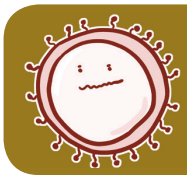

NOTES
ECTOPARASITES

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- *Ectoparasites:* arthropods that live on outside of host, extract nutrients at host's expense
  - *Uncommon:* viable parasites transferred without direct host contact

RISK FACTORS
- Commonly poor hygiene, close living quarters

COMPLICATIONS
- Predicated on individual's immune status, housing situation

SIGNS & SYMPTOMS

- Pruritus, bite marks, visible body parasites

DIAGNOSIS

OTHER DIAGNOSTICS

History
- Including close contacts/living quarters

Physical examination
- Dermatologic examination

TREATMENT

MEDICATIONS
- Topical neurotoxins, topical/oral parasidal drugs

OTHER INTERVENTIONS
- Proper hygiene, household measures, isolation (if necessary)

PEDICULOSIS CORPORIS, CAPITIS, AND PUBIS (LICE)

osms.it/lice

PATHOLOGY & CAUSES

- Infestation of easily transmissible sucking lice species
  - Commonly in hairy bodily areas, characterized by local pruritus
- Sucking lice infection
  - (Phylum) arthropoda → (class) insecta → (order) phthiraptera → (suborder) anoplura → (family) pediculidae/pthiridae

PATHOLOGY
- Lice live human hair → suck blood for nutrients
  - Bite → saliva injection → anticoagulation effect, ↑ histamine release → maculae cerulea (blue/copper-hued bite marks); pruritus

- Lice require humans for nutritional source (parasites)
  - Most climates allow mature louse 24 hours of viable life away from human source

Transmission

- Physical contact
  - *Ideal location:* slow-moving, parallel hair fibers
  - Louse on one hair fiber → transfer to another individual's hair → lay nits (eggs) on hair 1–2mm off of scalp → nymphs hatch within one week → mature over one week → female lice lay eggs for one month
- Fomites

TYPES

- *Pediculus humanus capitis* → head louse
  - Can survive 24–48 hours without blood meal/separated from host
- *Pediculus humanus humanus* → body louse
  - Larger than head louse; can survive < 72 hours without blood meal
- *Phthirus pubis* → pubic louse
  - AKA crab louse; can spread to body

RISK FACTORS

- School-aged children
- Homeless population
- Refugee population (if living in close quarters)

COMPLICATIONS

- Co-infections (also carried by louse)
  - *Bartonella quintana* → endocarditis
  - Epidemic typhus
  - Louse-borne relapsing fever
  - Trench fever
- Adolescents with pubic lice → ↑ gonorrhea/chlamydial infection risk
- Pruritus → skin excoriation → secondary infection
  - Commonly staphylococcal infection

SIGNS & SYMPTOMS

- Site pruritis (head, body, pubis)

DIAGNOSIS

OTHER DIAGNOSTICS

Dermatologic examination

- Examination of hair follicles, clothing seams
- *Nits:* more visible than nymphs/lice; most visible (white) after louse released from egg; does not dislodge easily from hair follicle
- Body louse → widespread dermatitis
- Often linear excoriations
- Maculae ceruleae → may have punctal hemorrhages → recent bites
- Hyperpigmentation/lichenification → older bites

Lymph node examination

- Capitis infection → posterior lymphadenopathy

TREATMENT

MEDICATIONS

Topical benzyl alcohol

- Mechanism of action → louse asphyxiation
  - Difficult for resistance to develop

Neurotoxic agents

- Resistance develops with
  - *Pyrethrin:* botanically-derived neurotoxin
  - *Permethrin:* Na^+ channel blocker → paralysis → death
  - *Malathion:* organophosphate cholinesterase inhibitor

OTHER INTERVENTIONS

Hair shaving

- Eradicate current infection

Mechanical removal

- Wet combing → tedious, poor compliance
  - *Pre-treatment:* vinegar/formic acid → flattened hair cuticle → better combing efficiency (does not dissolve/loosen nits)

Prevention

- Proper hygiene

- Household
  - *Housemates:* examination
  - *Bedmates:* prophylactic treatment
  - *Household cleaning:* washing > 54°C/130°F; unwashable material → place in sealed plastic bag for two weeks
- School
  - No nit policy (infected children stay home), education, screening during outbreaks

SARCOPTES SCABIEI (SCABIES)

osms.it/sarcoptes-scabiei

PATHOLOGY & CAUSES

- *Sarcoptes scabiei* mite infection
  - Elicits strong immune response
  - Nocturnal pruritus

Mite infection

- (Phylum) mite → (class) arachnida → (subclass) acari → (order) astigmata → (family) sarcoptidae
  - Usually obligate human parasite → *vars hominis*
  - Sometimes animal mange mites can infest

PATHOLOGY

Mite transfer

- Direct skin-to-skin contact for 15–20 minutes
- Average infested individual carries 5–12 mites
  - *Crusted scabies individuals:* > 1000 mites can be shed (transmission through objects more likely)

Type IV hypersensitivity reaction

- House dust mite cross reactivity
- Infestation → ↑ IL-6, vascular epithelial growth factor (VEGF) →TH1-cell activation → IL-2 release → lymphocyte proliferation, differentiation

RISK FACTORS

- Overcrowding (including long-term care facilities, prisons), poor hygiene/nutrition, homelessness, dementia, sexual contact

COMPLICATIONS

- Infestation → secondary staphylococcal infection
  - Low-income countries (mostly)
  - Impetigo → chronic kidney disease
  - Ecthyma, paronychia, furunculosis

Crusted scabies

- AKA Norwegian scabies
- Infection commonly scalp, hands, feet → diffuse spread over entire body
- Occurs in compromised cellular immunity setting
  - Acquired immunodeficiency syndrome (AIDS)
  - Human lymphocytic virus type 1 (HTLV-1)
  - Leprosy
  - Lymphoma
  - Long-term topical corticosteroid use
- *Risk factors:* age, Down syndrome
- *Complications:* fissional lesions develop → bacterial entryway → infection
  - Sepsis, poststreptococcal glomerulonephritis

SIGNS & SYMPTOMS

Classic scabies

- Intense, intractable, generalized pruritus
- Nodules, pustules at most intense pruritus sites
- Common areas → intertriginous spaces
  - Anterior axillary folds, webs of fingers, volar aspect of hand/wrist, beltline, penis, areolar region (biologically-female individuals)

Nodular scabies

- Hypersensitivity reactions → large, persistent, intensely pruritic 5–6mm nodules
  - Commonly groin, buttock, axillary folds

Crusted scabies

- Poorly defined, erythematous patches → scale
  - Untreated → entire integumental spread → warty appearance (especially over bony prominences); lesions crust, fissure develop → malodorous; nail involvement → thickened, dystrophic, discolored

DIAGNOSIS

LAB RESULTS

Microscopy

- *Confirmatory scraping:* fluorescein stain → highlights fecal material, ova fragments
  - Epithelial milieu (eosinophils, lymphocytes, histiocytes)
  - *Crusted scabies:* mate capture more likely due to disease burden

Polymerase chain reaction assays

- *S. scabiei* DNA polymerase

OTHER DIAGNOSTICS

History

- Close contact commonly present with concurrent symptoms
- Infected individual contact history (may be many weeks prior)

Dermatologic examination

- Serpiginous keratotic lines (1–4mm) → burrow marks
  - Often with vesicle on end (housing mite)

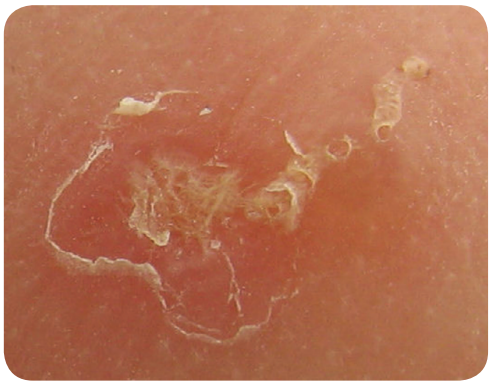

Figure 69.1 A high-magnification photograph of a single mite burrow in the skin of an individual with scabies. The mite is at the end of the burrow at the top right of the image.

TREATMENT

MEDICATIONS

Classic scabies

- Permethrin (5%) → synthetic neurotoxin → Na+ channel blocker → paralysis → death
- Precipitated sulfur (6%, 10%) in petroleum
- Benzyl benzoate (10%, 25%)
  - *Adverse reactions:* allergic dermatitis
  - *Contraindications:* pregnancy/lactation (neurotoxicity); children < two years old
- Oral ivermectin
  - One dose (200mcg/kg) repeated in 7–10 days

Nodular scabies

- Topical steroids
- Intralesional steroid injection

Crusted scabies

- Topical, systemic treatment required
- Oral ivermectin, topical permethrin (5%)/ benzyl benzoate (5%)
  - Two week oral regimen, topical therapy

persisting after that twice weekly until cure
    - Treatment cure → active lesion resolution, nocturnal pruritus absence for one week

OTHER INTERVENTIONS

- Isolation for infected
- Nail clipping
    - +/- brushing with scabicidal agent
- Thorough personal, household material laundering

Prevention

- Monosulfiram soap in communities with ↑ ↑ incidence