Cutaneous Larva Migrans: The Creeping Eruption

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Cutaneous larva migrans (CLM) is the most common tropically acquired dermatosis. It is caused by hookworm larvae, which are in the feces of infected dogs and cats. The condition occurs mainly in the Caribbean and New World, and anyone walking barefoot or sitting on a contaminated beach is at risk.

Ancylostoma braziliense *and* Ancylostoma caninum *are the most common hookworms responsible for CLM. The lesions, called creeping eruptions, are characteristically erythematous, raised and vesicular, linear or serpentine, and intensely pruritic. The conditions respond to oral and/or topical application of thiabendazole. Humans become an accidental dead-end host because the traveling parasite perishes, and its cutaneous manifestations usually resolve uneventfully within months.*

Cutis. 2003;72:111-115.

Cutaneous larva migrans (CLM), *creeping eruptions*, *creeping verminous dermatitis*, *sandworm eruptions*, *plumer's itch*, and *duckhunter's itch* are all terms that describe a clinical finding caused by several different parasites. CLM was first described in 1874. It has the most frequent serpiginous lesions seen in travelers and is the most common tropically acquired dermatosis. CLM is rated second to pinworm among helminth infections in developed countries.[1]

Most cases of CLM in North America and Europe involve travelers returning from tropical areas or hot climates, such as Africa, Latin America, the Caribbean, Southeast Asia, and even the southeastern United States. In a review of 60 cases presented to the Tropical Disease Unit of the Toronto Hospital in Ontario, Canada, 48% of patients with CLM had recently traveled to Jamaica.[2] CLM is an animal hookworm infestation usually caused by the *Ancylostoma* genus of nematodes. It is confined predominantly to tropical and subtropical countries, although its distribution is ubiquitous. Eggs of the nematode (usually *Ancylostoma braziliense*) are found most commonly in dog and cat feces. In Uruguay, 96% of dogs are infected by hookworms.[3] An individual is exposed to the larvae by sitting or walking on a beach that has been contaminated with dog or cat feces. In a retrospective survey of 44 cases of CLM presented at the Hospital for Tropical Diseases in London, 95% of patients reported a history of exposure at a beach.[4] Activities that pose a risk include contact with contaminated sand or soil, such as playing in a sandbox, walking barefoot on a beach, or working in crawl spaces under houses. Furthermore, carpenters, electricians, plumbers, farmers, ranchers, gardeners, pest exterminators, groundskeepers, and laborers are at an increased risk of acquiring CLM.

Etiology

Although CLM may be caused by a myriad of nematodes, the most common infective agents are *A braziliense and Ancylostoma caninum*.[5] *A braziliense* is a hookworm that infests wild domestic dogs and cats and can be found in the central and southern United States, Central and South America, and the Caribbean. *A caninum* is an Australian dog hookworm. Other causes include *Uncinaria stenocephala* (European dog hookworm) and *Bunostomum phlebotomum* (cattle hookworm). Rare etiologies include *Ancylostoma ceylonicum*, *Ancylostoma tubaeforme* (cat hookworm), *Strongyloides papillosus* (parasite of sheep, goat, and cattle), and *Strongyloides westeri* (parasite of horses).[1]

Adult hookworms release eggs while in the intestines of their hosts—dogs and cats. The eggs are passed through the stool onto warm sandy soil, which serves as a rich incubator (Figure 1). The eggs feed on soil bacteria and mature into noninfectious rhabditiform larvae and subsequently into

Accepted for publication May 30, 2003.

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The authors report no conflict of interest.

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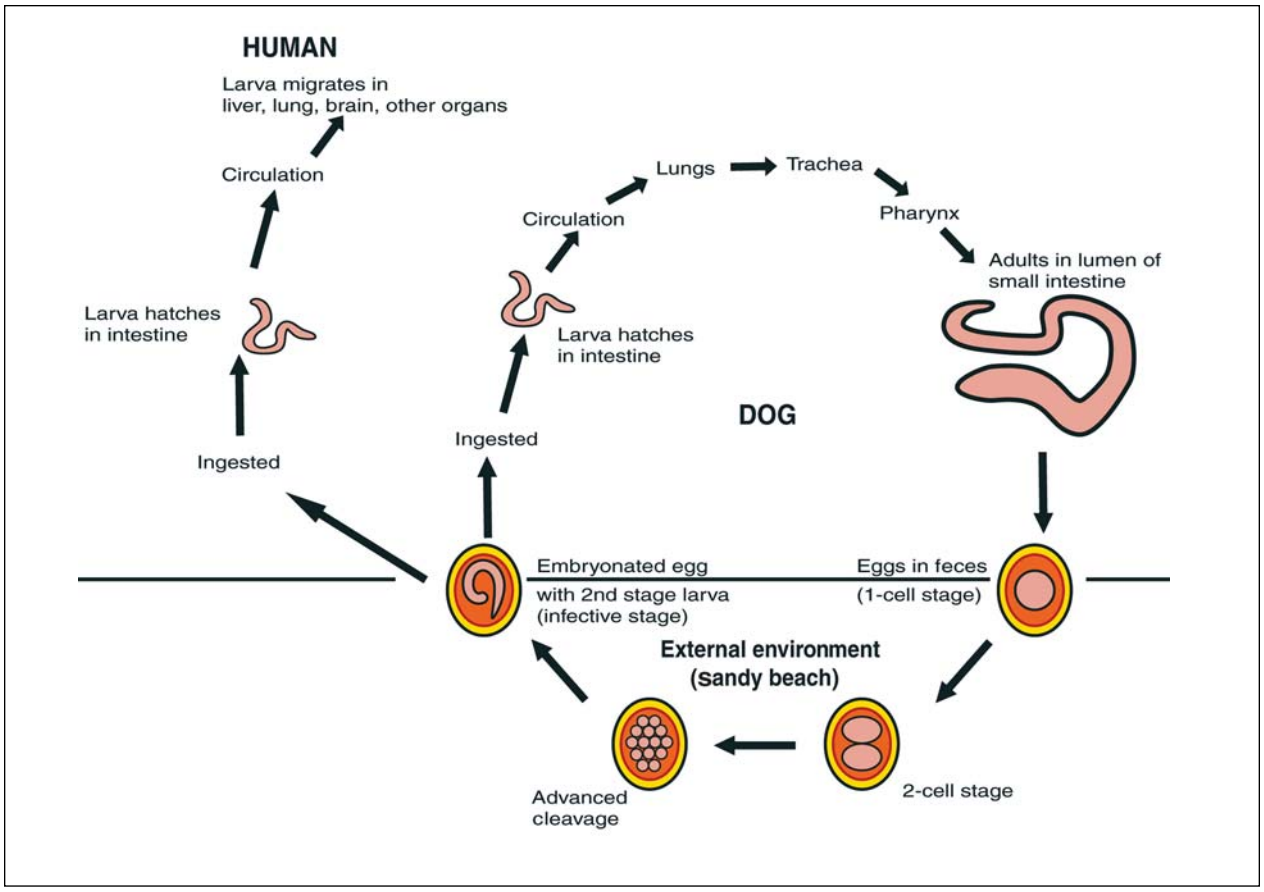

Figure 1. Life cycle of the larva. Graphic design by Taino Soba.

infectious filariform larvae.[6] These larvae become infectious after 2 months and acquire the ability to penetrate the skin of a new host. Humans are accidental hosts, who come in contact with soil contaminated with animal defecation. After contact, the infectious larvae penetrate the epidermis of intact skin by means of proteases. Hookworm larvae also can enter through broken skin or hair follicles.[7] It is believed that the larvae lack the collegenase required to penetrate the basement membrane to invade the dermis[1]; therefore, the larvae are blocked in the epidermis but still are able to move around (probably through the secretion and production of hyaluronidase).[8] After entering the epidermis, the larvae wander aimlessly through the skin. This migration, from the time of penetration to the onset of symptoms, can vary in length from days to weeks.[7] In a review, the mean period between exposure, penetration, and onset of symptoms was 2 to 50 days.[9] The movement of hookworm larvae causes a distinct lesion in the form of a highly pruritic, linear, serpiginous eruption. Tissue reaction is delayed 24 to 48 hours.[10] The larvae situate 1 to 2 cm ahead of the tract, which helps explain why local invasive treatment aimed at the tract is often ineffective.[11]

Lesions are characterized clinically by an almost pathognomonic creeping eruption, which is intensely pruritic. Patients often report a tingling or prickling sensation. The lesions are characteristically erythematous, raised and vesicular, and linear or serpentine (Figure 2). Lesions can be single or multiple and may be painful. They are approximately 3 mm wide and may reach 15 to 20 cm in length. The penetrating larvae advance from a few millimeters to a few centimeters a day. The larvae move ahead of the tracts, and vesicles may form in the tract as the worm changes direction. These vesicles become thick and encrusted.[12] Sinuous inflammatory trails may be clearly visible on the surface of the skin. The areas most frequently affected by infectious larvae are the dorsal and plantar aspects of the foot and interdigital spaces between the toes.[2] The buttocks is also a common area of eruption, especially in young children, because the larvae have been shown to penetrate

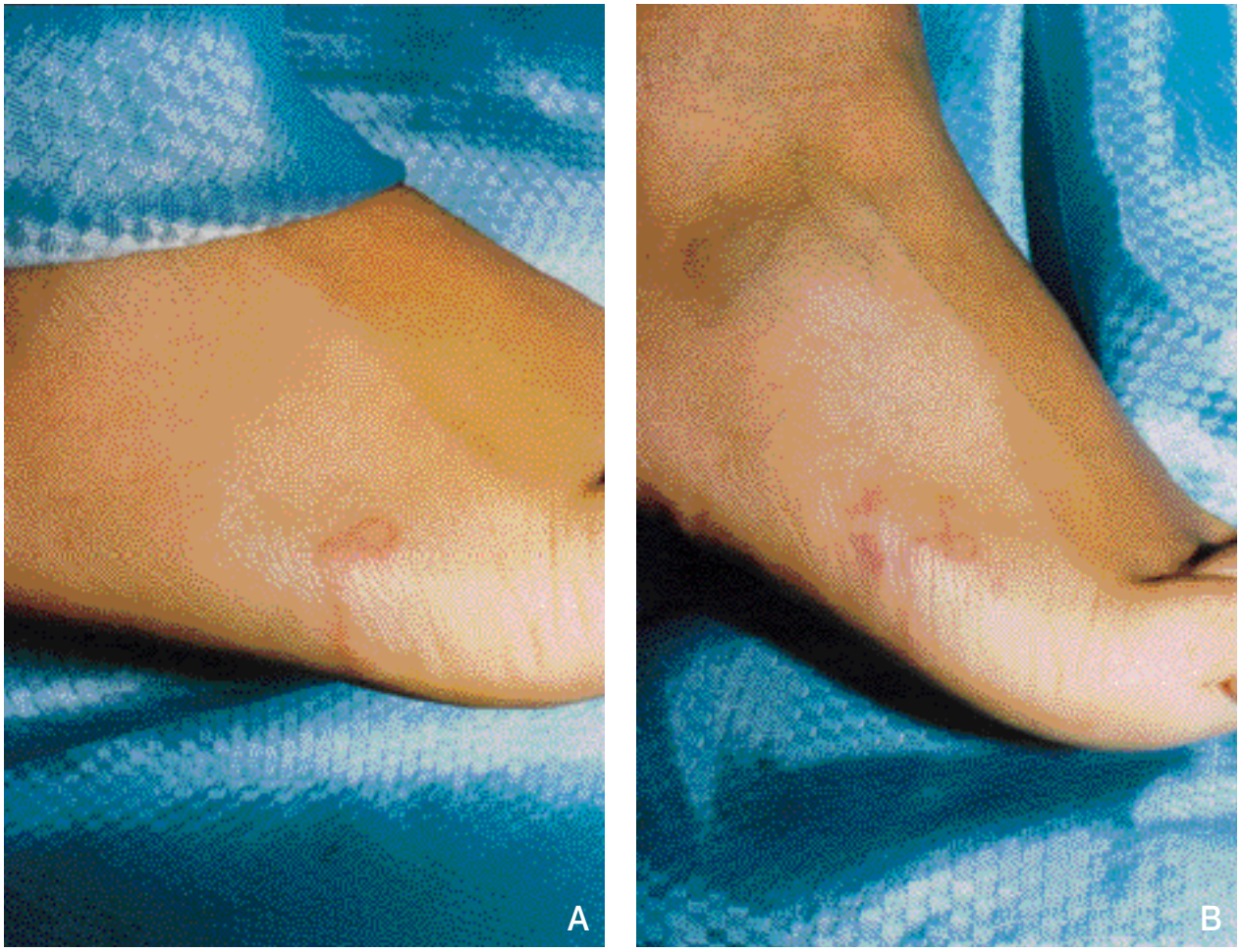

Figure 2. The lesions of cutaneous larva migrans are characteristically erythematous, raised, and linear or serpentine (A and B).

thin clothing such as bathing suits. Other areas less frequently affected include the arms and breasts.[7] Excoriation and impetiginization are common.

Systemic signs and symptoms (eg, wheezing, dry cough, urticaria) have been reported in patients with extensive infection. *A caninum* larvae can migrate to the gastrointestinal tract, causing human eosinophilic enterocolitis, a relatively rare condition that leads to acute abdominal pain, anorexia, nausea, and diarrhea. *A caninum* larvae also migrate into the dermis and enter the circulation, thus causing Löffler syndrome, which is characterized by asthma, pulmonary infiltrate, eosinophilia, fever, polmorphous erythema, and occasionally urticaria.[12-14] Löffler syndrome may accompany the cutaneous eruption.

Diagnosis of CLM is based on characteristic clinical findings of the eruption and known epidemiologic exposure. It is important for clinicians to obtain a full travel history, including a history of visiting beaches, being near uncovered sandboxes, or working in an occupation conducive to exposure. Laboratory results may include a transient peripheral eosinophilia on a complete blood count and increased IgE levels on total serum immunoglobulins. The intractable pruritus may be related to eosinophilia or elevated levels of IgE, which may be persistently abnormal up to 4 weeks after treatment of the infestation.[15,16] Results of a skin biopsy taken just ahead of the leading edge of a tract may show larvae (stained positive with periodic acid–Schiff) in a suprabasalar burrow, basal layer tracts, spongiosis with intraepidermal vesicles, necrotic keratinocytes, and an epidermal and upper dermal chronic inflammatory infiltrate with many eosinophils. However, detection of the causative pathogen by skin biopsy is difficult because of the rapid movement of the larvae.

The differential diagnosis of CLM includes cercarial or contact dermatitis, bacterial or fungal

infections, scabies, lichen planus, myiasis, loiasis, or other migratory parasites.

Prior to the 1960s, topical modalities such as ethyl chloride spray, liquid nitrogen, phenol, CO_2 snow, peperazine citrate, faudin, electrocautery, and even x-ray therapy were used unsuccessfully because the larvae were often missed and/or not killed. Chemotherapy with chloroquine, antimony, and diethyl carbamazine also were attempted, with similar haphazard results.[1]

Currently, treatment of CLM includes thiabendazole, ivermectin, mebendazole, or albendazole. Thiabendazole was first used in 1963 by Stone and Mullins.[17] The treatment of choice in the United States is the cutaneous application of 10% to 15% thiabendazole cream, made by crushing a 500-mg tablet of thiabendazole in 5 g of a water-soluble cream or by using an oral thiabendazole suspension topically. Oral thiabendazole is given 25 mg/kg per day divided in 2 doses, with a maximum of 3 g/day. Treatment length varies from 2 to 5 days. Decreased pruritus occurs within 24 to 48 hours, and lesions/tracts resolve within 1 week. Oral thiabendazole suspension of 500 mg/5 mL can be used twice per day as well. Oral thiabendazole is an excellent alternative for persistent cases, but it can have severe side effects (eg, nausea, vomiting, dizziness)[18] and rare, serious secondary effects (eg, seizures, erythema multiforme, toxic epidermal necrolysis).[19] A better tolerated therapy is topical thiabendazole. A 10% or 15% aqueous suspension of topical thiabendazole applied 4 times a day for 10 days is used for early localized lesions. Two studies have demonstrated a 98% efficacy for treating CLM with topical thiabendazole.[2,20]

Oral treatment is preferred for widespread lesions or unsuccessful topical therapy. Antibiotics are indicated in secondary bacterial superinfections, if they occur. Other systemic alternatives include oral albendazole, which has been reported to be effective with minimal to no side effects,[21,22] and oral ivermectin, which was reported to be effective without toxic side effects.[23] More extensive lesions can be treated with an oral dose of ivermectin 200 mg/kg for 3 to 7 days, albendazole 400 mg/day for 3 days, or 200 mg twice daily for 5 days. Treatment with topical thiabendazole is usually successful within 10 days of commencement. As an alternative therapy, liquid nitrogen cryotherapy can be used for a progressive end of larvae burrow. However, it is not always effective.[2,24]

Complications and Prognosis

A secondary bacterial infection, usually with *Streptococcus pyogenes*, may lead to cellulitis. Prognosis is excellent for CLM. CLM is a self-limiting disease but can last for up to 2 years.[25] Humans are accidental dead-end hosts with the larvae dying and lesions resolving within 4 to 8 weeks and, in rare cases, as long as 1 year.

Conclusion

Knowledge of the life cycle of dog and cat hookworms and the clinical manifestations of their infestation of human skin is vital to instituting prompt treatment. The clinician should take a complete travel history including review of initial presentation of symptoms, pattern of rash, location of lesions, course of symptoms, any previous attempts at treatment, and whether any family members are affected. Patient education is critical for preventing CLM. Patients should be advised to avoid walking barefoot when visiting tropical places, in particular beaches. Pet owners and breeders, as well as pet groomers, should be cautious.

Acknowledgment—The authors thank Taino Soba, Graphics Designer at New York College of Podiatric Medicine, New York, New York.

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