Hookworm-Related Cutaneous Larva Migrans

Patrick Hochedez, MD, and Eric Caumes, MD

Département des Maladies Infectieuses et Tropicales, Hôpital Pitié-Salpêtrière, Paris, France

Cutaneous larva migrans (CLM) is the most frequent travel-associated skin disease of tropical origin.[1,2] This dermatosis first described as CLM by Lee in 1874 was later attributed to the subcutaneous migration of *Ancylostoma* larvae by White and Dove in 1929.[3,4] Since then, this skin disease has also been called creeping eruption, creeping verminous dermatitis, sand worm eruption, or plumber's itch, which adds to the confusion. It has been suggested to name this disease hookworm-related cutaneous larva migrans (HrCLM).[5]

Although frequent, this tropical dermatosis is not sufficiently well known by Western physicians, and this can delay diagnosis and effective treatment. Indeed, misdiagnosis or inappropriate treatment affects 22% to 58% of the travelers with CLM.[6–8] In one case report, the time lag between the onset of disease and the diagnosis was 22 months.[9]

Five large (>40 patients each) published studies of imported cases of CLM in returning travelers have greatly helped improve knowledge of this disease.[2,6–8,10] This is particularly true as regards its natural history and response to treatment in short-term travelers without possibility of recontamination.

We reviewed the epidemiological, clinical, and therapeutic data drawn from studies of CLM in travelers. The aim of this review was to contribute to a better definition and description of the disease known as HrCLM.

Epidemiological data

HrCLM is one of the leading causes of dermatologic disorders observed in ill returned travelers.[1,2,11]

Risk factors for developing HrCLM have specifically been investigated in one outbreak in Canadian tourists: less frequent use of protective footwear while walking on the beach was significantly associated with a higher risk of developing the disease, with a risk ratio of 4. Moreover, affected patients were somewhat younger than unaffected travelers (36.9 vs 41.2 yr, $p = 0.014$). There was no correlation between the reported amount of time spent on the beach and the risk of developing CLM. Considering animals in the neighborhood, 90% of the travelers in that study reported seeing cats on the beach and around the hotel area, and only 1.5% noticed dogs.[12]

Geographical distribution

Published cases in Western countries mainly concern tourists returning from tropical areas.[6–8] The hookworms responsible for CLM are distributed worldwide, but infection is more frequent in the tropical and subtropical countries of Southeast Asia, Africa, South America, Caribbean, and the southeastern part of the United States.[8] In a prospective analysis of parasitic infections in Canadian travelers and immigrants, CLM was one of the leading causes of infections and was primarily acquired in beach destinations such as Jamaica, Barbados, Brazil, Thailand, and Mexico.[11] HrCLM is rare in temperate countries, but a few autochthonous cases have been reported from Europe, North America, and New Zealand.[13–18] HrCLM is common in the economically deprived communities of some areas in Brazil, but the clinicoepidemiologic pattern is different from that observed in travelers.[19,20]

Pathophysiology

HrCLM is caused by the penetration of the skin by cat, dog, or other mammal nematode larvae. The

Corresponding Author: Eric Caumes, MD, Département des Maladies Infectieuses et Tropicales, Hôpital Pitié-Salpêtrière, 45-83 Bld de l'hôpital, F-75013 Paris, France. E-mail: eric.caumes@psl.aphp.fr

main culprit species are *Ancylostoma braziliense* and *Ancylostoma caninum*, but other animal hookworms like *Uncinaria stenocephala* and *Bunostomum phlebotomum* are possible agents of HrCLM.[6,8,10,21] The adult hookworms live in animal intestines, and their eggs are spread on the soil during defecation. Egg hatching and larva survival are facilitated by moist grounds like beaches. After two molts in the soil, larvae become infectious and acquire the ability to penetrate the host skin.[8,13] The infection is usually acquired via contact with soil or sand contaminated with feces of infected cats or dogs, which explains why bare feet are the predominant site of penetration by the parasite larvae. In contrast to cats and dogs, humans are incidental hosts, and the larvae are unable to complete their natural cycle; thus, they are not able to deeply penetrate the human skin and consequently migrate within it for weeks.[8] During skin penetration, the secretion of hyaluronidase by hookworm larvae facilitates passage through the epidermis and dermis.[22] Usually, the typical eruption develops within a few days, and the larva itself is usually located 1 to 2 cm ahead of the eruption.[6] Without appropriate treatment, the larva dies and is resorbed within weeks or months of invasion.[23] Most of the time, the infection is restricted to the skin, but migration to the lung has been reported and is responsible for pulmonary eosinophilic infiltrate.[24,25]

Clinical presentation

The clinical features of HrCLM have been accurately described in five large series of travelers admitted to tropical medicine clinics[2,6–8,10] and in two outbreaks (Table 1).[12,26] The incubation period of HrCLM is usually a few days and rarely goes beyond 1 month. Cutaneous lesions appeared after return in 51% to 55% of the travelers, and the mean time of onset after return ranged from 5 to 16 days.[2,7,8] In a German study, the time of onset ranged from 16 weeks before return to 28 weeks after return (mean: 1.5 d after return). In the two above-mentioned outbreaks of CLM, the median time from the start of the trip to the development of the eruption ranged from 10 to 15 days.[12,26] However, some extremely long incubation periods have been reported, lasting for 4 to 7 months.[6,7]

The striking symptom of HrCLM is pruritus localized at the site of the eruption. It is reported in 98% to 100% of patients.[7,8] After effective treatment, the pruritus has been shown to disappear much sooner than the eruption (on average 7.2 d earlier).[12]

The most frequent and characteristic sign of HrCLM is "creeping dermatitis," a clinical sign defined as an erythematous, linear, or serpiginous track that is approximately 3 mm wide and may be up to 15 to 20 mm in length.[27] The creeping track, associated with the larva migration, may extend a few millimeters to a few centimeters daily.[8] The mean number of lesions per person varies from 1 to 3.[2,7,8]

Two other major clinical signs are edema and vesiculobullous lesions along the course of the larva. Local swelling is reported in 6% to 17% of patients and vesiculobullous lesions in 4% to 40%.[2,8,10,12]

Potentially, all unprotected parts of the skin in contact with contaminated soil may be involved. However, the most frequent anatomic locations of HrCLM lesions are the feet in more than 50% of individuals, followed by the buttocks and thighs.[2,6,8,12] Other sites may include the elbow, breast, legs, and face.[7,8]

Without any treatment, the eruption usually lasts between 2 and 8 weeks, but it may be longer. In one case report, an active disease was reported for almost 2 years.[9]

Hookworm folliculitis is a particular form of HrCLM. The largest series included seven travelers and consisted of numerous (20–100) follicular, erythematous, and pruritic papules and pustules, located mainly on the buttocks and associated with numerous relatively short tracks, generally arising from follicular lesions[28]. Hookworm larvae can be histologically found trapped in the sebaceous follicular canal.[28,29]

Complications

Local complications are led by secondary bacterial infection of the involved skin area (impetiginization). It occurs in up to 8% of cases.[7,8]

Systemic complications are uncommon and mainly involve the lungs, consisting of cases of eosinophilic pneumonitis associated with CLM.[25,30–32] Although the pathogenesis is not completely understood, the *Ancylostoma* larva has been identified in the sputum in one case report.[24]

One case of visceral larva migrans caused by *A caninum* has been reported,[33] together with one larval invasion of skeletal muscles in a man who also had pulmonary symptoms.[34] One case of erythema multiforme in which HrCLM was considered as one of the triggering events has also been reported.[35]

Diagnosis

HrCLM is usually a clinical diagnosis based on the typical clinical presentation in the context of recent

Table 1 Main clinical features of cutaneous larva migrans in eight series (>15 patients) of travelers returning from endemic countries

	1993 Davies[8]	1994 Jelinek[6]	1995 Caumes[2]	2000 Bouchaud[7]	2001 Blackwell[10]	2000 Tremblay[12]	2001 Green[26]
Date 1st author Study type	Retrospective case study	Retrospective case study	Prospective study of travel-associated dermatitis	Prospective study	Retrospective case study	Outbreak, retrospective (questionnaire-based study)	Outbreak
Study site	Canada	Germany	France	France	England	Canada	England
Population	Canadian (97%), immigrant (3%)	German (100%)	French (100%)	French (100%)	Europeans (98%), immigrant (2%)	Canadians (100%)	British military personnel
Number of patients	60	98	67	64	44	32	13
Male-to-female ratio	0.8	1.2	—	1.4	1	1	Men
Mean age	29.2 (5–73)	32	—	33 (1.5–73)	28.9 (3.5–70)	—	—
Country visited	Caribbean (75%), Brazil (8.3%), Mexico (5%)	Southeast Asia (31.6%), Caribbean (19.4%), South America (13.4%), East Africa (10.2%), Indian subcontinent (10.2%), Central America (8.2%), West Africa (5.1%)	—	Caribbean (39%), Asia (25%), Latin America (19%), Africa (17%)	Africa (32%), Caribbean (30%), Southeast Asia (25%), Central America (11%), South America (2%)	Barbados	Belize
Mean duration of travel (days)	14.4 ± 13.5 d (with a mode of 7 d)	6 (1–72)	—	28 (6–90)	—	7	15
Barefoot walk or sandy beach exposure	95%	—	—	—	95% beach	100% beach	Military exercises
Misdiagnosed or inappropriately treated prior to diagnosis (%)	58	22	—	55	27.5	46.7	—
Mean/median time from arrival in the tropics to onset of symptoms	15 d (2–50) (mean)	—	—	21 (5–135) (median)	—	15.5 (5–34) (median)	10 (4–38) (median)

Continued

Table 1 Continued

Mean/median time of onset of symptoms from departure from the tropics	5 (0–30) (mean)	—	8 (0–28) (median)	16 (1–120) (mean)	—	—
Onset after return (%)	45	75	51	55	—	70
Median duration of symptoms	—	5.6 wk (1–36)	—	4 wk	8 wk (1–104)	24.5 d (7–49)
Anatomic location (%)						
Feet	87	62	66	48	39	100
Buttocks	5	13	3	23	18	0
Thigh (upper legs)	5	9	—	16	—	0
Lower legs	1.7	3	—	—	12	92 calf or shin
Trunk	3.3 (breast)	7	—	17 (abdomen + chest)	16	—
Upper extremities	1.7	7	—	—	14	—
Face	0	0	—	1.5	2	—
Pruritus (%)	98	—	100	100	95	100
Serpiginous lesion (%)	96.7	—	99	—	93	96.9
Swelling (%)	17	—	6	—	—	—
Vesiculobullous lesion (%)	10	—	9	—	4.5	40.6
Pain	10	—	—	—	—	—
Mean/median number of lesions per person	1.7 (1–6) (mean)	—	1 (1–24) (median)	3 (1–15) (mean)	—	—
Hookworm folliculitis (%)	—	—	3	—	—	—
Superinfection	0%	—	—	5 (8%)	2 (5%)	—

travel to a tropical or a subtropical country and beach exposure.[5,6,8] Clinical characteristics of the creeping trail (length, width, speed of migration, location, duration) easily help differentiate HrCLM from other causes of creeping dermatitis.[27]

Blood tests are not necessary for diagnosis and are not currently recommended; no specific serological or culture methods are currently available. Theoretically, total blood count could detect eosinophilia; this feature was reported in 20% of the 40 patients tested among a series of 98 CLM in German travelers.[6] Nonetheless, in the particular case of hookworm folliculitis, skin biopsy specimens may reveal nematode larvae in the follicular canal.[29] In addition, this is the only clinical form of HrCLM that gives the opportunity to diagnose via a skin smear of a pustular lesion. Additionally, it allows the identification of the larvae of the culprit nematode.

Differential diagnoses

The differential diagnoses include all the dermatoses that give rise to creeping dermatitis, ie, serpiginous or linear migrating cutaneous lesions. Thus, noncreeping, linear or serpiginous dermatoses such as phytophotodermatosis, jellyfish stings, zoster, or lichenoid eruptions are easily ruled out. Nonetheless, there are numerous causes of creeping eruption, and, with the notable exception of creeping hair mostly described in Japan,[36,37] all of them are of parasitic origin.[5,38]

From a parasitological point of view, differential diagnoses include diseases in which creeping eruption may be due to the larvae of nematodes (other than hookworms), adult nematodes, larval forms of trematodes, fly maggots, and arthropods (Table 2).[27]

Animals host other nematode larvae that may infect men, such as *Gnathostoma* spp. (gnathostomiasis), *Pelodera strongyloides*, and *Spirurina* sp.,[5,39,42,43] which are other causes of the CLM syndrome.[27] But other larvae of helminths may cause creeping dermatitis such as *Strongyloides stercoralis* (larva currens),[44] a human nematode, and *Fasciola gigantica* (fascioliasis),[40] a human trematode. In the case of hookworm folliculitis, bacterial folliculitis is the main differential diagnosis, but it is nonpruritic.[28]

From a clinical point of view, differential diagnoses do not include parasites whose larvae do not give rise to migratory signs when they travel through the skin (cercarial dermatitis, onchocerciasis, dirofilariasis).[27]

Treatment

Oral ivermectin and albendazole have become the first-line treatments as thiabendazole is no longer marketed and thus is no longer available worldwide.[45] Ivermectin is a synthetic derivative of the antiparasitic class of avermectins.[46] Taken in a single dose, ivermectin is well tolerated and highly efficacious, with cure rates of 94% to 100% in all but one of the largest series.[7,45,47,48]

After ivermectin treatment, symptoms disappear within 1 week: 3 days (1–20) for pruritus and 7 days (1–30) for creeping dermatitis.[7]

In case of initial failure or relapse, cure can be obtained with one or two repeated courses of ivermectin.[7,48] Side effects related to ivermectin in this use are rare and not systemic; local bullous reactions have been reported.[7,47]

Albendazole, a third-generation, heterocyclic, anthelmintic drug, is also effective and well tolerated. In the absence of consensual optimal dosage, the regimen should be 400 to 800 mg/d (according to weight) for 3 days.[10,45,47,49,50]

In the case of hookworm folliculitis, treatment is more difficult than in traditional form and necessitates repeated courses of oral anthelmintic agents.[28] For lightweight children or when oral ivermectin and albendazole are contraindicated, then the application of a 10% albendazole ointment, twice a day for 10 days, is a safe and effective alternative treatment.[51]

Cryotherapy with liquid nitrogen is not recommended. Freezing is ineffective because the larva is usually located several centimeters beyond the visible end of the trail, and the larvae are capable of surviving temperatures as low as −21°C for more than 5 minutes; moreover, this procedure is painful and can lead to chronic ulcerations.[6,8,21,45]

Prevention

The best way to prevent HrCLM is to wear protective footwear when walking on the beach because avoiding tropical beaches frequented by dogs and cats or banning animals from beaches in tropical areas is impossible.[12,52] When lying on tropical beaches potentially frequented by dogs and cats, areas of sand washed by the tide are preferable to dry sand, and mattresses are preferable to towels.[45]

Conclusions

HrCLM is a striking example of how travel medicine contributes to improved knowledge of tropical

Table 2 Main parasites causing creeping eruption

Parasite	Form of the causal parasite	Distribution	Transmission	Cutaneous signs	Diagnosis	Treatment
Nematode's larvae						
Gnathostomiasis (*Gnathostoma* spp.)	Larva of animal nematodes*	Southeast Asia, Latin America	Eating raw or nearly raw fish	Creeping eruption, migratory swelling, nodules	Serological, parasitological (if the larva exists in the skin)	Albendazole, ivermectin
Animal hookworms, *Pelodera strongyloides*; zoonotic *Strongyloides* spp.	Larva of animal nematodes*	Tropical and subtropical	Skin penetration	Cutaneous trail, pruritus, bullae, folliculitis	Parasitological (in case of folliculitis)	Ivermectin, albendazole
Spirurina spp.[39]	Larva of animal nematodes*	Asia	Eating raw or nearly raw fish and shellfish	25 cm long serpiginous red track with vesicles	Serological (ELISA)	Ivermectin
Strongyloides stercoralis	Larva of human nematodes	Tropical and subtropical	Skin penetration	Usually one burrow, on the abdomen or buttocks; lasts for a few hours only, recurrences, larger and fast moving (larva currens)	Stool test	Ivermectin, albendazole
Adult nematodes						
Loiasis (Loa loa)	Adult nematode	Central African forest	Diptera bites (*Chrysops*)	Calabar swelling, loa loa migration under the skin/conjunctiva	Microfilaremia, serological	Ivermectin, albendazole, diethyl-carbamazine
Dracunculiasis (*Dracunculus medinensis*)	Adult nematode	Sub-Saharan Africa, extinction in view	Drinking contaminated water	Limb swelling, cutaneous ulcer, serpiginous trail	Clinical, parasitological	Extraction
Trematode's larvae						
Fascioliasis (*Fasciola gigantica*)[40]	Larva of animal trematode	Asia	Eating raw vegetables (cress, dandelion)	Dark red and serpentine, tunnel-like track	Serological, parasitological (after extraction), stool test	No data
Fly's maggot						
Migratory myiasis (*Gasterophilus* spp.)	Larva of Diptera (maggot)	Tropical and subtropical	Skin penetration	Cutaneous trail	Anatomical (after extraction)	Surgical
Arthropod						
Scabies (*Sarcoptes scabiei*)[41]	Adult arthropods	Worldwide	Skin-to-skin contact with a person already infested with scabies	Linear burrow, papules, vesicles	Direct examination of skin scrapings using microscope	Topical treatment or oral ivermectin

ELISA = enzyme-linked immunosorbent assay.
*Causes of the cutaneous larva migrans syndrome sensu stricto.[26]

diseases. Indeed, the description of HrCLM in large series of briefly exposed travelers has contributed to a better understanding of the incubation period and natural history of the disease. It has helped distinguish it from other causes of CLM and creeping dermatitis. Finally, it has provided the opportunity to assess the efficacy of various treatments more reliably than in inhabitants of endemic areas in whom it may be impossible to distinguish relapses from reinfections.

Declaration of Interests

The authors state that they have no conflicts of interest.

References

1. Freedman DO, Weld LH, Kozarsky PE, et al. Spectrum of disease and relation to place of exposure among ill returned travelers. N Engl J Med 2006; 354:119–130.
2. Caumes E, Carriere J, Guermonprez G, et al. Dermatoses associated with travel to tropical countries: a prospective study of the diagnosis and management of 269 patients presenting to a tropical disease unit. Clin Infect Dis 1995; 20:542–548.
3. Lee R. Case of creeping eruption. Trans Clin Soc Lond 1874; 8:44–45.
4. White GF, Dove WE. A dermatitis caused by larvae of Ancylostoma caninum. Arch Dermatol 1929; 20: 191–200.
5. Caumes E, Danis M. From creeping eruption to hookworm-related cutaneous larva migrans. Lancet Infect Dis 2004; 4:659–660.
6. Jelinek T, Maiwald H, Nothdurft HD, Loscher T. Cutaneous larva migrans in travelers: synopsis of histories, symptoms, and treatment of 98 patients. Clin Infect Dis 1994; 19:1062–1066.
7. Bouchaud O, Houze S, Schiemann R, et al. Cutaneous larva migrans in travelers: a prospective study, with assessment of therapy with ivermectin. Clin Infect Dis 2000; 31:493–498.
8. Davies HD, Sakuls P, Keystone JS. Creeping eruption. A review of clinical presentation and management of 60 cases presenting to a tropical disease unit. Arch Dermatol 1993; 129:588–591.
9. Richey TK, Gentry RH, Fitzpatrick JE, Morgan AM. Persistent cutaneous larva migrans due to Ancylostoma species. South Med J 1996; 89:609–611.
10. Blackwell V, Vega-Lopez F. Cutaneous larva migrans: clinical features and management of 44 cases presenting in the returning traveller. Br J Dermatol 2001; 145:434–437.
11. Boggild AK, Yohanna S, Keystone JS, Kain KC. Prospective analysis of parasitic infections in Canadian travelers and immigrants. J Travel Med 2006; 13: 138–144.
12. Tremblay A, MacLean JD, Gyorkos T, Macpherson DW. Outbreak of cutaneous larva migrans in a group of travellers. Trop Med Int Health 2000; 5:330–334.
13. Herbener D, Borak J. Cutaneous larva migrans in northern climates. Am J Emerg Med 1988; 6: 462–464.
14. Klose C, Mravak S, Geb M, et al. Autochthonous cutaneous larva migrans in Germany. Trop Med Int Health 1996; 1:503–504.
15. Zimmermann R, Combemale P, Piens MA, et al. Cutaneous larva migrans, autochthonous in France [Apropos of a case]. Ann Dermatol Venereol 1995; 122:711–714.
16. Diba VC, Whitty CJ, Green T. Cutaneous larva migrans acquired in Britain. Clin Exp Dermatol 2004; 29:555–556.
17. Bradley J. Home-grown cutaneous larva migrans. N Z Med J 1999; 112:241–242.
18. Roest MA, Ratnavel R. Cutaneous larva migrans contracted in England: a reminder. Clin Exp Dermatol 2001; 26:389–390.
19. Jackson A, Heukelbach J, Calheiros CM, et al. A study in a community in Brazil in which cutaneous larva migrans is endemic. Clin Infect Dis 2006; 43:e13–e18.
20. Heukelbach J, Wilcke T, Feldmeier H. Cutaneous larva migrans (creeping eruption) in an urban slum in Brazil. Int J Dermatol 2004; 43:511–515.
21. Elliot DL, Tolle SW, Goldberg L, Miller JB. Pet-associated illness. N Engl J Med 1985; 313:985–995.
22. Hotez PJ, Narasimhan S, Haggerty J, et al. Hyaluronidase from infective Ancylostoma hookworm larvae and its possible function as a virulence factor in tissue invasion and in cutaneous larva migrans. Infect Immun 1992; 60:1018–1023.
23. Katz R, Ziegler J, Blank H. The natural course of creeping eruption and treatment with Thiabendazole. Arch Dermatol 1965; 91:420–424.
24. Muhleisen JP. Demonstration of pulmonary migration of the causative organism of creeping eruption. Ann Intern Med 1953; 38:595–600.
25. Guill MA, Odom RB. Larva migrans complicated by Loeffler's syndrome. Arch Dermatol 1978; 114: 1525–1526.
26. Green AD, Mason C, Spragg PM. Outbreak of cutaneous larva migrans among British military personnel in Belize. J Travel Med 2001; 8:267–269.
27. Caumes E. It's time to distinguish the sign 'creeping eruption' from the syndrome 'cutaneous larva migrans'. Dermatology 2006; 213:179–181.
28. Caumes E, Ly F, Bricaire F. Cutaneous larva migrans with folliculitis: report of seven cases and review of the literature. Br J Dermatol 2002; 146:314–316.
29. Miller AC, Walker J, Jaworski R, et al. Hookworm folliculitis. Arch Dermatol 1991; 127:547–549.
30. Del Giudice P, Desalvador F, Bernard E, et al. Loeffler's syndrome and cutaneous larva migrans: a rare association. Br J Dermatol 2002; 147:386–388.

31. Butland RJ, Coulson IH. Pulmonary eosinophilia associated with cutaneous larva migrans. Thorax 1985; 40:76–77.

32. Ambrus JL, Klein E. Loffler syndrome and ancylostomiasis brasiliensis. N Y State J Med 1988; 88:498–499.

33. Gandullia E, Lignana E, Rabagliati AM, Penna R. Visceral larva migrans caused by Ancylostoma caninum. Minerva Pediatr 1981; 33:917–923.

34. Little MD, Halsey NA, Cline BL, Katz SP. Ancylostoma larva in a muscle fiber of man following cutaneous larva migrans. Am J Trop Med Hyg 1983; 32: 1285–1288.

35. Vaughan TK, English JC III. Cutaneous larva migrans complicated by erythema multiforme. Cutis 1998; 62:33–35.

36. Lehmuskallio EA. Hair fragment in the skin resembling larva migrans. Br J Dermatol 1975; 93: 349–350.

37. Sakai R, Higashi K, Otha M, et al. Creeping hair: an isolated hair burrowing in the uppermost dermis resembling larva migrans. Dermatology 2006; 213: 242–244.

38. Elgart ML. Creeping eruption. Arch Dermatol 1998; 134:619–620.

39. Hattori S, Niimi Y, Kawana S. Creeping eruption caused by a larva of the suborder Spirurina type X. Eur J Dermatol 2003; 13:87–89.

40. Xuan le T, Hung NT, Waikagul J. Cutaneous fascioliasis: a case report in Vietnam. Am J Trop Med Hyg 2005; 72:508–509.

41. Chosidow O. Clinical practices. Scabies. N Engl J Med 2006; 354:1718–1727.

42. Menard A, Dos Santos G, Dekumyoy P, et al. Imported cutaneous gnathostomiasis: report of five cases. Trans R Soc Trop Med Hyg 2003; 97: 200–202.

43. Jones CC, Rosen T, Greenberg C. Cutaneous larva migrans due to Pelodera strongyloides. Cutis 1991; 48:123–126.

44. Ly MN, Bethel SL, Usmani AS, Lambert DR. Cutaneous strongyloides stercoralis infection: an unusual presentation (case reports). J Am Acad Dermatol 2003; 49(2 Suppl):S157–S160.

45. Caumes E. Treatment of cutaneous larva migrans. Clin Infect Dis 2000; 30:811–814.

46. Dourmishev AL, Dourmishev LA, Schwartz RA. Ivermectin: pharmacology and application in dermatology. Int J Dermatol 2005; 44:981–988.

47. Caumes E, Carriere J, Datry A, et al. A randomized trial of ivermectin versus albendazole for the treatment of cutaneous larva migrans. Am J Trop Med Hyg 1993; 49:641–644.

48. Van den Enden E, Stevens A, Van Gompel A. Treatment of cutaneous larva migrans. N Engl J Med 1998; 339:1246–1247.

49. Jones SK, Reynolds NJ, Oliwiecki S, Harman RR. Oral albendazole for the treatment of cutaneous larva migrans. Br J Dermatol 1990; 122:99–101.

50. Sanguigni S, Marangi M, Teggi A, De Rosa F. Albendazole in the therapy of cutaneous larva migrans. Trans R Soc Trop Med Hyg 1990; 84:831.

51. Caumes E. Efficacy of albendazole ointment on cutaneous larva migrans in 2 young children. Clin Infect Dis 2004; 38:1647–1648.

52. Burry JN. No dogs on beaches, please! Med J Aust 1978; 1:40.