

# Cutaneous Manifestations of Selected Parasitic Infections in Western Pacific and Southeast Asian Regions

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**Abstract** Cutaneous manifestations of parasitic infections often result in discomfort, debilitation, and even stigmatization. Data on cutaneous manifestations of parasitic infections, however, are limited. This article provides updates on the cutaneous manifestations of parasitic infections which are known to occur in Western Pacific and Southeast Asian regions, such as scabies, pediculosis, cutaneous larva migrans, larva currens, cutaneous schistosomiasis, cutaneous enterobiasis, cutaneous cysticercosis, acute dermatolymphangioadenitis (lymphatic filariasis), and cutaneous amoebiasis. The lack of epidemiological data on these conditions suggests the need for improvements in recording and reporting of cases. Utilization of advance diagnostic modalities and capacity building of health workers are important for proper case management. Cutaneous manifestations of parasitic infections are a topic rarely studied and thus represent an opportunity for further research.

**Keywords** Parasitic infections · Pediculosis · Strongyloidiasis · Schistosomiasis · Enterobiasis · Amoebiasis · Cysticercosis · Acute dermatolymphangioadenitis · Emporiatrics

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## Introduction

Cutaneous manifestations of parasitic infections, such as cutaneous larva migrans (CLM, hookworm infection) and larva currens (strongyloidiasis), occur worldwide and have been known since ancient times [1]. These often result in discomfort, debilitation, and even stigmatization. Available data on cutaneous manifestations of parasitic infections are limited and new and atypical cutaneous manifestations are being described. Thus, there is a need to update the existing knowledge on cutaneous manifestations of parasitic infections. This will be important in guiding physicians and public health workers in managing these conditions. It may also guide policy makers in improving strategies on management of parasitic infections. This aim of this review is to provide updates on the etiology, pathology, clinical manifestations, epidemiology, diagnosis, and treatment of selected cutaneous manifestations of parasitic infections which are known to occur in Western Pacific and Southeast Asian regions.

## Methods

Searches of PubMed using Medical Subject Headings (MESH), and of Scopus and the Public Library of Sciences (PLOS) were performed using the search terms, “parasitic infections”, “cutaneous manifestation of parasitic infections”, “scabies”, “pediculosis”, “cutaneous larva migrans”, “hookworm infections”, “larva currens”, “strongyloidiasis”, “schistosomiasis”, “enterobiasis”, “cysticercosis”, “lymphatic filariasis”, “acute dermatolymphangioadenitis”, “amoebiasis”, and other related terms. Google Scholar, the websites of the World Health Organization (WHO) and the Centers for Disease Control and Prevention (CDC), and the University of the Philippines Manila College of Public Health

Library were also searched using similar key words. Articles gathered were examined and assessed for inclusion based on the authors' objectives.

## Results

The cutaneous manifestations of parasitic infections described in this article are those with relative importance in the Western Pacific and Southeast Asian regions. These include scabies, pediculosis, CLM, larva currens, cutaneous schistosomiasis, cutaneous enterobiasis, cutaneous cysticercosis, acute dermatolymphangioadenitis (lymphatic filariasis, LF), and cutaneous amoebiasis (Table 1).

### Scabies

Scabies, caused by *Sarcoptes scabiei* var. *hominis*, is a contagious skin infestation manifested as a pimple-like rash. It is commonly found on the hands, especially the web between the fingers, skin folds of the wrist, elbow or knee, penis, and breast or shoulder [2]. The pathognomonic sign is the burrow, characterized by a short, wavy, scaly, grey line on the skin surface [3]. Transmission occurs through direct skin-to-skin contact for at least 15 minutes to allow the transfer of mites from one person to another [4]. Crusted or Norwegian scabies, which is the severe form of scabies, may also be transmitted through fomites, such as infested clothing or bedding [5]. Common predisposing factors are overcrowding, migration, poor hygiene, poor nutritional status, homelessness, dementia, and sexual contact [4]. Scabies can also cause secondary bacterial skin infection or impetigo, which can lead to serious complications, such as septicemia, renal disease, and rheumatic heart disease [6].

Scabies is diagnosed through the burrow ink test and dermatoscopy with a hand-held dermatoscope along with clinical judgment [7]. Scabies is treated with permethrin, while malathion remains an excellent topical alternative. Ivermectin is also an effective oral treatment that is useful in crusted scabies, bed-ridden patients, and in institutional outbreaks [8]. Prevention and control of scabies is achieved through observing proper personal hygiene and avoiding physical contact with infected people and their clothing [9••]. Prevalence of scabies is highest in the Pacific and Latin American regions, and is substantially higher in children than in adolescents and adults [6].

### Pediculosis

Pediculosis, or lice infestation, may be caused by three species, which differ in localization: *Pediculus humanus capitis* (head louse), *P. humanus corporis* (body or clothes louse), and *Phthirus pubis* (crab or pubic louse) [9••].

Pruritus is the most common symptom of lice infestation. The location of pruritus can help identify the type of pediculosis. In head louse infestation, the scalp, the back of the neck, and postauricular areas are usually affected and symptoms may develop after 4 to 6 weeks. Scratching can cause secondary infection with bacterial sores. Patients infested with body lice, on the other hand, experience nocturnal pruritus, particularly in the axillary, truncal, and groin regions. Pubic lice infestations usually involve the groin and body hair. In addition to pruritus, patients may describe papules or wheals, indicating bite reactions [10]. Body lice may serve as vectors of diseases, such as typhus, trench fever, and relapsing fever [11].

Diagnosis of pediculosis requires identification of an adult louse and/or a viable nit. Cellulose tape may be used to isolate adult lice. Wood's lamp examination of the infested area shows yellow–green fluorescence of lice and nits. Dermatoscopy can be used to reliably differentiate nymph-containing eggs from empty cases or pseudonits [12].

Head and pubic lice infestations are treated with topical preparations of permethrin, lindane, malathion, or benzyl benzoate. Body lice infestations are treated with powdered preparations of lindane. Infestation of the eyelids, on the other hand, may be treated by applying a thick coat of petroleum jelly on the infested area [13]. Treatment should be repeated after 7–10 days, the time needed for the eggs to hatch, because nits are less effectively killed than adults [14].

Pediculosis may be under-reported because of social stigma, particularly the notion that it is related to poor personal hygiene. The prevalence of *Pediculus humanus capitis* infestation is usually higher in girls and women, and in Australia has been found to reach 13 % [15]. Children aged 3–11 years are most likely to become infested with head lice because of close contact in classrooms and day-care facilities. Body lice are more common in adults. *Pediculus humanus corporis* infestation is now uncommon in developed countries except among homeless persons [16]. *Phthirus pubis* infestation is more common in people aged 14–40 years who are sexually active [14].

### Cutaneous larva migrans

CLM is a condition in which the larvae of animal hookworms burrow through intact skin but remain confined to the upper dermis [9••]. Larval penetration may manifest as a skin rash called ground itch. CLM is mostly caused by *Ancylostoma braziliense* [17], but may also be caused by other species, such as *Gnathostoma* spp. [18], or even non-hookworm species, such as *Dirofilaria conjunctivae*, *Capillaria* spp., and *Strongyloides* spp. CLM cases caused by *Strongyloides* spp. may be accompanied by larva currens [19].

CLM is marked by a linear or serpiginous track known as a creeping eruption [9••] and intense pruritus often associated with pain. The most common site for creeping eruption is the

**Table 1** Cutaneous manifestations of selected parasitic infections

Parasite	Cutaneous manifestations	
Ectoparasites	<i>Sarcoptes scabiei</i> var. <i>hominis</i>	Scabies
	<i>Pediculus humanus capitis</i>	Pediculosis
	<i>Pediculus humanus corporis</i>	
	<i>Pthirus pubis</i>	
Helminths	Animal hookworm	Cutaneous larva migrans
	<i>Strongyloides</i> spp.	Larva currens
	<i>Schistosoma</i> spp.	Cutaneous schistosomiasis
	<i>Enterobius vermicularis</i>	Cutaneous enterobiasis
	<i>Taenia solium</i>	Cutaneous cysticercosis
	<i>Wuchereria bancrofti</i>	Acute dermatolymphangioadenitis (lymphatic filariasis)
	<i>Brugia malayi</i>	
Protozoa	<i>Brugia timori</i>	
	<i>Entamoeba histolytica</i>	Cutaneous amoebiasis

dorsal side of the feet [19], followed by the hands, arms, buttocks, and genitalia [17]. It may also be found in various sites, such as the scalp [20], oral mucosa [21], and on the trunk [22].

CLM begins as a transient reddish papule appearing within a few hours of larval penetration. The characteristic erythematous, slightly raised, and vesicular lesions, 2–4 mm wide, eventually appear in a serpiginous track and reach 15–20 cm in length. The migratory nature of the lesion varies, as larvae of various hookworm species travel between a few millimeters and 2 cm per day. The incubation period, likewise, may vary with the hookworm species, but the onset of symptoms usually occurs in the range 1–6 days after exposure. It is possible, however, that the larva may remain dormant in the skin for several weeks or months before migrating [17]. Less classic presentations of CLM may include folliculitis, eczematous eruptions [17], and vesicobullous lesions [23].

CLM is diagnosed clinically based on the presence of the characteristic signs and symptoms and exposure history. No serological testing for zoonotic hookworm infection is available, while skin biopsy has low sensitivity [9••]. While CLM is self-limiting, anthelmintics, such as albendazole and ivermectin, given orally are commonly used to treat hookworm infections. Topical treatment may be applied over the affected areas of the skin, but may not be effective since the larvae are mobile [9••].

CLM has a worldwide distribution [17] and is the most frequent skin disease among travelers returning from tropical and subtropical countries [20]. It affects various age groups from school-age children [20] to older adults [22].

### Larva currens

Larva currens, caused by *Strongyloides* spp., is a recurrent serpiginous maculopapular or urticarial rash on the buttocks, perineum, and thighs due to autoinfection [9••]. The condition is pathognomonic of strongyloidiasis [9••], most often

*S. stercoralis*, and very rarely *S. fülleborni* [17]. The larvae migrate more rapidly than in CLM at up to 10 cm/h [17].

Unusual manifestations of larva currens may include presentation as linear urticaria [24], as well as extensive purpura and skin invasion due to migrating larvae [25]. It may also appear as generalized petechial papules concentrated on the trunk [26] or as progressive purpuric petechial eruptions with a reticulated pattern concentrated over the abdomen [27]. Larva currens may be latent and may manifest as long as 38 years after exposure [28]. Disseminated strongyloidiasis, which may present as larva currens, may be triggered in immunocompromised individuals [26, 27].

The diagnosis of larva currens may be based on clinical manifestations and travel history. Filariform larvae may also be demonstrated on histological examination of the site with rash and/or on dermal granulomas [26, 29]. Serial stool examination remains as the gold standard for the diagnosis of strongyloidiasis. The Baermann concentration and Harada-Mori methods are also used as alternative diagnostic tools due to the low sensitivity of microscopy [9••].

Strongyloidiasis is treated with a single dose or two daily doses of ivermectin. Albendazole may also be given orally two times a day for 7 days as an alternative treatment [9••]. If not treated, larva currens has been reported to persist for as long as 65 years due to autoinfection [30].

Strongyloidiasis is known to occur in all continents, but is most common in the tropics, subtropics, and warm temperate regions. There are limited epidemiological data available on strongyloidiasis and larva currens, but it is estimated that 30–100 million people have strongyloidiasis worldwide [9••].

### Cutaneous schistosomiasis

Schistosomiasis, caused by *Schistosoma* spp. blood flukes and known for its visceral form, may also present with cutaneous manifestations [31]. Cutaneous schistosomiasis may be caused

by various schistosome species including *S. haematobium*, *S. mansoni*, and *S. japonicum* [32]. The primary risk factor is exposure to fresh water contaminated with schistosome cercaria [9••].

Cutaneous manifestations of schistosomiasis may occur with any schistosomal species, but most prominently *S. japonicum* [33•]. Cercarial dermatitis may or may not occur after cercarial penetration. This depends on previous exposures or sensitization and whether the patient is a native of an endemic area. In the Philippines, 4 out of 42 American soldiers who went to Leyte Province in 1944 experienced itching, which may have been cercarial dermatitis. Cercarial dermatitis is rarely seen among natives of endemic areas, which may be due to prenatal induction of tolerance. During initial infection or exposure to cercariae, there is minimal cellular response at the site of penetration even up to 48 h [34].

Cutaneous manifestations of schistosomiasis may also include erythematous, pruritic or asymptomatic isolated or coalescent papules with a zosteriform distribution [35]. Lesions caused by the disease may be 3–7 mm in diameter [36]. Lesions may appear as clustered reddish macules and papules [37]. In the majority of patients the anogenital region is affected because this region is perfused by the venous circulation, and is contiguous with the usual location of the worms [35]. Few extragenital cases have been reported [38] in sites, such as the neck, back, abdomen, scapular, and forehead regions [32, 36].

Cutaneous schistosomiasis may occur during the invasive or oviposition stages. During the invasive stage, nonspecific manifestations include severe itching and a generalized anaphylactoid reaction with an urticarial or erythema multiforme-like eruption. During the oviposition stage, on the other hand, specific manifestations include genital, perigenital, and extragenital cutaneous schistosomal granulomata [39]. Late cutaneous schistosomiasis is usually preceded or accompanied by visceral schistosomiasis. Cutaneous schistosomiasis as an isolated skin manifestation without systemic involvement, however, has also been reported [32, 40].

Cutaneous schistosomiasis is rare and difficult to diagnose clinically, even in areas where the disease is endemic [41]. Skin biopsy of the lesions often reveals a dermal granuloma surrounding schistosome eggs [42]. Schistosomiasis is treated with a single dose of praziquantel. Dermatological cases may be treated with niridazole or oxamniquine [43].

### Cutaneous enterobiasis

Enterobiasis, caused by *Enterobius vermicularis*, is transmitted through ingestion or inhalation of embryonated eggs and autoinfection through hand-to-mouth spread [44]. The primary cutaneous manifestation of enterobiasis is nocturnal anal and perianal pruritus that begins 4–6 weeks after infection and may last for months [45]. This is caused by the migration of female worms depositing eggs in the perianal area [44].

Scratching may cause skin irritation, and in more serious cases, eczematous dermatitis, hemorrhage or secondary bacterial infections [46].

*Enterobius vermicularis*, if found in ectopic locations, can elicit a severe granulomatous inflammation [44]. Ectopic migration of *E. vermicularis* often results in infestation of the genital tract in women. This may cause granuloma of the uterus, ovary, fallopian tubes, and pelvic peritoneum [46]. In some cases, this may result in a polypoidal mass in the anal region [44] and pruritus vulvae with vaginal discharge [47]. Vulval involvement in enterobiasis has been mistaken for carcinoma because of the presence of differentiated cell proliferation [48].

Enterobiasis is diagnosed using the “Scotch test”, or the cellulose-tape slide test, performed in the morning before defecation [9••]. Enterobiasis can also be diagnosed by demonstrating helminth eggs in the feces (incidental), perianal scrapings or swabs from under the fingernails, or by finding adult worms around the anus, usually at night. Albendazole is the treatment of choice, although mebendazole and pyrantel pamoate are also effective. Considering its familial distribution, the entire family should be treated to prevent reinfection [49]. Enterobiasis is common among children and institutionalized persons. It is estimated that more than 200 million people are infected worldwide [44].

### Cutaneous cysticercosis

Cysticercosis, caused by the larvae of *Taenia solium*, occurs when a person ingests the parasite’s eggs. The larvae lodge in the tissues, including muscle and brain tissues, and form spherical milky white cysts or cysticerci [9••].

Approximately half of all cysticercosis cases have cutaneous manifestations [50]. Cysticercus lesions are seen as well-defined anechoic or hypoechoic lesions with or without calcification. The number of lesions may vary and may be too many to count. Cysticercosis also has the uncommon etiology of subcutaneous swellings in children [51]. Although less common, cutaneous cysticercosis may also result in neurocysticercosis [52].

Diagnosis of cysticercosis requires imaging and serological examinations. The history of travel and food intake can also be considered [9••]. Treatment of cutaneous cysticercosis may include the use of anthelmintics (albendazole or praziquantel) and surgery. There is, however, no single, universally accepted treatment regimen for the condition [53]. Cases with cutaneous involvement, or cysticercosis cutis, have been reported in India, Africa, Mexico, and South America [52].

### Acute dermatolymphangioadenitis (lymphatic filariasis)

LF is a parasitic infection caused by *Wuchereria bancrofti* and *Brugia malayi* [9••]. Cutaneous manifestations of LF

include acute dermatolymphangioadenitis. Other cutaneous manifestations may include facial infestation with the filarial worm [54] and vulval filarial elephantiasis [55]. Testicular involvement in LF is not limited to hydrocele; testicular swelling caused by filarial worms mimicking neoplasms has been reported [56]. Likewise, elephantiasis may be caused by nonspecific chronic inflammation as well as by filarial worms. [57].

The immunochromatographic card test, which detects circulating filarial antigen, is considered the gold standard for LF diagnosis [58, 59]. Active filarial infection is typically associated with elevated levels of antifilarial IgG4 in the blood, and this can be detected using routine assays [9••]. Diagnosis of LF may also be based on the demonstration of microfilariae in the blood by microscopy. Blood is collected at night to coincide with the appearance of the microfilariae which show nocturnal periodicity [9••]. Microfilariae may not be seen in chronic infections due to various factors, including low intensity of infection, dead worms, and obstructed lymph vessels. In low-intensity infections, filtration using a nucleopore filter or Knott's method for concentration may be required to identify the microfilariae. The diethylcarbamazine (DEC) provocation test [60] stimulates microfilariae to come out into the peripheral circulation allowing blood smear collection even during the day time.

Annual mass administration of DEC and albendazole combined to all individuals at risk is recommended to interrupt transmission and prevent new cases of infection [58]. Ivermectin is also effective against LF.

Currently, over 120 million people are infected, with about 40 million disfigured and incapacitated by the disease [58]. Bancroftian filariasis accounts for 90 % of cases in endemic countries and affects more than 100 million people in tropical areas, such as Southeast Asia and the Pacific Islands. *Brugia malayi* and *B. timori* infections affect 12.5 million people in Southeast Asia [61].

### Cutaneous amoebiasis

Amoebiasis, caused by *Entamoeba histolytica*, can destroy various tissues, such as the intestinal mucosa, liver and to a lesser extent the brain and skin [62•]. It is transmitted via direct or indirect fecal–oral route [63].

Cutaneous amoebiasis is caused by infection of wounds with *E. histolytica* trophozoites. It may present as a well-defined, indurated, painful, progressively enlarging plaque with overlying ulcers and sinuses discharging pus [64]. Amoebiasis may also manifest as skin abscess, which may start as erythematous nodules and erythema nodosum [65].

Clinical suspicion and a simple wet drop examination may be used to diagnose the lesion [66]. Sensitivity is increased when tissues or smears are prepared from the edges of the ulcer [67]. Erythrophagocytosis is a sign of the pathogenicity

of the disease [68]. Oral metronidazole is the drug of choice for amoebiasis [9••].

### Conclusions

Cutaneous parasitic infections differ considerably in their biological and epidemiological manifestations as well as life cycles [1]. Cutaneous manifestations of ectoparasitic infections, such as scabies and pediculosis, as well as some endoparasitic infections, such as cysticercosis and amoebiasis result in considerable discomfort. These conditions require prompt treatment to alleviate the discomfort of patients. On the other hand, cutaneous manifestations of other endoparasitic infections, such as hookworm infection, strongyloidiasis, schistosomiasis, LF and enterobiasis, are less severe and cause less discomfort. Likewise, some cutaneous manifestations may appear late in the course of the disease. These manifestations, however, may be useful indicators in a patient's history.

The increase in travel makes the epidemiology of cutaneous manifestations of parasitic infections more complicated [69]. Cases are no longer limited to tropical areas where these diseases are endemic. Unfortunately, physicians and other health workers may not be familiar with such conditions which may thus be misdiagnosed. Misdiagnosis, which remains a continuing challenge in addressing cutaneous manifestations of parasitic infections, may lead to continuing morbidity and reduced productivity.

Orientation and training of health workers, even those from non-endemic areas, may be important to ensure proper treatment and management of patients. The field of emporiatrics, which deals with the prevention and management of health problems of international travelers, can be introduced to health workers. The development and application of more advanced diagnostic modalities will also be important to prevent misdiagnosis. Good history taking and physical examination also remain indispensable in obtaining an accurate diagnosis and thus may be emphasized to health workers, especially to physicians.

There are existing knowledge gaps on cutaneous manifestations of parasitic infections. Most of the available data are limited to case reports and series. This may limit the usefulness of these reports in guiding physicians in the diagnosis and management of patients with parasitic infections. Thus there is an opportunity for further research, especially more reliable studies on cutaneous manifestations of parasitic infections. These studies could focus on various aspects, such as spatial distribution, incidence and prevalence, seasonal variation, and risk factors for the development of severe disease. Such studies could also systematically assess disease occurrence and morbidity [1]. Addressing cutaneous manifestations of parasitic infections as a public health problem will require more collaboration between experts in medical parasitology and dermatology.



## Compliance with Ethical Standards

**Conflicts of Interest** Drs. Belizario, Caesar delos Trinos, Garcia and Reyes declare no conflicts of interest.

**Human and Animal Rights and Informed Consent** This article does not describe studies with human or animal subjects performed by any of the authors.

## References

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. Feldmeier H, Heukelbach J. Epidermal parasitic skin diseases: a neglected category of poverty-associated plagues. *Bull World Health Organ.* 2009;87:152–9.
2. World Health Organization. Water-related diseases: scabies. 2016. [http://www.who.int/water\\_sanitation\\_health/diseases/scabies/en/](http://www.who.int/water_sanitation_health/diseases/scabies/en/). Accessed 13 Jul 2016.
3. Johnston G, Sladden M. Scabies: diagnosis and treatment. *BMJ.* 2005;331:619–22.
4. Hicks M, Elston D. Scabies. *Dermatol Ther.* 2009;22:279–92.
5. Monsel G, Chosidow O. Management of scabies. *Skin Ther Lett.* 2012;17:1–4.
6. Romani L, Steer A, Whitfeld M, Kaldor M. Prevalence of scabies and impetigo worldwide: a systematic review. *Lancet Infect Dis.* 2015;15:960–67.
7. Leung V, Miller M. Detection of scabies: a systematic review of diagnostic methods. *Can J Infect Dis Med Microbiol.* 2011;22:143–46.
8. Golant A, Levitt J. Scabies: a review of diagnosis and management based on mite biology. *Pediatr Rev.* 2012;33:e1–e12.
9. •• Centers for Disease Control and Prevention. Parasites - scabies: prevention and control. 2015. <http://www.cdc.gov/parasites/scabies/prevent.html>. Comprehensive reference for etiology, pathology, diagnosis, and treatment of various parasitic infections.
10. Centers for Disease Control and Prevention. Pediculosis. 2013. <http://www.cdc.gov/dpdx/pediculosis/>. Accessed 13 Jul 2016.
11. Badiaga S, Brouqui P. Human louse-transmitted infectious diseases. *Clin Microbiol Infect.* 2012;18:332–37.
12. Di Stefani A, Hofmann-Wellenhof R, Zalaudek I. Dermoscopy for diagnosis and treatment monitoring of pediculosis capitis. *J Am Acad Dermatol.* 2006;54:909–11.
13. Centers for Disease Control and Prevention 2015). Pediculosis. Retrieved from [www.cdc.gov/parasites/http://www.cdc.gov/parasites/scabies/prevent.html](http://www.cdc.gov/parasites/http://www.cdc.gov/parasites/scabies/prevent.html).
14. Guenther LC, Maguiness S. Pediculosis and phthiasis (lice infestation). 2016. [emedicine.medscape.com/article/225013-overview](http://emedicine.medscape.com/article/225013-overview). Accessed 13 Jul 2016.
15. Falagas M, Matthaiou D, Rafailidis P, Panos G, Pappas G. Worldwide prevalence of head lice. *Emerg Infect Dis.* 2008;14:1493–4.
16. Elston DM. Treating pediculosis – those nit-picking details. *Pediatr Dermatol.* 2007;24:415–6.
17. Meinking T, Burkhart C, Burkhart C. Changing paradigms in parasitic infections: common dermatological helminthic infections and cutaneous myiasis. *Clin Dermatol.* 2003;21:407–16.
18. Mukherjee A, Ahmed NH, Samantaray JC, Mirdha BR. A rare case of cutaneous larva migrans due to *Gnathostoma* sp. *Indian Med Microbiol.* 2012;30:356–8.
19. Corte L, da Silva M, Souza P. Simultaneous larva migrans and larva currens caused by *Strongyloides stercoralis*: a case report. *Case Rep Dermatol Med.* 2013;2013:381583.
20. Meotti C, Plates G, Nogueira L, Silva R, Paolini K, Nunes E, et al. Cutaneous larva migrans on the scalp: unusual presentation in a typical clinical presentation. *An Bras Dermatol.* 2014;89:332–3.
21. Damante J, Chinellato L, de Oliveira F, Soares C, Fleury R. Larva migrans in the oral mucosa: report of two cases. *Braz Dent J.* 2011;22:166–70.
22. Sharma A, Hazarika NK, Gupta D. Extensive cutaneous larva migrans – a case report. *Indian J Public Health Res Dev.* 2014;5:287–90.
23. Gupta M. Bullous cutaneous larva migrans – a case report. *J Dermatol Dermatol Surg.* 2016;20:65–6.
24. Cunliffe W, Silva L. Linear urticaria due to larva currens. *Br J Dermatol.* 1968;80:108–10.
25. von Kuster L, Genta R. Cutaneous manifestations of strongyloidiasis. *Arch Dermatol.* 1988;124:1826–30.
26. Kao D, Murakawa G, Kerschmann R, Berger T. Disseminated strongyloidiasis in a patient with acquired immunodeficiency syndrome. *Arch Dermatol.* 1996;132:977–8.
27. Galimberti R, Ponton A, Zaputovich F, Velasquez L, Galimberti G, Torre A, et al. Disseminated strongyloidiasis in immunocompromised patients – report of three cases. *Int J Dermatol.* 2009;48:975–8.
28. Showler A, Boggild A. Strongyloidiasis presenting as larva currens 38 years after presumed exposure. *J Cutan Med Surg.* 2012;16:433–5.
29. Gordon S, Gal A, Solomon A, Bryan J. Disseminated strongyloidiasis with cutaneous manifestations in an immunocompromised host. *J Am Acad Dermatol.* 1994;31:255–9.
30. Leighton P, MacSween H. *Strongyloides stercoralis*: the cause of an urticarial-like eruption of 65 years' duration. *Arch Intern Med.* 1990;150:1747–8.
31. World Health Organization. Schistosomiasis. 2016. <http://www.who.int/mediacentre/factsheets/fs115/en/>. Accessed 13 Jul 2016.
32. Nunes K, Cardoso A, Pereira F, Batista L, Houly R. Ectopic cutaneous schistosomiasis – case report. *An Bras Dermatol.* 2013;88:969–72.
33. • Barsoum RS, Esmat G, El-Baz T. Human schistosomiasis: clinical perspective: review. *J Adv Res.* 2013;4:433–44. **A comprehensive review of clinical aspects of schistosomiasis.**
34. Garcia EG. State of the art: Schistosomiasis japonica. Philippine Council for Health Research and Development, Department of Health, The Philippines; 1988.
35. Mota LS, de Silva SF, Almeida FC, Mesquita LS, Teixeira RD, Soares AM. Ectopic cutaneous schistosomiasis – case report. *An Bras Dermatol.* 2014;89:646–8.
36. Atanda A, Mohammad M, Atallah L. Cutaneous schistosomiasis: case report and literature review. *Ann Nigerian Med.* 2012;6:98–100.
37. Vargas T, Lopes R, Moraes M, de Azevedo K, Sousa M. Ectopic cutaneous schistosomiasis. *An Bras Dermatol.* 2013;88:820–2.
38. Tranquillini G, de Freitas Ferreira Hostalçacio I, Tadeu Villa R, Guevara Silva LA, Leitão R, Bedin V. Ectopic cutaneous schistosomiasis: case report. *Med Cutan Iber Lat Am.* 2011;39:268–71.
39. El Mofly A, Nada M. Cutaneous schistosomiasis. *Egypt J Bilharz.* 1975;2:23–30.
40. Kick G, Schaller M, Korting H. Late cutaneous schistosomiasis representing an isolated skin manifestation of *Schistosoma mansoni* infection. *Dermatology.* 2000;200:144–6.
41. Al-Samawi A. Case report cutaneous schistosomiasis. *Yemeni J Med Sci.* 2014;8:37–9.
42. Attia S, Mofteh N, Abdel-Aziz E. Expression of IFN- $\gamma$ , IL-4, and IL-17 in cutaneous schistosomal granuloma. *Int J Dermatol.* 2014;53:991–8.
43. Poderoso W, Santana WB, Costa EF, Cipolotti R, Fakhouri R. Ectopic schistosomiasis: description of five cases involving skin, one ovarian case and one adrenal case. *Rev Soc Bras Med Trop.* 2008;41:668–71.
44. Bharathi K, Anuradha S, Chandrasekar V, Thirunarayanan R. *Enterobius vermicularis* worm granuloma mimicking like a pseudo

- tumor in the anal canal: an unusual clinical presentation. *Trop Parasitol.* 2012;2:124–6.
45. Caumes E. Helminthic skin diseases in travelers. In: Schwartz E, editor. *Tropical diseases in travelers.* Oxford: Wiley; 2009. p. 341–51.
  46. St Georgiev V. Chemotherapy of enterobiasis (oxyuriasis). *Expert Opin Pharmacother.* 2001;2:267–75.
  47. Shetty J, Kulkarni D, Prabhu V. Eggs containing larvae of *Enterobius vermicularis* in vaginal smear. *J Cytol.* 2012;29:94–6.
  48. Konanahalli P, Menon P, Walsh M, McCluggage W. *Enterobius vermicularis* (pinworm) infestation of the vulva: report of 2 cases of a pseudoneoplastic lesion mimicking squamous carcinoma. *Int J Gynecol Pathol.* 2010;29:490–3.
  49. Cook G, Zumla A. *Manson's tropical diseases.* 22nd ed. Philadelphia: Saunders Elsevier; 2009.
  50. Wolff K, Goldsmith L, Katz S, Gilchrist B, Paller A, Leffell D. *Fitzpatrick's dermatology in general medicine.* 7th ed. New York: McGraw Hill; 2008. p. 2027–8.
  51. Prasad K, Prasad A, Verma A, Singh A. Human cysticercosis and Indian scenario: a review. *J Biosci.* 2008;33:571–82.
  52. Sacchidanand S, Namitha P, Mallikarjuna M, Nataraj H. Disseminated cutaneous cysticercosis and neurocysticercosis: a rare occurrence. *Indian Dermatol Online J.* 2012;3:135–7.
  53. Kraft R. Cysticercosis: an emerging parasitic disease. *Am Fam Physician.* 2007;76:91–6.
  54. Vaid S, Luthra A, Karnik S, Ahuja A. Facial wrigglies: live extralymphatic filarial infestation in subcutaneous tissues of the head and neck. *Br J Radiol.* 2011;84:e126–9.
  55. Ipyana HM, Bonaventura CT, Januarius H. Vulval filarial elephantiasis in a Tanzanian woman; rare presentation of lymphatic filariasis: a case report and a review of literature. *Sudan JMS.* 2014;9:265–9.
  56. Barreto S, Rodrigues J, Roque G. Filarial granuloma of the testicular tunic mimicking a testicular neoplasm: a case report. *J Med Case Rep.* 2008;2:321.
  57. Denzinger S, Watzlawek E, Burger M, Wieland W, Otto W. Giant scrotal elephantiasis of inflammatory etiology: a case report. *J Med Case Rep.* 2007;1:23.
  58. World Health Organization. Lymphatic filariasis. 2016. [www.who.int/mediacentre/factsheets/fs102/en/](http://www.who.int/mediacentre/factsheets/fs102/en/). Accessed 13 Jul 2016.
  59. Dreyer G, Lins R, Norões J, Rizzo J, Figueredo-Silva J. Sensitivity of the immunochromatographic card test relative to detection of adult *Wuchereria bancrofti* worms by ultrasound. *Am J Trop Med Hyg.* 2008;78:28–34.
  60. Wijeyaratne P, Singha P, Verma O, Motha B. Evaluation of the diethylcarbamazine provocative test in the diagnosis of *Wuchereria bancrofti* infections in the Nigerian savanna and the effects on *Dipetalonema perstans*. *Trans R Soc Trop Med Hyg.* 1982;76:387–91.
  61. World Health Organization, Philippines. Neglected tropical diseases in the Philippines. 2015. [www.wpro.who.int/philippines/areas/communicable\\_diseases/mvp/story\\_ntd/en/index1.html](http://www.wpro.who.int/philippines/areas/communicable_diseases/mvp/story_ntd/en/index1.html). Accessed 13 Jul 2016.
  62. Kelly P. Intestinal protozoa. In: Farrar J, Hotez P, Junghans T, Kang G, Lalloo D, White NJ, editors. *Manson's tropical diseases.* 23rd ed. Philadelphia: Saunders Elsevier; 2013. **Provides comprehensive information on intestinal protozoa.**
  63. World Health Organization. Amoebiasis. 2015. <http://www.who.int/ith/diseases/amoebiasis/en/>. Accessed 13 Jul 2016.
  64. Verma G, Sharma N, Shanker V, Mahajan V, Kaushik R, Verma S, et al. Amoebiasis cutis: clinical suspicion is the key to early diagnosis. *Australas J Dermatol.* 2010;51:52–5.
  65. Satish G, Rajam L, Regi S, Nazar PK. Multiple amoebic abscesses with erythema nodosum. *Indian J Pediatr.* 2012;79:532–4.
  66. Fernández-Diez J, Magaña M, Magaña M. Cutaneous amoebiasis: 50 years of experience. *Cutis.* 2012;90:310–4.
  67. Parshad S, Grover P, Sharma A, Verma D, Sharma A. Primary cutaneous amoebiasis: case report with review of the literature. *Int J Dermatol.* 2002;41:676–80.
  68. Magaña M, Magaña M, Alcántara A, Pérez-Martín M. Histopathology of cutaneous amoebiasis. *Am J Dermatopathol.* 2004;26:280–4.
  69. Moro P. Echinococcosis. In: Centers for Disease Control and Prevention. *Infectious diseases related to travel.* Centers for Disease Control and Prevention. 2015. <http://wwwnc.cdc.gov/travel/yellowbook/2016/infectious-diseases-related-to-travel/echinococcosis>. Accessed 13 Jul 2016.