Ectoparasites

Pediculosis and tungiasis

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Learning objectives
After completing this learning objective participants should be able to discuss the clinical features and risk factors for body louse infestation, including rising homelessness in US coastal cities; describe the emerging evidence that head lice may act as a vector for severe infectious diseases; review geographic distribution, clinical signs, and risk factors for tungiasis; and identify promising new treatments for tungiasis.

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Pediculosis is an infestation of lice on the body, head, or pubic region that occurs worldwide. Lice are ectoparasites of the order Phthiraptera that feed on the blood of infested hosts. Their morphotype dictates their clinical features. Body lice may transmit bacterial pathogens that cause trench fever, relapsing fever, and epidemic typhus, which are potentially life-threatening diseases that remain relevant in contemporary times. Recent data from some settings suggest that head lice may harbor pathogens. The epidemiology, clinical manifestations, and management of body, head, and pubic louse infestation are reviewed. New therapies for head lice and screening considerations for pubic lice are discussed. Tungiasis is an ectoparasitic disease caused by skin penetration by the female *Tunga penetrans* or, less commonly, *Tunga trimamillata* flea. It is endemic in Latin America, the Caribbean and sub-Saharan Africa and seen in travelers returning from these regions. Risk factors for acquiring tungiasis, associated morbidity, and potential strategies for prevention and treatment are discussed. (J Am Acad Dermatol 2020;82:551-69.)

Key words: body lice; ectoparasite; epidemic typhus; flea; head lice; homeless; homelessness; infestation; lice; pediculosis; *Pediculus capitis*; *Pediculus humanus*; *Phthirus pubis*; poverty; pubic lice; refugee; relapsing fever; returning traveler; trench fever; *Tunga penetrans*; *Tunga trimamillata*; tungiasis.

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HUMAN BODY LOUSE (*PEDICULUS HUMANUS HUMANUS*)

Key points

- Body lice infestation is associated with poor hygiene or neglect
- Given a homelessness epidemic, the diagnosis and management of body lice are essential dermatology skills
- Body lice are vectors for potentially life-threatening pathogens

Lice are obligate parasites, feeding exclusively on the blood of infested hosts.[1] Among thousands of lice species, only *Pediculus humanus* (*P humanus*) and *Phthirus pubis* (pubic lice) require humans as hosts. *P humanus* includes 2 morphotypes: *P humanus corporis* (body) and *P humanus capitis* (head) lice.[2]

Body lice are ovoid-shaped and measure 2.3 to 3.6 mm (Fig 1, *A*).[3] They live on clothing and attach to nearby skin for blood meals. Females lay eggs ("nits") on clothing seams (Fig 1, *B*). In favorable environments, eggs hatch into nymphs after 6 to 10 days, which mature into adults that live 1 to 3 months.[2] Detached from hosts, lice die within 3 to 5 days.[2] Their ideal survival conditions are 29°C to 32°C and 70% to 90% humidity; they cannot survive above 50°C or below 40% humidity.[4]

Epidemiology

Lice infestations are not reportable diseases in most countries, limiting high-quality epidemiologic data collection.[5] Known risk factors include close contact with infested persons, infrequent showering, an inability to wash and heat-dry clothing, and cold weather, which decreases the frequency of showering and changing clothes. These are especially prevalent among homeless persons,[4,6,7] making body lice infestation more common in this population.[7-10] In urban settings of high-income countries, ≤30% of homeless persons are infested with body lice.[8] Unsheltered persons are more frequently affected.[8,9] Additional risk factors include substance abuse, not showering, and previous pubic lice infestation.[9,10] Untreated mental illness, which is more prevalent among homeless people in Western countries compared with the general population,[11,12] may contribute to poor hygiene. Known prevalence and risk factors for infestation among homeless persons are shown in Table I.

The United States is currently experiencing a homelessness "epidemic," particularly on the West Coast, where several cities have declared states of emergency.[13-15] From 2016 to 2017, homelessness increased for the first time in 7 years, by 9%, mostly because of a rise in unsheltered homeless persons.[14] Chronic (>1 year) homelessness also increased by 12%.[14] During this period, homeless populations have risen in several European countries.[16]

Clinical manifestations

Patients with body lice infestation present with generalized pruritus and lesions distributed on the neck, shoulders, upper back, flanks, and waist—sites of close contact between clothing and the skin (Fig 2). Common lesions include excoriations and eczematous patches; papular urticaria and bullae may be seen. Prurigo nodules, lichenification, and hyperpigmentation are present in chronic infestation. Scratching predisposes to impetigo, ecthyma, and cellulitis. Diagnosis is achieved by carefully examining clothing seams ("clothing biopsy") for lice and nits. Severe iron deficiency anemia[17-19] and eosinophilia[17,19] have been associated with heavy/chronic body lice infestation.

Implications

Body lice transmit several potentially life-threatening infections (Table II). Despite being rare overall, these conditions remain major public health concerns in poor hygiene conditions.

Body lice transmit *Bartonella quintana*, which causes trench fever, named for its high prevalence among soldiers fighting together in unhygienic trenches during World War I.[20,21] More recently, trench fever was seen in refugees after the civil war in Burundi,[22] and since the 1990s, "urban trench fever" has been recognized in homeless and urban poor populations worldwide.[23-25] *B quintana* was identified in 21% of body lice in Marseilles, France,[26] 28.2% in Bogotá, Colombia,[27] 13.35% in northern Algeria,[28] 13.1% in Turkey,[29] and 15.9% in San Francisco, California.[8] Trench fever classically manifests as headache, dizziness, conjunctival injection, severe shin pain, lymphadenopathy, a macular evanescent rash, and relapsing fevers lasting 4 to 8 days.[2,21,30] Contemporary *B quintana* infections present with variable manifestations: asymptomatic infections, relapsing fevers, headache, leg pain, "culture-negative" endocarditis, and, in immunocompromised persons with HIV or a previous transplant, bacillary angiomatosis.[25,31,32] Diagnosis relies on culture, serology, immunohistochemical staining, or polymerase chain reaction assay. *B quintana* grows slowly in culture, often requiring ≥14 days and up to 45 days.[33] The microbiology laboratory should be notified when

Abbreviations used:

FDA: US Food and Drug Administration
LBRF: louse-borne relapsing fever
RCT: randomized controlled trial

Bartonella is suspected because several techniques exist to optimize growth.[33] First-line treatment is gentamicin (3 mg/kg/day for 2 weeks) followed by doxycycline (200 mg/day for 4 weeks).[30] In bacillary angiomatosis, first-line treatment is erythromycin (500 mg 4 times daily) or doxycycline (100 mg 2 times daily) for ≤3 months in immunocompromised hosts.[30,34]

The body louse also transmits *Borrelia recurrentis*, which causes louse-borne relapsing fever (LBRF). Most LBRF cases originate from the horn of Africa (Ethiopia, Eritrea, and Somalia), where *B recurrentis* is endemic and concentrated poverty contributes to sporadic epidemics.[35,36] With the ongoing global refugee crisis, LBRF is an emerging infectious disease in Europe, seen in refugees and asylum seekers who acquired the infection in the horn of Africa, either during their journey through North Africa where migration routes into Europe join or in Europe through contact with new refugee arrivals.[37-46] LBRF presents with an initial phase of high-grade fever, headache, dizziness, myalgias, and fatigue,[35] followed by shorter, less severe relapses every 7 to 19 days.[2] Complications include mucocutaneous hemorrhage, neurologic dysfunction, and liver or renal failure. Mortality is ≤40% for untreated cases[2] and 2% to 5% when treated.[47] Serum polymerase chain reaction studies or observing organisms on blood smears confirms the diagnosis.[48] Treatment is with a single dose of intramuscular penicillin G (400,000-800,000 units) or doxycycline 200 mg once[2]; the optimal treatment remains unclear.[49] After antibiotic administration, observation for 4 to 6 hours is recommended[50] because a Jarisch–Herxheimer reaction is common. In LBRF, this typically occurs within 2 hours of antibiotic administration and manifests with chills/rigors, tachycardia, and hypotension that may require hospitalization for supportive care.[51,52]

P humanus also transmits *Rickettsia prowazekii*, which causes epidemic typhus. While infected lice die within weeks, humans are the principle reservoir, remaining infected for life.[22] The organism may reemerge years later during stressful periods.[22] Outbreaks are associated with war, famine, crowding, refugee camps, and cold weather.[22,53] Flying squirrels serve as a zoonotic reservoir in periods between outbreaks, when human-to-louse-to-human transmission is less common.[54] Widespread maculopapular exanthema that may become petechial are seen.[22] Other features include fever, nausea, diarrhea, delirium, respiratory failure, and shock.[2,22] Diagnosis is made via serology.[55] Mortality is ≤60% if untreated but approximately 4% when treated.[2] First-line treatment is doxycycline 200 mg once.[55,56]

Management

The management of body lice must address poor hygiene and clothing infestation. Regular showering/bathing is important. Clothes should be discarded appropriately or washed and dried using high heat (>50°C).[3] Associated bacterial infections should be treated with appropriate antibiotics and eczematous lesions with topical corticosteroids. Physicians should involve social services to address underlying factors driving infestation.

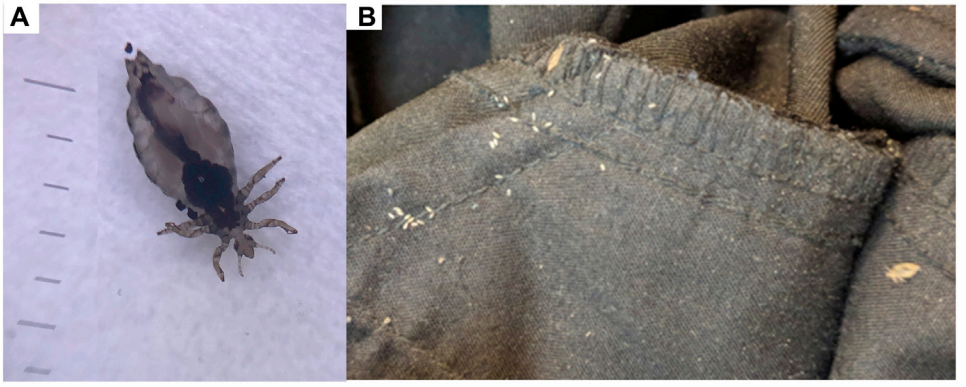

Fig 1. Body lice. **A**, Body louse morphology (courtesy of Charlotte Bernigaud, MD, Olivier Chosidow, MD, PhD, and Francoise Foulet, MD). **B**, Two lice and numerous nits (pinpoint white-yellow structures) in clothing seams (courtesy of Aileen Chang, MD).

Table I. Epidemiology of body lice infestation among homeless populations

Year	Location	Population	Prevalence of body lice	Risk factors for infestation
2014	San Francisco, California, United States[8]	203 sheltered and unsheltered homeless persons	30% with body lice, 4.9% with head lice, and 3% with body and head lice	Male sex, African American race, and sleeping outdoors
2016	Paris, France[9]	667 sheltered and 341 unsheltered homeless persons	5.4% of unsheltered persons with body lice and 0.15% of sheltered persons with body lice	Begging, history of pubic lice, and not taking showers in municipal baths
2017	Marseilles, France[10]	2288 sheltered homeless persons	12.2% with body lice, 4.5% with head lice, and 3.2% with pubic lice	Older age, alcohol consumption, and tobacco smoking
2017	Bogotá, Colombia[27]	153 sheltered homeless persons	11.7% with body lice	Not reported

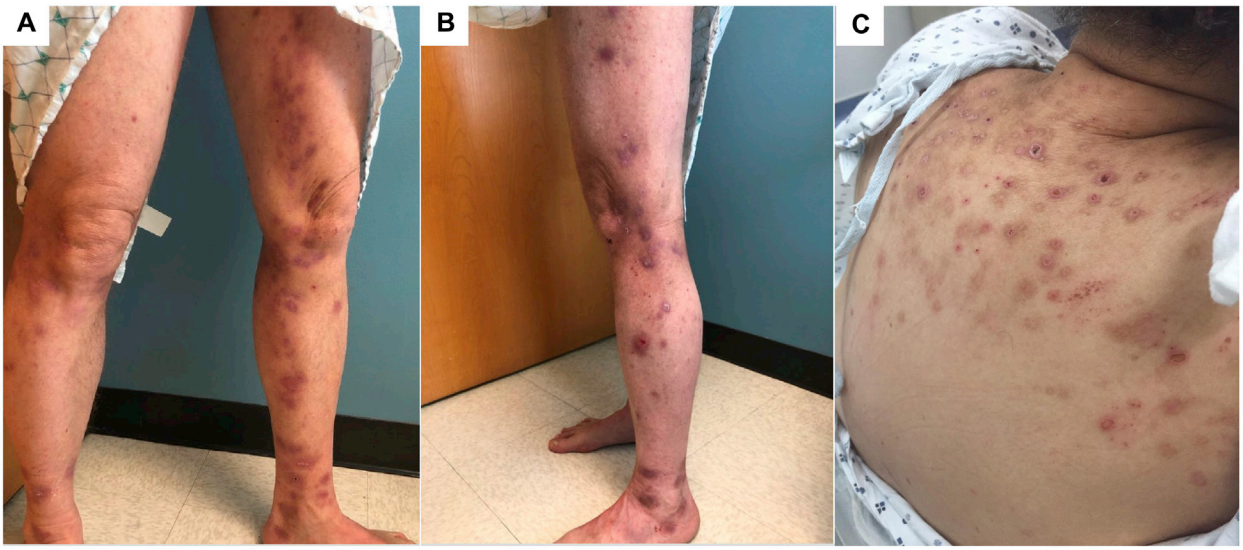

Fig 2. Clinical manifestations of body lice infestation. **A** and **B**, Bite marks along clothing seams that have evolved into prurigo nodules after chronic rubbing (courtesy of Sarah Coates, MD). **C**, Excoriations and prurigo nodules localized to upper back and shoulders in a patient with body lice infestation (courtesy of Kelly Fitzgerald, MD).

These measures can be effectively implemented during large outbreaks. In 1991, an Ethiopian refugee camp achieved large-scale delousing via shaving head and pubic hair, a 15-minute shower with soap, boiling clothing for 30 minutes, burning and replacing blankets, and spraying personal belongings with pesticide.[57] After a civil war in Burundi in the 1990s, a nationwide epidemic typhus outbreak was successfully managed with delousing interventions and doxycycline administered to all confirmed and suspected cases of typhus.[22]

In a randomized controlled trial (RCT) among sheltered homeless persons in France, persons who received permethrin-impregnated underwear were freer of lice (based on the absence of living lice in underwear) on day 14 compared with placebo, but these effects were not sustained 45 days later and thus this intervention was not recommended given the lack of sustained

Table II. Human pathogens associated with body lice infestation

Pathogen	Clinical manifestations	Microbiology	Diagnosis	Treatment
Bartonella quintana	Trench fever: headache, dizziness, severe shin pain, lymphadenopathy, macular evanescent rash, relapsing fevers lasting 4-8 days, recurring for weeks[2,21,30]; "culture-negative" endocarditis[174]; bacillary angiomatosis (in immunocompromised hosts)[31,32]	Facultative intracellular Gram-negative bacillus	Serology (anti-*Bartonella* antibodies); blood or tissue culture possible, but not sensitive. Slow growth in culture (≥14 days, ≤45 days)[33]	Gentamicin (3 mg/kg/day for 2 weeks), followed by doxycycline (200 mg/day for 4 weeks)[30]; erythromycin is the first-line treatment for bacillary angiomatosis
Borrelia recurrentis	Relapsing fever: high-grade fever, headache, dizziness, pain, anorexia, and fatigue; may progress to mucocutaneous hemorrhage, meningitis, encephalitis, liver and renal failure; relapses occur every 7-19 days[2]	Spirochete	Thin and thick blood smears to identify organisms; PCR testing of serum if smear negative; CSF PCR if neurologic signs are present	Single dose IM penicillin G (400,000-800,000 units); oral doxycycline, once (200 mg for adults) monitor for Jarisch—Herxheimer reaction during first 24 hours
Rickettsia prowazekii	Epidemic typhus: fever, acute exanthem, diarrhea, neurologic dysfunction, respiratory failure, and shock; organism may emerge during stressful periods years later[22]	Obligate intracellular bacillus; infected lice die within 3 weeks; humans remain infected for life	Diagnose via serology; isolation in culture is impractical	Oral doxycycline 100 mg BID for 7-10 days[55,56]; chloramphenicol 500 mg QID for 5 days (second-line); doxycycline 200 mg, once, in outbreaks[2]

BID, Twice daily; *CSF*, cerebrospinal fluid; *IM*, intramuscular; *PCR*, polymerase chain reaction; *QID*, 4 times daily.

Fig 3. Head lice. **A**, Head louse morphology (courtesy of Arezki Izri, MD, PhD). **B**, Head louse nit attached to a hair shaft, visualized under microscopy (courtesy of Charlotte Bernigaud, MD, Olivier Chosidow, MD, PhD, and Francoise Foulet, MD). **C**, Head louse nit on a hair shaft, visualized under dermoscopy (courtesy of Aileen Chang, MD).

Table III. Treatment of head lice

Therapy	Mechanism of action	Instructions	Adverse events	Level of evidence*	Considerations
Over the counter					
Pyrethrin with piperonyl butoxide shampoo (A−200, Pronto, R&C, Rid, or Triple X)	Natural extract from the chrysanthemum flower. Blocks sodium transport, leading to depolarization of neuromembranes and respiratory paralysis	Apply to dry hair. Let sit for 10 min. Repeat in 7-10 days. Nit combing recommended (not ovicidal)	Contact dermatitis	Level 1B RCT: 50% ovicidal at day 14[175]	Approved by the FDA for children >2 years of age; efficacy has waned because of resistance
Permethrin 1% lotion or cream rinse (Nix)	Synthetic pyrethrin. Blocks sodium transport, leading to depolarization of neuromembranes and respiratory paralysis	Apply after shampoo. Let sit for 10 min. Rinse. Repeat in 7 days. Nit combing recommended (not ovicidal)	Contact dermatitis	Cream rinse: level 1A RCT: 97% efficacy after 14 days[176] Metaanalysis: >90% cure rates[177]	Approved by the FDA in 1986 for children >2 months of age; efficacy has waned because of resistance
Dimethicone 4% lotion (LiceMD)	Long-chain linear silicone dissolved in a volatile silicone base. Physical material (not an insecticide) that coats hair shafts	Apply to dry hair. Let sit for two 8-hour treatments, 7 days apart[178] (reported to be ovicidal at higher concentration)[179]	Skin/eye irritation[178]	Level 1B RCT: Eradicated lice in 70% of patients[178]	Not approved by the FDA
Prescription					
Malathion 0.5% lotion (Ovide) or gel	Organophosphate cholinesterase inhibitor. Causes louse respiratory paralysis	Lotion: apply to dry hair. Let sit for 8-12 hours. Shampoo: repeat in 7-9 days if lice still present (partially ovicidal) Gel: Apply to dry hair. Let sit for 30 min. Repeat in 7-9 days if still present	Flammable; do not use a hair dryer/iron[59]; skin irritation[180]	Lotion: Level 1B RCT: 100% effective at 24 hours. 95.3% effective at 7 days[180] Gel: Level 1B RCT: 98% response after 30 min[181]	Withdrawn in 1995 because of flammability, then reinstated in 1999; approved for children >6 years of age[96] Useful for permethrin-resistant lice[93]
Benzyl alcohol 5% lotion (Ulesfia)	Aromatic alcohol that kills lice via asphyxiation; not neurotoxic	Apply to dry hair. Let sit for 10 min. Rinse. Two applications needed (not ovicidal)	Skin irritation[182]	Level 1B Two RCTs: 75-76.2% elimination[182]	Approved by the FDA 2009 for children >6 months of age

Spinosad 0.9% suspension (Natroba)	Paralyzes lice by agonizing acetylcholine and antagonizing gamma-aminobutyric acid receptors	Apply to dry hair. Let sit for 10 min. Rinse. No nit combing required.[97] Repeat in 7 days if lice are visualized (ovicidal)[97,98]	Skin/eye irritation[97]	Level 1B RCT: 84.6-86.7% effective (after 1 or 2 applications)[97]	Approved by the FDA 2011 for children >4 years of age; expensive[183]
Ivermectin 0.5% lotion (Sklice)	Binds glutamate-gated chloride channels in invertebrate nerve and muscle cells, leading to cellular hyperpolarization[99]	Apply to dry hair. Let sit for 10 min. Rinse. Two applications recommended (not ovicidal)[99,184]	Skin irritation[184]	Level 1B Two RCTs: 94.9% lice-free at day 2; 73.8% lice-free at day 15[184]	Approved by the FDA in 2012 for children >6 months of age
Abametapir 0.74% lotion	Metalloproteinase inhibitor	Apply to dry hair. Let sit for 10 min. Rinse. No nit combing required (ovicidal)	Skin irritation[185]	Level 1B Two RCTs: 81.1-88.2% lice-free 14 days after 1 application[185]	Not approved by the FDA
Oral ivermectin (Stromectol)	Same as topical ivermectin above	200 μg/kg dosed once, or 400 μg/kg dosed twice at 7-day interval (not ovicidal)[101]	Pruritus, headache	Level 1B RCT: 95.2% lice free by day 15[101]	Not approved by the FDA for the treatment of head lice; not for pregnant women or children weighing <15 kg[59]
Hot air delivered to the scalp	Causes louse death via overheating	30 min of heat applied to scalp (multiple delivery modalities studied)	None reported	Level 2B >88% effective in killing eggs at 14 days; variable efficacy in killing hatched lice[109]	Not approved by the FDA

Drug names are trademarks of their respective owners.
FDA, US Food and Drug Administration; *RCT*, randomized controlled trial.
*Level 1A, evidence from metaanalysis of randomized controlled trials; level 1B, evidence from ≥1 randomized controlled trial; level 2A, evidence from ≥1 controlled study without randomization; level 2B, evidence from ≥1 other type of experimental study; level 3, evidence from nonexperimental descriptive studies, such as comparative studies, correlation studies, and case-control studies; level 4, evidence from expert committee reports or opinions or clinical experience of respected authorities, or both.

response and potential promotion of permethrin resistance.[58]

HUMAN HEAD LOUSE (*PEDICULUS HUMANUS CAPITIS*)

Key points

- Head lice are a public health problem affecting all demographics
- Head lice can be infected with pathogens
- Novel US Food and Drug Administration (FDA)–approved topical therapies include benzyl alcohol 5% lotion, spinosad 0.9% suspension, and ivermectin 0.5% lotion

Head lice are ovoid-shaped, 2- to 3-mm arthropods (Fig 3, *A*).[59,60] Head lice are obligate human parasites that spend their entire life cycle on the scalp, feeding off blood every few hours.[61] Female lice live ≤30 days and lay approximately 10 eggs daily.[61] Nits are transparent, flask-shaped, 0.5-mm eggs found on hair shafts, typically 1 to 4 mm above the scalp, where warmth promotes survival (Fig 3, *B*).[60,61]

Epidemiology

Head lice infest >100 million people worldwide and 6 to 12 million people in the United States annually.[6,62] Transmission occurs via direct head-to-head contact, but lice can survive ≤4 days on fomites, including hairbrushes or headgear.[61] Most cases occur in children, particularly females, likely because of cultural hair length differences.[6,63] Head lice affect people of all socioeconomic statuses.

Clinical manifestations

Pruritus, papular urticaria, excoriations, and cervical/occipital lymphadenopathy can occur.[60,64] Diagnosis is achieved via direct observation of lice or nits on hair shafts. Dermoscopy distinguishes eggs containing nymphs (Fig 3, *C*) from empty, translucent "pseudonymphs" (hair casts, hair product debris, or seborrheic dermatitis).[65-68] Pruritus and disturbed sleep may cause school and work absences.[69] Severe iron deficiency anemia[17,70,71] has been associated with heavy/chronic head lice infestation.

Implications

Head lice can carry and transmit *Staphylococcus aureus* and *Streptococcus pyogenes*.[61] Recent evidence suggest head lice may also harbor other pathogens. *B recurrentis* DNA was identified in 23% of head lice from patients with LBRF in Ethiopia[72] and the Republic of the Congo.[73] *B quintana* was detected in head lice from France[74] (although it was not detected in another French study[75]), Senegal (3-5% of lice in 2 studies),[76,77] Ethiopia (9.2%),[78] San Francisco (25%),[79] Nepal,[80] and various African countries (2%).[81] Investigators in Mali identified *Coxiella burnetti* and *Rickettsia aeschlimannii* in 5.1% and 0.6% of head lice, respectively.[82] Pathogenic Acinetobacter species were identified in head lice from Thailand,[83] Algeria,[84] and 2 locations in France.[75,85] Whether the presence of these organisms yields an increased risk of clinical disease remains unclear.

Management

Management of head lice requires eradication of all living lice and eggs. Mechanical removal is possible but labor intensive and is more efficacious when combined with medications.[59]

Topical therapies are summarized in Table III.[60] Over-the-counter treatments include pyrethrins (neurotoxins derived from chrysanthemums), permethrin 1% lotion/cream (synthetic pyrethrin), and dimethicone (often spelled "dimeticone" outside the United States).[59] Topical pyrethrin[86-89] and 1% permethrin resistance is widespread.[90-94] Prescription options include malathion 0.5% lotion/gel, benzyl alcohol 5% lotion, spinosad 0.9% suspension, and ivermectin 0.5% lotion. Lindane is no longer recommended because of potential neurotoxicity.[95] Malathion was temporarily withdrawn from the US market because of flammability.[96] Ovicidal agents, including malathion and spinosad, are advantageous because they may not require repeat treatments.[97-99] Persistent infestations (live lice observed 24 hours after treatment) should be retreated with a new medication class because resistance is likely.[69,100] Oral ivermectin is effective for refractory head lice but is not approved by the FDA.[101]

Essential oils have demonstrated efficacy in eradicating head lice in many studies (Table IV).[102-108] Most studies were in vitro, although combination Melaleuca and lavender oil, and also combination eucalyptus oil and *Leptospermum petersonii* solution, were effective in in vivo RCTs.[106,108] No essential oils have been approved by the FDA for treating head lice, and many carry a risk of allergic contact dermatitis.[69]

Conditioner should not be applied before topical medications and hair should not be rewashed for 1 to 2 days after removing the medication.[96] For patients deferring medications, delivering hot air to the scalp for 30 minutes showed up to 88% efficacy in killing lice.[109] Nit combs can be used every 2 to 3 days

Table IV. Efficacy of essential oils in treating head lice

Year	Compound	Study type	Instructions	Results
2007	Combined eucalyptus and peppermint in total 10% concentration, dissolved in 50% ethanol plus isopropanol in water	In vitro	N/A	Showed the greatest (93%) knockdown (lice death) 10 min after application[186]
2010	*Cinnamomum porphyrium* oil	In vitro	N/A	Killed >50% of lice within 2 min[105]
2010	Melaleuca oil and lavender oil in combination	In vivo	Applied 3 times at 7-day intervals	97.6% efficacy (louse-free) and superiority to pyrethrins at 24 hours[106]
2011	Citronellol and geraniol (both active components of 2 essential oils)	In vitro	N/A	>60% mortality of adult and late-stage nymphs[187]
2012	Tea tree (Melaleuca) oil, 1% concentration	In vitro	N/A	100% lice mortality at 30 min, showing both pediculicidal and ovicidal effects[102]
2016	Fumigant bioassays of leaves/fruits of *Schinus areira* (Anacardiaceae) and *Thymus vulgaris* (Lamiaceae)	In vitro	N/A	*S areira* derivatives were most toxic against adult lice (10 min to achieve 50% knockdown); *T vulgaris* was most ovicidal (0% hatching after 24 hours)[188]
2018	10% *Curcuma xanthorrhiza* and 10% *Eucalyptus globulus* oils	In vitro	N/A	5-min immersion halted egg hatching[103]
2018	Clove oil diluted in either coconut or sunflower oil	In vitro	N/A	90% lice mortality within 2 hours of a 30-min contact[104]
2018	Eucalyptus oil and *Leptospermum petersonii* solution	In vivo	Applied 3 times at 7-day intervals	More than twice as effective in curing lice than pyrethrin-containing mousse applied twice[108]
2018	Eucalyptus oil and *Leptospermum petersonii* solution	In vitro	N/A	Exposure in vitro killed 100% of lice and eggs[108]

after treatment to decrease the chance of reinfestation.[96] Along with scalp treatment, decontaminating clothing/linens at temperatures >50°C is recommended.[110] Children should not be sent home from school early to treat infestation and should be allowed to return immediately after beginning treatment.[59] The American Academy of Pediatrics recommends against strict no-nit policies in schools.[59] All household members should be screened.

HUMAN PUBIC LOUSE (*PHTHIRUS PUBIS*)

Key points

- Pubic lice infestation can involve multiple body regions
- A diagnosis warrants screening for other sexually transmitted infections and, in children, considering the possibility of abuse

Pubic lice measure 0.8 to 1.2 mm in diameter and are wider than they are long, unlike ovoid-shaped head and body lice.[60] This crab-like shape enables them to grasp widely spaced pubic hairs (Fig 4, *A*). Female lice lay roughly 30 eggs during their 3- to 4-week lifespan.[111]

Epidemiology

Pubic lice are transmitted via close physical contact, including sexual contact and shared sleeping arrangements.[112] Infestation occurs worldwide and affects both genders. Pubic hair removal via shaving, waxing, or laser has been associated with a decreased incidence.[113]

Clinical manifestations

P pubis infestation occurs on hairs of the scalp, axilla, chest, thighs, pubic area, eyebrows, and

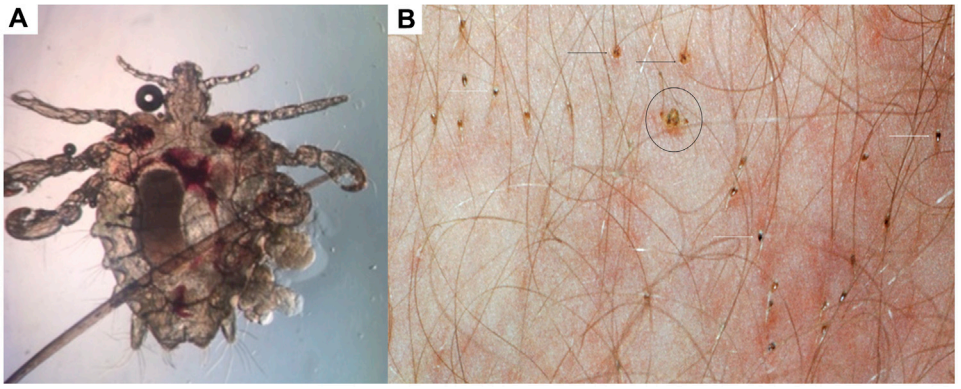

Fig 4. Pubic lice. **A**, Pubic louse morphology (courtesy of Charlotte Bernigaud, MD, Olivier Chosidow, MD, PhD, and Francoise Foulet, MD). **B**, Pubic louse (black circle) and numerous nits (several highlighted by white arrows) attached to hair shafts. Hemorrhagic macules (several highlighted by black arrows) at site of pubic lice bites (courtesy of Tim Berger, MD).

eyelashes. Symptomatic patients present with localized pruritus. Maculae ceruleae (blue-gray macules), red papules, and rust-colored feces can be seen at bite sites (Fig 4, *B*).[60,114,115] In children, eyelashes are commonly involved[112,116-118] and coinfestation with head lice has been reported.[118] Diagnosis is achieved by visualizing lice or nits on hair shafts (Fig 4, *B*). Bacterial superinfection of excoriations may occur. Pubic lice are not known to transmit other infections.

Management

A diagnosis of pubic lice warrants screening for other sexually transmitted infections.[111] While pediatric pubic lice infestation can occur through shared sleeping arrangements with infested individuals or contact with fomites,[117] abuse should be considered.[112,118]

The first-line treatment of pubic lice is topical 1% permethrin.[111,114] Topical 0.5% ivermectin lotion is also approved by the FDA.[111] Petrolatum jelly is useful for treating eyelash infestation.[119] Oral ivermectin is a second-line therapy but has not been approved by the FDA.[114,120] Clean clothing should be worn when starting treatment.[111] Shaving is therapeutic and decreases the likelihood of recurrence. All clothing/linens used in the preceding 3 days should be washed and dried at temperatures >50°C.[121]

TUNGIASIS

Key points

- Tungiasis is an infestation, endemic to tropical world regions, caused by a flea burrowing into the skin
- Tungiasis is seen in travelers returning from endemic areas
- Tungiasis is associated with not wearing closed-toe footwear and dirt floors

Tungiasis is an ectoparasitic disease caused by skin penetration by the female *Tunga penetrans* or, less commonly, *Tunga trimamillata* flea.[122] It is endemic in Latin America, the Caribbean, and sub-Saharan Africa[123] and occurs in travelers returning from endemic regions.[124-133]

Epidemiology

In travelers to endemic areas, tungiasis is usually acquired from walking barefoot or with open-toed shoes. In returning travelers between 2007 and 2011, 87 cases were reported in GeoSentinel, the largest global surveillance network for travel-related morbidity.[133] The top exposure countries were Brazil, Madagascar, Uganda, and Ethiopia. In returning travelers from Brazil who visited a GeoSentinel clinic between 1997 and 2013, 35 cases (2% of 1586) were diagnosed with tungiasis.[132] At a German travel clinic, most returning travelers with tungiasis had been exposed in Africa or Latin America.[134]

In endemic areas, the prevalence of tungiasis ranges from 19.1% to 58.7% (Table V).[135-141] In these settings, tungiasis is linked to inadequate health education,[135,140] the lack of regular closed-toe footwear use,[137,141,142] and poor housing conditions,[135,140] including dirt floors or unclean floors,[137,138,142] crowded living spaces,[140] and sleeping on the ground outside.[142] Causative fleas may also exist in domestic or wild animal reservoirs, and therefore tungiasis is considered a zoonosis.[143-145] Living on compounds with animals

Table V. Tungiasis prevalence and risk factors in endemic settings

Year	Location	Population	Prevalence of tungiasis	Risk factors for infestation
2006	Rural Northeast Brazil[135]	496 individuals in 132 households	51.0%	Poor housing conditions (OR = 4.7), lack of health education (OR = 4.1), and the presence of animals on the compound (OR = 1.9)
2006	Haiti (4 regions)[189]	383 patients	31.1%	Not studied
2007	Rural Western Nigeria[142]	643 individuals	42.5%	Presence of pigs (aOR = 17.98), sand or clay floor inside houses (aOR = 9.33), common resting place outside (aOR = 7.14), and no regular use of closed footwear (51% pop AR)
2012	Rural Western Tanzania[136]	586 individuals	42.5%	Age ≥45 years (OR = 3.71)
2015	Murang'a County, Kenya[137]	508 children	19.1%	Classrooms with dusty floors (aOR = 14.657), earthen mud walled houses (aOR = 13.78), and not regularly using closed footwear (aOR = 10.45)
2015	Maguye District, Eastern Uganda[138]	422 households in 12 villages	22.5%	Cracked house floor (aOR = 6.28), dirty feet (aOR = 3.86), dirty clothes (aOR = 3.46), dirty floor (aOR = 3.21), littered compounds (aOR = 2.95), and rearing cattle (aOR = 2.38)
2017	Southwest Nigeria[139]	545 children	24.4%	Not studied
2017	Kilifi County, Kenya[140]	1086 individuals from 233 households in 8 villages	25.0%	Only mud puddles as a washing source (OR = 25.48), washing the body without soap (OR = 7.36), mud walls (OR = 3.35), lack of water, permitting washing only once daily (OR = 2.23), number of people sleeping per room (OR = 1.77), and sleeping on the floor (OR = 1.68)
2017	Yirgacheffe, Southern Ethiopia[156]	343 children	34.7%	Closed footwear associated with increased lesion number
2018	Wensho District, Southern Ethiopia[141]	366 children	58.7%	Never using footwear (aOR = 12.55), occasionally using footwear (aOR = 7.42), cat-owning household (aOR = 4.95), illiterate mothers (aOR = 3.62), and mothers who have attended only primary school education (aOR = 2.22)

aOR, Adjusted odds ratio; *OR*, odds ratio; *pAR*, population attributable risk.

confers a higher risk.[135,138,141] Leading animal reservoirs differ by region, including stray cats/dogs in urban Brazil,[146] domestic cats in southern Ethiopia,[141] and domesticated pigs in Uganda and Nigeria.[142,147]

Clinical manifestations

Lesions predominantly affect the feet. Skin findings reflect the lesion's clinical stage, which is linked to the life cycle of the embedded flea and correlates with disease severity according to the Fortaleza classification (Table VI).[148] After painlessly burrowing into the skin, typically on the feet, embedded fleas mature for several weeks.[123] An early-stage lesion is a 1-mm red-brown macule that evolves into a nodule with a central dark punctum (Fig 5, *A*). Subsequent flea engorgement from egg production leads to swelling, erythema, pruritus, and pain.[123] Eventually, egg release and parasite death trigger severe inflammation, leading to a black crusted papule that heals with a punched-out scar (Fig 5, *B*). Dermoscopy can be useful in diagnosing specific features, which in 1 series included dark central pores, whitish oval structures, silver dendritic fibers, and blue-black

Table VI. Fortaleza classification of the clinical stages of tungiasis

Stage	Duration	Parasite findings	Clinical findings	Histopathology findings
I	3-7 hours	Penetration at angle of 45°-90°; abdominal segments 2 and 3 begin separating	Itchy or painful red-brown 1-mm macule	Epidermis: hyperplasia, hyperkeratosis, parakeratosis Dermis: mild inflammatory infiltrate of neutrophils and eosinophils
II	1-2 days	Hypertrophic zone between abdominal segments 2 and 3	Pearly white nodule with surrounding erythema and a central dark punctum	Epidermis: hyperplasia, hyperkeratosis, parakeratosis, spongiosis, neutrophilic infiltrate, and intracorneal microabscesses Dermis: perivascular inflammatory infiltrate of neutrophils, lymphocytes, eosinophil, and plasma cells and mast cells
IIIa	2-3 days	Head of parasite at dermoepidermal interface	Growing, yellow-white halo around black dot. Fecal coils visible. Pain, foreign body sensation, severe itching	Epidermis: marked hyperplasia and hyperkeratosis, reactive pseudoepitheliomatosis; marked mixed inflammatory infiltrate Dermis: mixed inflammatory infiltrate
IIIb	1-2 weeks	Thickening of chitin exoskeleton	Caldera formation; soft consistency; severe pain	Epidermis: same as above Dermis: dilated vessels and neutrophils
IV	3-5 weeks	Flea dying or dead	Crusted black lesion (containing dead parasite) with or without superinfection	Epidermis: same as above, plus vascularization of stratum corneum Dermis: moderate to severe mixed infiltrate
V	6 weeks to several months	No parasite	Residual scar, punched out depression; nail dystrophy or loss; lymphedema	Epidermis: hyperplasia, thickening and blunting of epidermal ridges, and thickened stratum spinosum Dermis: residual mild inflammation

Adapted from Eisele et al.[148]

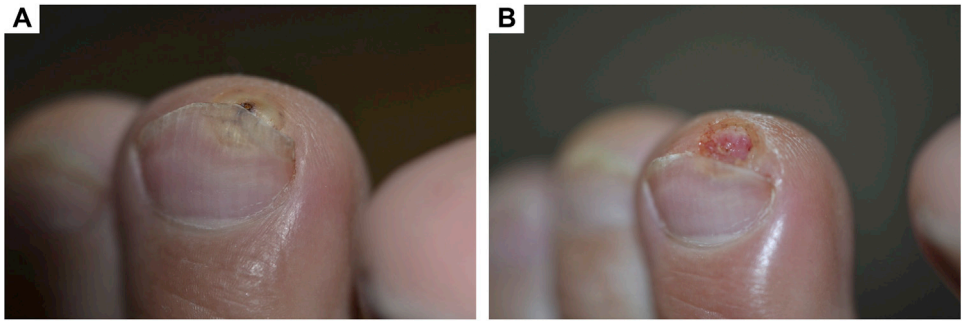

Fig 5. Tungiasis. **A**, Early clinical stage of tungiasis with embedded flea (courtesy of Jorg Heukelbach, MD, PhD). **B**, Immediate aftermath of tungiasis infestation after death and expulsion of an embedded flea (courtesy of Jorg Heukelbach, MD, PhD).

blotches in most cases.[149] The penetration site measures ≤500 μm in diameter,[150] and therefore secondary bacterial infection occurs frequently, including cellulitis and necrotizing skin and soft tissue infection.[150,151] In returning travelers, the differential diagnosis depends on the clinical stage and includes arthropod bite, wart, pyogenic granuloma, abscess, leishmaniasis, myxoid cyst, myiasis, and foreign body.[152]

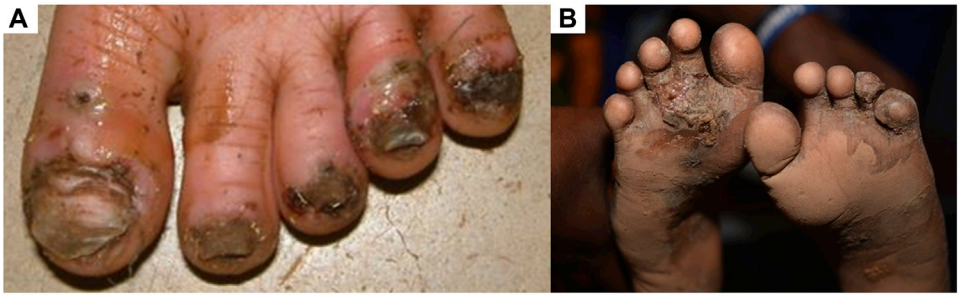

Fig 6. Tungiasis. **A**, Multiple embedded fleas on the dorsal surfaces of the toes in a person living in an endemic area (courtesy of the Regional Dermatology Training Centre, Moshi, Tanzania). **B**, Multiple embedded fleas on the plantar surface of the foot in a person living in an endemic area (courtesy of Jean Marie Rukanikigitero, MD).

Quality of life

Tungiasis-associated morbidity in endemic settings is caused by severe foot pain, progressive foot mutilation, and nail dystrophy, complicating walking and contributing to stigmatization.[153,154] Children may experience teasing and have higher school absenteeism and lower school performance than unaffected peers.[155] Tungiasis negatively impacts children's quality of life,[156,157] with disturbed sleep and concentration reported most frequently in 1 Kenyan study.[157]

Treatment

Tungiasis is self-limited because the organism typically dies within 6 six weeks after penetration.[148] Treatment aims to reduce symptom severity and prevent secondary infection. Surgical removal of the organism is crucial. This can be achieved through shave or punch biopsy procedures. Early on, sterile needles can also be used before multiple embedded fleas or extensive inflammation occur.[158] Secondary bacterial infection requires appropriate antibiotics.

In endemic settings without access to appropriate equipment for surgical removal, commonplace sharp instruments, such as pins, needles, thorns, and sharpened wood pieces, are frequently reused by different people.[155] This can lead to bloodborne pathogen transmission,[155] tetanus,[159,160] and life-threatening complications of secondary infection, including necrotizing fasciitis, gangrene, and sepsis.[160-162] As such, nonsurgical alternatives have been investigated. Topical Zanzarin, derived from coconut oil, jojoba oil, and aloe vera, decreases tungiasis incidence and morbidity[163,164] but is no longer commercially available. Topical dimethicone has been shown to reduce inflammation and hasten parasite death in 1 RCT,[165] with targeted application to areas of parasite protrusion being more effective than to the entire foot.[166] Oral ivermectin showed no efficacy in a RCT.[167]

Screening and prevention

For prospective travelers to endemic areas, the best preventive measure is wearing closed-toe shoes.[168] Only 54% of returning travelers diagnosed with tungiasis reported a pretravel medical visit,[133] and even when a visit occurs tungiasis may not be discussed.[133] Clinicians should therefore counsel patients with relevant planned travel.

In endemic areas, where nearly 95% of lesions are restricted to the feet,[169] targeted evaluation of periungual feet is useful for estimating prevalence, severity, and identifying persons needing treatment.[170] While improving education and health care access are important, clinical knowledge does not necessarily translate into effective prevention and treatment.[171] Large-scale provision of closed-toe footwear is essential. In a metaanalysis, footwear use was associated with significantly lower odds of acquiring tungiasis.[172] However, regular shoe replacement is costly[143] and may not reduce exposure inside houses, where shoes are not typically worn.[143] As such, modifications are important, including switching to sealed cement floors and cleaning floors daily.[162] Unfortunately, human hosts living in endemic areas are frequently reinfested unless proper footwear and housing changes are widely implemented.[162] Finally, increasing tetanus vaccination coverage in tungiasis-endemic regions will help prevent secondary tetanus.[173]

In conclusion, in the context of a global refugee crisis and homelessness epidemic, diagnosing and managing lice infestations are important dermatologic skills. Dermatologists should know that careful clothing inspection is key to diagnosing body lice and that head lice have widespread

resistance to topical pyrethrin and permethrin. Tungiasis is an infestation affecting the feet that has substantial negative impact on communities in endemic regions. It is seen in returning travelers and can be prevented by wearing closed-toe shoes.

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Answers to CME examination

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