the tumbu fly, causes furunculoid (boil-like) lesions. After hatching, the larvae descend into the skin and remain stationary. A pruritic papule occurs with larval infestation within 24 hours after contact, which enlarges to 1 to 3 cm in diameter and almost 1 cm high. These lesions can be painful and may become crusted and purulent. There is typically an opening at the top of these boil-like swelling for entry of oxygen. Via this opening, the larva with its characteristic black, backward-pointing spines that anchor it in the host's skin and make removal difficult, can be seen. After 2 to 3 months, the larvae leave the skin and drop to the ground to pupate. Orbital, ear, and nasal myiasis can cause extensive necrosis, sloughing, and destruction of tissue and need immediate removal.[58,59]

Another common presentation of this infestation is wound myiasis in which flies deposit larvae in a suppurating wound or on decomposing flesh. *Cochliomyia hominivorax*, a screwworm, is the most common type of this form of myiasis in the Americas, while *Chrysomyia bezziana* is typically seen in Africa, Australia, and Asia. The normal mode of contact is with an infested domestic animal. Attracted to the wound, a female fly may lay as many as 300 eggs in a few minutes at the site. The congregation of larvae matures within 1 to 3 weeks. Although many of the larvae will decompose within the tissue or drop to the soil to pupate, some may burrow deeply into living tissue. The diagnosis is obvious when larvae are visible on the surface and more difficult when they have burrowed beneath the surface.

Creeping (or migratory) cutaneous myiasis may be caused by *Hypoderm bovis*, if exposed to infested cattle,

or by *Gasterophila intestinalis* in stable help working with horses or donkeys. This form of myiasis resembles cutaneous larvae migrans, as the organism is not stationary, and wanders in the skin tissues, exciting a mobile pattern of inflammation. Migratory myiasis is usually differentiated from cutaneous larva migrans from hookworms by three clinical features. Firstly, igratory myiasis is generally more restricted and extends more slowly. Secondly, larvae are larger and can generally be visualized. Lastly, botfly larvae can survive for months in human skin, unlike hookworms.

Myiasis is a self-limiting infestation, with the vast majority of cases having minimal morbidity. Indeed, the main reason for treatment is usually for a person's psychologic satisfaction of removing a maggot from his or her body. Some larvae, such as *C. hominivorax*, can occur near body orifices of the head with possible burrowing to brain tissue; some cases of myiasis therefore can be quite dangerous to the host.

Cutaneous myiasis needs differentiation from cellulitis, furunculosis, leishmaniasis, onchocerciasis, skin abscess, subcutaneous cyst, adenopathy, and tungiasis. In terms of diagnostic testing, ultrasound can assist in the diagnosis, determining the size of larva and course of treatment.[60,61]

The larvae in furuncular cutaneous myiasis should not be forcibly removed through its punctum because its tapered shape with rows of spines and hooks prevents simple extrusion. Surgical debridement under local anesthesia is curative, although a foreign body response can occur if parts of the larvae remain. Given that myiasis can be a portal of entry for *Clostridium tetani*, vaccination should be considered with this infestation.[62]

Occlusion and suffocation approaches include petroleum jelly, liquid paraffin, beeswax, nail polish, and heavy oil placed over the central punctum. Application of lard or a strip of bacon taped over the site also coaxes the larva to emerge spontaneously headfirst over the course of several hours.[63] Alternatively, an inexpensive, disposable, commercial venom extractor has also proved efficacious.[64] Such blockage forces the aerobic larvae to reposition into an upward position so that its backward-pointing spines are in position to cause minimal tissue damage if then grabbed with tweezers, or forceps, and removed from the skin's surface. Ethyl chloride sprays, liquid nitrogen, chloroform in vegetable oil, or insecticides have been used alone or in combination; however, the success rate of these methods and possible damage to normal tissue is unknown. Additionally, lidocaine can be injected at the base of the tissue cavity in which the larva inhabits, thereby assisting the larvae to surface. After removal of larvae, antiseptic dressings are indicated as well as an antibiotic if secondary infection is present.[65]

Wound myiasis requires debridement with irrigation to remove larvae from the wound or surgical removal. If burrowing has occurred, the method of extraction is identical to that for furuncular cutaneous myiasis. An alternative treatment for all types of myiasis would be oral ivermectin that alters larval development.[66] Surgical removal would not usually be required unless requested, as the larvae are naturally sloughed off over the ensuing 2 weeks.

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