CHAPTER 37

Ectoparasites, Cutaneous Parasites, and Cnidarian Envenomation

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This chapter describes common infestations by ectoparasites, including mites, lice, ticks, creeping eruption (cutaneous larva migrans), maggots (myiasis), and jiggers (tungiasis). Waterborne swimmer's itch (cercarial dermatitis) and jellyfish, or cnidarian ("nye-*dare*-ee-uhn"), envenomation are also discussed.

Living in close quarters, hiking, camping, visiting beaches, and swimming can increase exposure to many of these organisms. Avoiding contact with sand and soil, using chemical insect repellents, wearing long sleeves, pants, and shoes, as well as using bed nets, can aid in preventing many of these diseases.

ECTOPARASITES

Scabies

Etiology

Scabies is caused by the nearly microscopic human "itch mite" *Sarcoptes scabiei* var. *hominis*. Scabies causes intense pruritus, generally developing several weeks after infestation, with itching that worsens at night. Scabies is spread by skin-to-skin contact and rarely via contaminated clothing, bedding, or other fomites. The scabies mite lives for about a day away from a human host.

Mites burrow into the skin's outer layer and lay 2-4 eggs along the burrow path each day. A single female can lay up to 40 eggs. The time from ovum to mature mite is about 10-14 days. This cycle can repeat indefinitely, though some people can clear the infestation through their own immune response.

Epidemiology

Scabies occurs worldwide and is epidemic in much of the tropics. People of all ages are affected. Prevalence in the developing world is estimated at 10% in the general population and up to 50% among children. Crowding is a likely important factor, because scabies is highly contagious through skin-to-skin contact.

Clinical Features

In most people, lesions and pruritus develop several weeks after infestation due to a delayed-type hypersensitivity immune response to the mites, ova, saliva, and/or feces. Secondary papules, pustules, and/or vesicles are often present. The distribution of lesions includes finger webs, flexor surfaces of the wrists, axillae, breasts, umbilicus, genitals, buttocks, and feet. Burrows are 3-5 mm, threadlike, linear lesions seen typically in the finger webs, on the wrists, and on the glans penis. Secondary bacterial infection is common, and impetigo occurring in the aforementioned distribution suggests scabies.

In temperate climates, the face and scalp are spared except in infants and the elderly. In the tropics, the scalp and face may be involved; secondary infection is more frequent; and burrows are often absent. Crusted scabies (formerly called "Norwegian scabies"), in which thousands to millions of mites are present on an immune-compromised host, may occur anywhere on the body, although an acral distribution is common. Hyperkeratotic plaques and crusts predominate, resembling psoriasis. Burrows are obscured by overlying crusts, and, surprisingly, pruritus is often mild or absent.

Diagnosis

Identification of burrows in a typical distribution can make a clinical diagnosis. But burrows are not always present and are less common in tropical climates. Exposure history and typical distribution of lesions can also be used to make a clinical diagnosis.

A definitive diagnosis of scabies is made by identification of the mite, eggs, or feces in skin scrapings. Take scrapings from burrows whenever possible: with a scalpel blade coated with mineral or immersion oil, scrape the lesions firmly enough to cause pinpoint bleeding. Place the scrapings on a slide with a coverslip and then examine under a low-power objective lens. Sensitivity is dependent on experience; failure to find evidence of mites cannot rule out scabies.

Treatment

All household members and intimate contacts should be treated simultaneously, even if asymptomatic, to avoid reinfestation.

- Permethrin 5% cream offers a high cure rate and minimal toxicity. It is safe for infants >2 months old. Apply 30-50 mL from the neck down, leave 8-12 hours, then wash. One treatment is generally effective, but it can be reapplied after 5-7 days.
- Lindane 1% is a second-line topical treatment but is banned in some countries due to neurotoxicity presenting as seizures and neuromuscular rigidity, usually affecting children or elderly patients. It is applied like permethrin. Avoid using in infants and young children; it is not recommended for women who are pregnant or nursing.
- Ivermectin is an oral alternative with increasing popular use and is well tolerated. A single dose of 200 μg/kg body weight is as effective as lindane, and two doses, 2 weeks apart, is as effective as permethrin.
- An alternative treatment for infants <3 months includes 10% crotamiton cream, applied on two consecutive nights and washed off 48 hours after the second treatment. A second treatment in 2 weeks may be given.
- 5-10% sulfur ointment is commonly used in Africa and South America; it is safe for infants and is the treatment of choice for pregnant women.

Clothing and linens should be washed in hot water at the time of treatment. Nonwashable clothing should not be worn for 3-5 days. Household fumigation is not necessary.

Lice

Etiology

Head lice (pediculosis capitis) and body lice (pediculosis corporis) are caused by the human louse *Pediculus humanus*, a light-gray insect, 3-4 mm long, that feeds on blood. The head and body subspecies are practically identical, with distinct patterns of infestation. All forms of lice require a human host to survive and would die after 2-3 days without feeding.

Female head lice live on the scalp for 3-4 weeks and lay about 10 eggs, or nits, a day. Nits are deposited on the base of the hair shaft and incubate for 7-12 days; female nymphs mature in roughly another 10 days. The total number of adult lice is usually less than 10 at any time, and the infestation is primarily at the back of the head. Body lice live in the seams of clothing and move to the body transiently to feed.

Phthirus pubis lice, or pubic lice, are 1-2 mm and brown in color. Pubic lice live on pubic hair but may be found on any body hair and on eyelashes and eyebrows. Nits take about a week to hatch and another 2-3 weeks to mature.

Epidemiology

Head lice are found worldwide, are common among children across socioeconomic classes, but are associated with poor hygiene and crowding when present on adults.

Body lice occur worldwide in conditions of poor hygiene, especially where clothing is not changed and washed regularly. Transmission occurs through close body contact or sharing infested clothing. Lice may live for several days on clothing or bedding, but routine laundering will kill them. Body lice are vectors for typhus (*Rickettsia prowazekii*), trench fever (*Bartonella quintana*), and relapsing fever (*Borrelia recurrentis*).

Sometimes called crabs, pubic lice are primarily transmitted sexually and also endemic worldwide. In regions where pubic hair shaving is common, this condition has become very unusual.

Clinical Features

Head lice cause pruritus of the scalp, and scratching leads to excoriations. Further complications include furunculosis or impetigo. Scalp pyoderma should prompt an inspection for head lice.

The hair is often matted or lusterless, and regional lymph nodes may be enlarged. Light-gray adults and nymphs can be seen crawling among the hair and nits are usually apparent on examination. Nits are 0.5-mm ovals cemented to individual hairs where the shaft emerges from the scalp and grow out with the hair. Nits are initially translucent near the base of the hair shaft, where they are first laid, but after they hatch, when they have grown about 1 cm from the scalp, they appear white.

Body lice produce generalized pruritus, with excoriations usually worse over the back, shoulders, and arms. In contrast to scabies and head lice, the head, hands, and feet are spared. Typical lesions are excoriated papules with or without secondary bacterial infection. Lice and nits are not found on the body but may be seen in clothing.

Pubic lice cause pruritus from the umbilicus to the mid-thighs, most severely in the pubic area. Finding the tiny lice or nits may require a careful search. Often, no skin lesions are present, but excoriations or small bluish macules, called maculae caeruleae, are seen occasionally. These are thought to be hemosiderin deposition from bite trauma.

Diagnosis

For travelers in tropical areas, it is important to distinguish nits from white and black piedra, a fungal infection that can appear similar to nits (Chapter 38). White piedra loosely adheres to hair shafts, as opposed to nits, which are strongly attached. White piedra tends to affect the axillae, groin, and face more often than the scalp. Black piedra commonly occurs in the scalp and facial hair, can be similarly sized to nits, and is strongly attached to the hair. Visualization with a KOH preparation and microscopic inspection will reveal characteristic septate hyphae, distinguishing black piedra from lice.

Lice are diagnosed by finding the louse or nits. The use of a hand lens may aid diagnosis.

Treatment

Head lice

All topical treatments should be reapplied at about 7 days to prevent reinfestation.

- 10-minute application of 1% permethrin shampoo, which is repeated at 7 days.
- 0.5% malathion lotion left on for 8-12 hours, used in cases of permethrin resistance (not for use in children under the age of 2)
- For those who fail to respond to topical treatment, drug resistance may be the cause; in this case, use ivermectin (single dose of 200 μg/kg body weight) and ensure that all close contacts have been treated as well.
- Bed linens and hats should be laundered or dry-cleaned.

Body lice

- Bathe the body and wash all clothing or apply insecticide powder to the inner surface of clothing. DDT powder or 1% malathion powder is effective.

Pubic lice

- 10-minute application of 1% permethrin shampoo from the axillae to the thighs
- 10-minute application of 1% pyrethrin with piperonyl butoxide, as above
- 0.5% malathion lotion left on for 8-12 hours
- Sexual partners should be treated simultaneously to prevent reinfestation. Sexual partners from the previous month should be contacted. Evaluate patients for other possible sexually transmitted infections.
- Eyelashes cannot be treated with the above chemical remedies. In this location treatment is with thick application of petrolatum or other occlusive ointment twice a day for 10 days, accompanied by mechanical removal of lice and nits.

Topical 0.1% triamcinolone or other low-potency topical steroid may be used for symptomatic itch relief. In all cases bacterial superinfection should be considered and treated where appropriate.

CUTANEOUS LARVA MIGRANS

Cutaneous larva migrans is caused by invasion into the skin by the larvae of animal hookworms.

Etiology

The most common cause is the larva of the dog and cat hookworm, *Ancylostoma braziliense*. Eggs, passed in the stool of the animal, mature into infective larvae in the soil. These larvae penetrate the skin of humans and produce serpiginous lesions by burrowing aimlessly through the skin. They are not able to penetrate deeper and continue their life cycle in humans, so we are dead-end hosts. If untreated, the larvae usually die within 2-8 weeks.

Epidemiology

The disease occurs in warm and humid conditions throughout the world. Infections peak during rainy seasons. The most important risk factor is skin exposure to soil contaminated with dog or cat feces. In travelers, this typically occurs when walking barefoot, such as on beaches. Feet are most often affected, but any skin exposure to contaminated soil can result in infection.

Clinical Features

After larval penetration, mild itching and nonspecific papules may occur and subside. After 1-3 days the larva begins to migrate, leaving a tortuous, raised, linear track marked by pruritus and erythema (**Fig. 37.1**). The lesion advances a few millimeters to several centimeters each day, and there may be resolution of the older parts of the track. Multiple lesions are common. Excoriation and secondary infection are seen.

Diagnosis

The diagnosis is usually made clinically when a characteristic lesion appears in a person with a history of possible exposure. Biopsy often fails to demonstrate the organism, which usually lies 1-2 cm beyond the leading edge of the track.

Treatment

- Ivermectin: one oral dose (200 μg/kg body weight)
- Albendazole: 400 mg orally for 3-7 days
- Thiabendazole: 10-15% topically three times daily for 5-7 days.

Topical 0.1% triamcinolone or other low-potency topical steroid may be used for symptomatic itch relief. Bacterial superinfection should be considered and treated where appropriate.

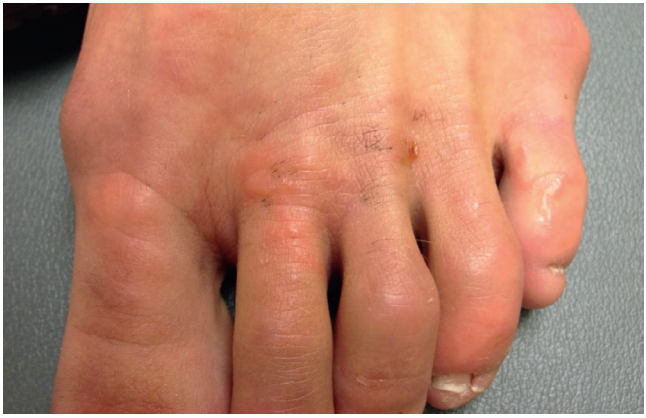

Fig. 37.1 Cutaneous larva migrans. Note the serpiginous track on the little toe.

MYIASIS

Myiasis refers to infestation by the larvae of flies. Animals, including livestock, are more typical hosts, but humans can also function as hosts. The larvae mature over several weeks to 2 months. Infestation sites are characterized by intermittent pain and irritation, often accompanied by exudate, and may not present until travelers have returned home. Patients may report a sensation of movement under the skin.

The three cutaneous forms are furuncular myiasis, dermal myiasis, and wound myiasis, with some overlap among these and with cutaneous larva migrans. Furuncular myiasis occurs primarily in Mexico, Central and South America, and Africa and may be found among travelers returning from these areas. Dermal myiasis occurs in Central and South America and is a variant of cutaneous larva migrans. Wound myiasis is a ubiquitous condition that occurs whenever flies deposit eggs into an open wound or ulcer, which then develop into larvae. Most infestations are superficial. However, infestation by several species of screwworm flies results in dangerous sequelae due to the tendency of the larvae to penetrate deep tissue.

Etiology, Epidemiology, and Clinical Features

Furuncular Myiasis

Furuncular myiasis in Mexico and Central and South America is caused by the human botfly, *Dermatobia hominis*. This 1.5-cm-long yellow-brown fly catches other biting insects such as mosquitoes in midair and attaches its eggs to their bodies. When the biting insect feeds, the eggs hatch and the larvae enter, often through the puncture wound. The larvae develop in the dermis, breathing through an opening (pore) in the skin. Botfly myiasis may occur on any exposed surface but commonly is found on the scalp as single or multiple 2- to 3-cm domed erythematous papule(s) with a central pore. Careful inspection may reveal movement beneath. Lesions can produce paroxysmal pain and pruritus and can lead to secondary infection. Larvae leave the host after 5-10 weeks.

African furuncular myiasis is due to the tumbu fly, *Cordylobia anthropophaga*. Tumbu fly larvae infestations are more common during rainy seasons, when fly populations rise. They develop from eggs that have been laid on shady ground or on objects such as clothes or diapers hung out to dry. Larvae hatch and can live up to 9 days before finding a host. They are sensitive to warmth and vibration, allowing them to find a host. They penetrate human skin quickly, typically at the head, neck, breast, or back, or any areas in contact with

contaminated clothing. Larvae mature and vacate the host after 8-12 days; in the meantime, they produce a boil-like lesion, often accompanied by exudate and pruritus. Heavy infestation can also cause fever and malaise.

Rare cases of North American furuncular myiasis have been associated with endemic botflies (*Cuterebra* species).

Dermal myiasis

Gasterophilus species of horse fly cause creeping eruptions worldwide. *Hypoderma ovis* and *Hypoderma lineatum* are cattle bot flies found in the Northern Hemisphere causing presentation similar to cutaneous larva migrans.

Wound myiasis

Most wound myiasis consists of superficial infestation of existing wounds or ulcers. However, two varieties of screwworm can cause deeper and more serious infestation. The *Cochliomyia hominivorax*, or New World screwworm (blowfly larvae) is found in parts of South and Central America and some Caribbean islands. Human infestation is rare but can be fatal because larvae penetrate deep tissue. Also, larvae are laid in multiple batches, with mature flies attracted back by a scent given off by maturing larvae. This can result in as many as 3000 larvae at a given site. The blowfly responsible for the New World screwworm has a blue-green body and is slightly larger than a housefly. It produces pink larvae that can reach 2 cm in length. The *Chrysomya bezziana*, or Old World screwworm, is found in tropical Africa and parts of Asia, including Indonesia and the Philippines, as well as New Guinea. The mature screwworm fly is 8-12 mm, with a blue-green body and two stripes across the thorax.

Diagnosis

Diagnosis requires identification of larvae. Consider myiasis in patients returning from affected countries presenting with lesions resembling boils. Typically a pore is maintained to allow larvae to breathe, and sometimes feces or bubbles can be seen in the exudate. Myiasis lesions in which infected patients feel sense of movement are more easily diagnosed.

Treatment

- First, try to prevent exposure with physical and chemical protection from biting insects, and iron all air-dried clothes in areas of tumbu fly myiasis to kill larvae before wearing the clothes.
- There is no medical treatment for myiasis; the larvae may be left to mature and fledge on their own, usually without harm to the host, or they can be extracted. Patients usually opt for extraction.
- If office visit time allows, apply an occlusive substance such as petroleum jelly over the air holes to block the air supply to larvae. Within an hour this should agitate them and drive them to the surface, where they may be more easily grasped with forceps and removed. If time is limited, apply a small occlusive adhesive bandage over each lesion. The bandage is peeled off after 3-4 days, and the dead (asphyxiated) larva can be extracted from the burrow. This technique may be expeditious for patients with multiple lesions.
- Surgical extraction may be necessary, particularly in the case of deeper burrowing screwworms. Extraction of the Old World screwworms can be complicated by the presence of spiny processes.

TUNGIASIS

Etiology

Tungiasis is caused by *Tunga penetrans*, a tiny sand flea, known as the nigua, chigoe, or jigger flea. Dogs, cats, pigs, cows, and rats are known reservoirs. The fertilized female flea burrows into the bare skin of the host, where it resides beneath the stratum corneum of the epidermis and feeds on blood. It maintains a connection to the outside and discharges

feces and lays eggs through this connection. Over the course of a few weeks, the female discharges 100 eggs or more. The flea dies within the epidermis and is sloughed off over time.

Epidemiology

Tungiasis is indigenous in Central and South America, the Caribbean islands, sub-Saharan Africa, India, Pakistan, and in the Indian Ocean islands of Madagascar and the Seychelles.

Clinical Features

Penetration by the flea occurs typically around the toenail or in interdigital spaces, as well as other areas of the foot, in patients who wear open shoes. It may cause pruritus, typically without visible manifestations. Five days later, small white papules or nodules appear, with a black point that corresponds to the fleas' connection to the outside. As the flea engorges, pain increases. The nodule can reach a diameter of up to 1.2 cm. This also corresponds to the time when the flea releases ova. She then dies, resulting in a black scab that heals spontaneously. Lesions can also ulcerate and are prone to superinfection. Multiple or numerous infestations can cause significant deformity.

Diagnosis

Diagnosis can be difficult outside the endemic area, where it may be misidentified as a wart, insect bite, or inflammation due to a splinter or foreign body. Diagnosis should be made by removing and identifying the flea.

Treatment

- Removal of the flea is easier in the earlier stages of infection. Use a sterile needle or curette, accompanied by careful cleaning of the area. Later stages may necessitate an incision to remove the flea. Rupture of the egg sac is not harmful to the patient and can be handled with copious lavage; however, it is important to remove the entire flea, whether intact or not.
- Topical and systemic treatments have been ineffective. Recently, application of low-viscosity silicone oils such as NYDA dimeticone has been promising. This is believed to smother the flea.
- Secondary bacterial infection should be treated with appropriate antibiotics.
- To prevent infection in endemic areas, wear shoes and inspect feet routinely.

TICK BITES

Ticks are blood-sucking insects that can cause significant local and systemic reactions and are vectors for a variety of viral and bacterial diseases.

Etiology

Ticks are ectoparasites found worldwide, encompassing more than 800 recognized species. Ticks feed by anchoring mouth parts in the skin and inserting a hollow proboscis to suck blood. They are members of the class Arachnida and are divided into two families: Ixodidae, or hard-bodied ticks, and Argasidae, or soft-bodied ticks.

Ixodidae live in forest and grassland areas, attach to warm-blooded hosts, and remain on the skin for days to weeks before dropping off. Ixodid ticks are vectors for Lyme disease (Chapter 24), ehrlichiosis, Rocky Mountain spotted fever, babesiosis, Colorado tick fever, tick-borne encephalitis, and other infectious diseases. The Argasidae are mainly parasites of birds and live in nesting areas. They feed rapidly at night, often on several hosts in succession. Argasid ticks are vectors for relapsing fever and other illnesses.

Clinical Features

Tick bites are usually painless and unnoticed. Argasid ticks feed only transiently at night, and thus are virtually never discovered by patient or physician. The ixodid tick body may be noticed incidentally on the skin as a pea-sized tumor. Urticarial papules and pruritus may occur, calling attention to the tick. These papules subside within a few days after removal.

Argasid ticks cause more frequent inflammatory or papular reactions, which subside over several weeks. One Argasid species, *Ornithodoros tholozani*, produces deep red macules or papules with a central punctum at the bite site.

Tick bite granuloma is a persistent pruritic reaction occurring at the site of attachment of the tick. In some cases, tick bite granuloma is associated with retention of mouth parts in the skin. These granulomas are firm, slightly erythematous nodules that persist for months or years.

Tick fever is a systemic reaction with fever, headache, vomiting, and abdominal pain. This occurs after several days of attachment by the tick and subsides within 12-36 hours after its removal.

Tick paralysis is a rare, potentially fatal systemic reaction caused by the bite of several species of ticks endemic to western North America and Australia. Paralysis is attributed to a toxin elaborated by the tick. The reaction begins after 5-6 days of attachment, accompanied by irritability and sometimes low-grade fever. An ascending lower motor neuron paralysis develops rapidly and may lead to death from bulbar paralysis or aspiration. Symptoms abate rapidly after removal of the tick.

Diagnosis

Diagnosis is easiest if the tick is found attached to the skin. Otherwise tick bite reactions can be difficult to distinguish from reactions to other biting insects. Tick bite granuloma is distinguished from many other granulomatous processes by the intense pruritus that is present, but biopsy may be necessary.

Treatment

When traveling in tick-infested areas, wear light-colored clothing, because ticks are typically dark in color, which aids in spotting them before attaching to skin. Tuck pant cuffs into socks. Insect repellents containing DEET or picaridin and permethrin spray applied to clothing also offer some protection.

Treatment

- Grasp the tick as close to the skin as possible with tweezers, small forceps, or protected fingers. Apply steady traction perpendicular to the skin surface. Avoid pressure on the tick body. Tick fever and tick paralysis are treated by removal of the tick and supportive measures.
- Tick bite granuloma responds to intralesional steroids.
- Antibiotic prophylaxis after tick bites is controversial and should be considered only in select circumstances (see Chapter 24).

CERCARIAL DERMATITIS

Cercarial dermatitis, also known as swimmer's itch, is a self-limited, common parasitic infection in which humans are dead-end hosts. The number of outbreaks reported is increasing worldwide.

Etiology

Cercarial dermatitis is caused by penetration of the skin by avian schistosomal larval forms called cercariae. Snails infected with schistosome (blood fluke) species shed the infective cercariae into the water. The cercariae penetrate the wet skin of warm-blooded animals, including people. The cercariae can penetrate the upper layers of human skin but are unable to enter the vascular system, and soon die. In contrast, pathogenic species of schistosomes can enter the vascular system, where maturing flukes cause systemic disease (see Chapter 48).

Epidemiology

Cercarial dermatitis occurs worldwide where either fresh or saltwater is heavily contaminated with infected avian feces. People are exposed by swimming or wading in contaminated water.

Clinical Features

There are two phases to cercarial dermatitis: transient symptoms soon after exposure and delayed symptoms. Penetration of cercariae is accompanied by a prickling sensation and urticarial wheals, which resolve. Hours later, pruritic macules, papules, or vesicles may develop in the same sites. Recurrent infections result in increased inflammatory response. These lesions reach maximal intensity 2-3 days after infection and then subside within 1-2 weeks. Secondary bacterial infection can occur in excoriated lesions. Subsequent attacks tend to become more severe. Cercarial dermatitis usually spares areas covered by clothing, in contrast to seabather's eruption (discussed later in this chapter).

Diagnosis

Diagnosis rests on the history of exposure to contaminated water and typical clinical findings. Differential diagnosis includes insect bites, contact dermatitis, and scabies.

Treatment

- Immediate rubbing of the skin with a towel after leaving the water may remove adherent cercariae before they can penetrate the epidermis.
- Mild cases can be treated with compresses and topical steroids. Severe cases may require a brief course of systemic steroids.
- Secondary infection should be treated with appropriate antibiotics.
- Antiparasitic medications are not necessary.

CNIDARIAN ENVENOMATION

People swimming or wading in seawater are at risk for envenomation by cnidarians ("seabather's eruption"). Reactions to envenomation can range from very mild to fatal.

Etiology

The phylum Cnidaria includes more than 9000 invertebrate species that are most abundant in tropical waters, including sea anemones, coral, and jellyfish. By definition, all are capable of stinging their prey, but consequences in humans are usually subclinical or minor irritation; a few species, however, may cause dangerous envenomation. The injuries caused by Cnidaria are due to venom-laden organelles called nematocysts, containing a coiled filament with a barbed end. With the proper stimulus, these embed in the skin, discharging venom. Tentacles stuck to the skin often contain undischarged nematocysts that can be triggered by pressure or fresh water. Other stinging sea animals include sea urchins, sea anemones, sponges, stingrays, sea cucumbers, catfish, lionfish, and cone snails. True coral may cause stinging and can also cause foreign body granulomas.

Epidemiology

People in contact with seawater, or those walking on the beach in contact with washed-up jellyfish, are at risk for cnidarian stings. All ages are affected, but most severe or fatal reactions occur in children.

Clinical Features

Cnidarian stings present with a wide range of cutaneous and systemic features (**Table 37.1**). The severity depends on the number of nematocysts discharged into the skin, the nature of the venom, and the sensitivity of the victim. The best known is the Portuguese man-of-war, while the most deadly is the box jellyfish. The majority, however, cause only mild to moderate irritation.

Symptoms begin immediately or soon after contact and include stinging or burning pain, which may be severe. Pruritic or painful urticarial papules may become vesicular or bleed. Any part of the body can be affected, but because tentacles can become trapped in the fabric of water-permeable swimwear, lesions tend to cluster in areas that were covered during the swim. Lesions are typically distributed along a linear pattern (**Fig. 37.2**). Systemic reactions are variable among species and can include generalized urticaria, muscle spasms, anaphylaxis, and cardiovascular collapse.

TABLE 37.1 Common or Clinically Important Cnidarian Stings

Species	Distinguishing Features	Location	Clinical Effects and Considerations
Portuguese man-of-war, *Physalia physalis*	30-cm "sail" and 10-m long tentacles	Tropical Atlantic and Indo-Pacific	Cutaneous: "whip" lesions, wheals >7 cm Necrosis after 24 h Systemic: arrhythmia, headaches, fatalities associated
Bluebottle (or Indo-Pacific Portuguese man-of-war), *Physalia utriculus*	4-5 m long	Tropical Indo-Pacific, Australian, South Atlantic	Cutaneous: local pain, wheals, vesicles Systemic: none confirmed
Box jellyfish, *Chironex fleckeri*		Mainly Indo-Pacific and Australian tropical and subtropical waters, concentrated around northern Australia, with reports in east Pacific and Atlantic (Related *C. quadrigatus* found in Indo-Pacific including northern Australia, the Philippines, Malaysia, and Japan. The related Chiropsalmus *quadrumanus* is found in the Gulf of Mexico and in Brazilian coastal water.)	Special toxins: cardiotoxin and necrotoxin Cutaneous: massive wheals, vesicles persisting for 10 days resulting in scarring Systemic: 20% of stings are fatal. Arrythmia, cardiac arrest, hypotension, pulmonary hypertension Envenomations involving 6 m of total tentacle length may result in immediate loss of consciousness and 15 m of tentacle length of envenomation may be fatal. **Important: A single antivenom is made by Commonwealth Serum Laboratory Group based in Australia and is sold directly to consumers in some countries.**
Irukandji jellyfish, *Carukia barnesi* (and some related species)	Tiny jellyfish, up to 3 cm	Found in northern Australia October-May. Rare cases in southern Australia, Hawaii, Florida, Papua New Guinea, and Thailand.	Special toxins: mechanism unknown, possibly related to catecholamine release Cutaneous: oval erythematous area of 5-7 cm surrounded by papules Systemic: Several deaths reported. **Irukandji syndrome:** backache, hypertension, headache, muscle cramps, nausea, and vomiting **Important: Acetic acid/vinegar may amplify the effect.**
Fire coral, *Millepora*		Shallow reefs of tropical Atlantic and Caribbean	
Sea nettles, *Chrysaora* and *Cyanea*		Worldwide	Cutaneous: frequent cause of less severe stings
Thimble jellyfish, *Linuche unguiculata*, or sea anemone, *Edwardsiella lineata*		Bahamas, Bermuda, Philippines, Florida (USA), Thailand, Brazil, New Zealand	Cutaneous: causes "seabather's eruption," severely pruritic erythematous lesions. Thought to be caused by larvae trapped against the skin in clothing or hair, releasing nematocysts when they desiccate and die.

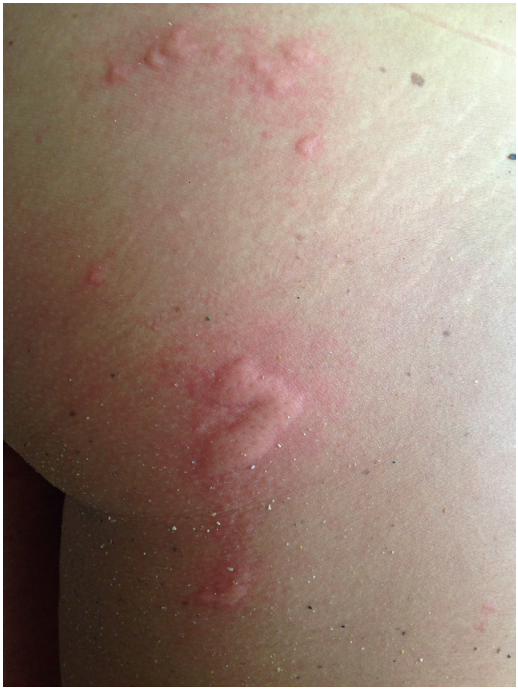

Fig. 37.2 Linear urticarial papules and plaques following acute cnidarian sting. (Courtesy of Jennifer Gardner, MD.)

Diagnosis

Cnidarian envenomation should be suspected whenever pain or itching begins during or after contact with seawater. A specific history of activities may help to distinguish between envenomation by free-floating or sessile forms. The pattern of skin lesions may also be helpful in identifying the causative organism, if it was not seen. The tentacles of the Portuguese man-of-war produce a characteristic whiplash appearance. Fragments of tentacles may adhere to the skin. Different species can be identified by microscopic examination of the nematocysts after removal by pressing cellophane tape against the skin.

Treatment

- The first step is to prevent further envenomation. Rinse the area with seawater to remove nematocysts. Fresh water may trigger further injection of venom.
- Hot water immersion (40-41° C) has been effective in relieving pain.
- Nematocysts may be inactivated with 5% acetic acid (household vinegar), although unexpected discharge of venom may occur.
- Tentacles and unseen nematocysts can be removed by unidirectional scraping with a thin firm object, such as a credit card, through a shaving motion along the affected area.
- Local reactions are treated with compresses, analgesics, and antihistamines if pruritus is prominent.
- A specific antivenom is available for stings by the box jellyfish *Chironex fleckeri* (Commonwealth Serum Laboratory, Melbourne, Australia).

- Severe cases of cutaneous pain and swelling may require a short course of systemic corticosteroid therapy.
- Ulcerated lesions may become secondarily infected and require antibiotics.
- Anaphylaxis is treated with standard supportive measures. Calcium gluconate or diazepam is sometimes used to control muscle spasms.

People planning activities involving significant water exposure in unknown waters, such as scuba diving, should inquire about local hazardous species. Cnidarians found washed ashore should not be handled. Swimmers, divers, and others participating in water activities where risks of cnidarian envenomation exist should wear protective gloves, footwear, and other garments such as wetsuits that are too thick to allow nematocyst penetration.

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