Common Child and Adolescent Cutaneous Infestations and Fungal Infections

Sherman J. Alter, MD,[a,b] Megan B. McDonald, MD,[c] Julie Schloemer, MD,[d] Ryan Simon, MD,[a,b] and Julian Trevino, MD[d]

Cutaneous infections and infestations are common among children and adolescents. Ectoparasitic infestations affect individuals across the globe. Head lice, body lice, scabies, and infestations with bed bugs are seen in individuals who reside in both resource poor areas and in developed countries. Superficial cutaneous and mucosal candida infections occur throughout the life cycle. Dermatophyte infections of keratin-containing skin and skin structures result in tinea capitis (scalp), tinea corporis (body), tinea pedis (foot), and tinea unguium (nails). Less frequent endemic fungal infections such as blastomycosis, coccidiodomycosis, and histoplasmosis may present with skin findings. This article will describe the epidemiology and transmission of these conditions as well as their clinical manifestations. The approach to diagnosis will be addressed as well as primary prevention and current therapies.

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Introduction

Human cutaneous infestations and infections are widespread throughout the world. Ectoparasitic organisms live on the surface of the host. Lice, scabies, and bed bugs infest children and adolescents in the developed world and are highly prevalent in resource poor populations. These infestations may be associated with considerable morbidity. Fungal infections of the skin and mucous membranes occur in all age groups. Candida yeasts normally reside in the intestinal tract and can be found on mucous membranes and skin without causing infection. Candidiasis results from overgrowth of the organism. Symptoms of candidiasis vary depending on the area of the body that is infected. Certain fungi invade and proliferate in keratin-containing layers of the integument. These fungal pathogens predominantly infect the hair, skin, and nails (dermatophytes) and cause the most common mycotic cutaneous diseases (dermatophytoses) worldwide. While far less frequent, one might encounter patients who present with cutaneous manifestations of endemic fungal infections (coccidiodomycosis, blastomycosis, or histoplamosis). This article describes the epidemiology of these conditions, their clinical presentation and approach to diagnosis. Current treatment recommendations and primary prevention will also be addressed.

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Infestations

Bites and infestations represent common complaints among the pediatric population presenting to both primary care clinicians and dermatologists. While these infestations are not life-threatening, they can cause significant physical and psychologic distress. Once a child is diagnosed as having head lice, scabies or bed bugs, he or she may be stigmatized, leading to social isolation. Primary care clinicians play a pivotal role in both the diagnosis and treatment of these problems. This article reviews the epidemiology, clinical presentation, methods of diagnosis and treatment recommendations of

From the [a]Division of Infectious Diseases, Dayton Children's Hospital, One Children's Plaza, Dayton, OH; [b]Department of Pediatrics, Wright State University, Boonshoft School of Medicine, Dayton, OH; [c]United States Air Force, OIC Pediatrics Clinic, 28 MDOS/SGOK, Ellsworth Air Force Base, SD; and [d]Department of Dermatology, Wright State University, Boonshoft School of Medicine, Dayton, OH.

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FIG 1. Bedbugs.

these three common conditions: bed bug infestation, scabies infestation, and head lice.

Bed Bugs

Bed bugs are wingless ectoparasites belonging to the family Cimicidae and include the subtypes *Cimex lectularius* (common bed bug found in temperate climates) and *Cimex hemipterus* (found in warmer climates).[1] The common bed bug is reddish-brown in color, oval and flat in shape with 3 pairs of legs, and typically ranges between 3 and 6 mm in length (Fig 1).[1,2] Its average life span is approximately 6–12 months and consists of five nymphal stages prior to reaching adulthood. Nymphs are much smaller in size and lighter in color than adults but are still visible to the naked eye. Bed bugs are nocturnal and typically hide in inconspicuous locations such as underneath mattresses, inside cracks and crevices or behind wall coverings during the day. These insects are hematophagous and require blood meals from either mammals or birds, depending on the species. They can, however, survive months to even years without feeding.[1] Although controversial, bed bugs are generally not thought to act as vectors for transmission of infectious diseases including HIV and hepatitis B.[1,3] However, there is growing concern these parasites may transmit antibiotic-resistant bacteria including MRSA and further studies on this subject are needed.[4]

Over the past few decades, bed bug infestations have been on the rise in the United States as well as in other countries.[5] For example, the number of reports of bed bug infestations in San Francisco more than doubled between 2004 and 2006.[6] Another study found that 1 in 5 Americans reported a bed bug infestation in their home or they knew someone who encountered bed bugs at home or in a hotel.[7] This resurgence is attributed to many factors, including increase in worldwide travel and resistance to commonly used pesticides.[8]

Victims of bed bug bites typically present with multiple pruritic, erythematous papules on exposed areas such as the head, neck, arms and legs. Papules tend to be grouped or in linear array giving rise to the characteristic "breakfast, lunch, and dinner" sign and may have a central hemorrhagic punctum[9] (Fig 2). The characteristic eruption is thought to result from the host's immune response to salivary proteins released during feeding.[10] Bed bug infestation should still be considered even when some household members do not report bites as it is possible for only one or a few household members to be afflicted.[11] Some individuals may develop bullous or urticarial reactions at bite sites and rarely may even progress to anaphylaxis.[12,13] Secondary infection may result from scratching bite sites.

Diagnosis of bed bug bites is primarily made by suggestive history and physical exam findings. Occasionally, bed bugs may be observed on the patient or patient's clothing during examination. Skin scrapings prepared in mineral oil may be examined to rule out other infestations such as scabies. Although typically not necessary for diagnosis, a skin biopsy may show histopathologic changes consistent with arthropod assault. The differential diagnosis should include flea bites when lesions are located primarily on the lower legs, other arthropod assaults, scabies and urticaria from other contactants or allergens.

Treatment of bed bug bite reactions is symptomatic and includes low- to medium-potency topical corticosteroids (hydrocortisone, fluocinolone, triamcinolone, or desonide creams), topical antipruritic agents (pramoxine and calamine lotion), and oral antihistamines. In general, topical steroids should be used for no longer than 2 weeks at a time. Sites that are secondarily infected should be treated with topical or oral antibiotics such as mupirocin ointment. The most important intervention, however, is eradication of the bed bug infestation and elimination of potential sources. Because *C. lectularius* is much more

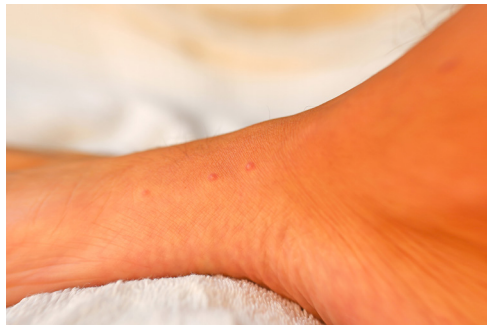

FIG 2. "Breakfast, lunch, dinner "sign from bedbug bites.

active during the night, inspection of mattresses, headboards, box springs, crevices in furniture or peeling wallpaper/paint at night may yield the best results. Often a professional exterminator must be employed to inspect and treat infested homes, apartments, shelters, or other living quarters. Unfortunately, eradication may prove to be financially burdensome and even unaffordable. Prevention is definitely the best method of control. Luggage and personal items should be inspected after traveling away from home. When a known or suspected exposure has occurred, bedding and clothing items should be laundered in water temperatures greater than 130°F or placed in the dryer on a high heat cycle for at least 30 min.[14] Exposure to temperatures above 140°F (60°C) leads to rapid killing of bed bugs.[15] Regardless of method, complete eradication is imperative to prevent spread and propagation of this pesky bug.

Scabies

Scabies is caused by the eight-legged mite *Sarcoptes scabiei* var. *hominis*, which burrows into the stratum corneum of humans causing an extremely pruritic dermatitis. The female mite lives 4–6 weeks and lays 2–4 eggs daily, each taking approximately 10 days to mature.[16] Mites are capable of surviving away from the host for 24–36 hours in room conditions (21ºC and 40–80% relative humidity).[17] The adult mite is approximately 0.3–0.5 mm in length, making it almost invisible to the human eye.[16] Thus, diagnosis relies more on clinical presentation and techniques such as mineral oil prep of skin scrapings. Pruritus induced by infestation may lead to incessant scratching, causing secondary bacterial infection with Streptococcal or Staphylococcal species. *S. scabiei* is also capable of infesting other mammals, causing Sarcoptic mange in livestock.[16]

According to the Global Burden of Disease Study in 2010, the estimated worldwide prevalence of scabies is 100 million.[18] While the exact prevalence in the United States is not known, it is certainly a common diagnosis encountered in primary care and dermatology clinics. Scabies is transmitted by intimate personal contact including sexual contact and less so by fomites such as clothing and bed linens.[16] It can be particularly problematic in places where people live in close quarters or areas plagued by poor sanitation. Although the sequelae of scabies infestation can include impetigo and superficial skin infections, the scabies mite is not known to act as a vector for infectious disease.[19]

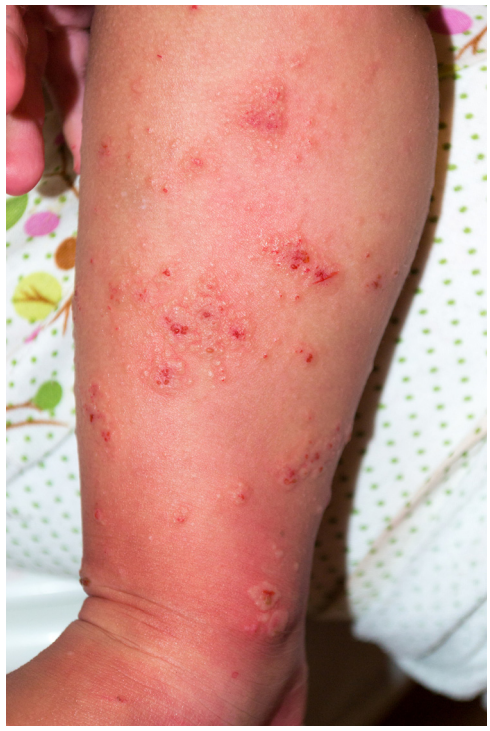

FIG 3. scabies on the leg of an infant.

The presentation of scabies infestation can be quite varied among individuals but has some classic features readily identified during examination. Patients most often complain of extreme pruritus associated with mild dermatitis that results from the underlying host immune response. Pruritus is typically worse at night or after a hot bath or shower. Symptoms usually develop several weeks after initial infestation but can present much sooner upon re-infestation.[19] Some individuals do not mount a significant immune response and are therefore asymptomatic carriers. Classic areas of infestation and dermatitis include the interdigital webspaces of the hands and feet, flexural wrists, axillae, breasts, penis, scrotum, and buttocks. However, infants are susceptible to infestation on all body surfaces including the head and may present with an atypical distribution and morphology. Classic skin findings include erythematous papules, excoriations and linear burrows, sometimes with a barely visible black "dot" at one end. Vesicopustules on the hands and feet or papulonodules on the head and/or extremities of an infant should also raise clinical suspicion for scabies infestation (Fig 3). Secondary impetiginization by group A Streptococcus or *Staphylococcus aureus* results from scratching, with yellow crusting frequently visible on physical examination. In immunocompromised individuals, such as those

with HIV/AIDS or organ transplant recipients, and patients with sensorineural impairment, infestation may be much more florid leading to crusted (Norwegian) scabies.[20] These individuals are highly contagious and typically develop thick, hyperkeratotic, crusted plaques on the ears, trunk and extremities.[20] Development of keratoderma-like plaques on acral surfaces associated with dermatitis should also prompt consideration of crusted scabies in high risk individuals. Paradoxically, individuals with crusted scabies are typically asymptomatic due to impaired immune response or itch sensation.[20] Youth with Down syndrome are also at increased risk for crusted scabies.[21,22] Regardless of presentation, scabies significantly impacts the quality of life for children and their families for a few weeks or months.[23]

Diagnosis is based on clinical presentation. A scalpel blade or disposable skin curette can be used to scrape scale and keratin debris from clinically suspicious areas for mineral oil prep and visualization under microscopy. The scabies mite itself, eggs or scybala (feces) may be seen under microscopic examination of these scrapings and confirm the diagnosis (Fig 4). In classic scabies infestation, approximately 10–15 female mites are present on the body at any given time.[24] While mites may be difficult to find on youth who present with classic scabies, patients with crusted scabies are often infested with more than one million mites and scrapings typically yield positive results.[25] Though skin scrapings are a common method of confirming diagnosis in adults, approaching a young child with a scalpel can be anxiety-provoking and traumatizing. Dermoscopy (a magnified examination of the skin employing skin surface microscopy, typically utilized by dermatologists) can also be used to visualize burrows and mites.[26,27] Similar to an otoscope, the typical dermatoscope has a magnification 10× and uses an illumination system. Mites can be readily visualized as dark-brown triangular structures located at the end of a burrow, giving rise to the "delta-wing jet" or "jet with contrail" sign.[28] The delta-shaped structure represents the anterior body of the female mite.[29] Burrows may also be seen filled with eggs, giving rise to an image likened to a "string of pearls."[29] The differential diagnosis of scabies infestation includes dyshidrotic eczema, atopic dermatitis, arthropod bites, viral exanthema, urticaria pigmentosa, and folliculitis. In infants, it may also include Langerhans cell histiocytosis and acropustulosis of infancy.

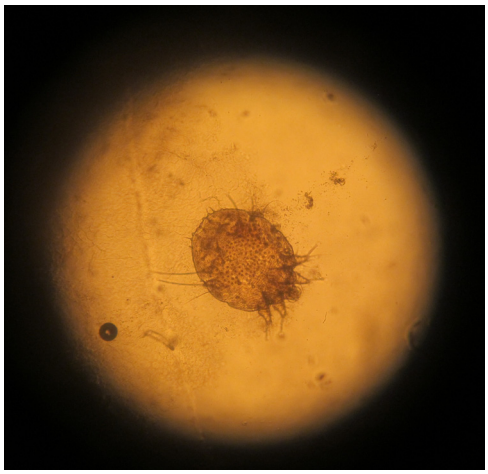

FIG 4. Human scabies mite.

Topical permethrin 5% cream is the treatment of choice for scabies infestation and is approved by the U.S. Food and Drug Administration (FDA) for use in infants 2 months and older.[30] Permethrin is applied to the skin from the neck down at bedtime and washed off 8–12 hours later. Though permethrin is both scabicidal and ovicidal, application should be repeated 1–2 weeks later to eliminate any eggs or mites that have matured in the interim period to insure maximum efficacy. Topical sulfur 2–10% in petrolatum ointment base is safe for use in infants less than 2 months of age. It should be applied to the entire skin surface, rinsed off after 24 hours and then re-applied every 24 hours for the next 2 days (with a bath taken between each application). Topical permethrin cream or sulfur ointment should be applied to the scalp and face of infants because these areas are commonly affected. Oral ivermectin, an alternative therapy used off-label in the United States, can be given in cases of treatment failure or to children who cannot tolerate topical therapy. The recommended dosage is 200 μg/kg given on day 1 and repeated 7–14 days later as it is not ovicidal.[24] Bioavailability of the drug is increased when taken with food. However, oral ivermectin is not approved by the FDA for use in children less than 5 years of age. The treatment protocol for crusted scabies as outlined by the Centers for Disease Control and Prevention (CDC) depends on disease severity but generally consists of the combination of oral ivermectin 200 μg/kg given on days 1, 2, 8, 9, and 15 (and days 22 and 29 if severe) in addition to topical permethrin cream applied every 2–3 days for 1–2 weeks.[24,31] Topical keratolytics can also be used to facilitate degradation of scale and crust and increase absorption of topical therapy.[24] Pruritus can persist for several weeks despite adequate treatment of infestation.

Education and counseling of the patient and parents regarding expectations of treatment is important. Oral antihistamines such as hydroxyzine and topical hydrocortisone cream can be used for symptomatic relief. Household contacts, including those without signs or symptoms of infestation, should be treated simultaneously to prevent re-infestation. After the completion of treatment, patients should use fresh, clean bedding and clothing. Contaminated clothes and bedding should be washed at high temperature (>50°C) or kept in a plastic bag for up to 72 hours, because mites that are separated from the human host will die within this time period. Complications of scabies infestation including secondary bacterial infection and impetigo should be treated promptly with either topical or systemic antibiotics.[16]

Head Lice

Head lice are human parasites that affect more than 100 million people worldwide each year and 6–12 million cases annually in the United States.[32,33] Treatment of head lice is a multimillion-dollar industry.[33] Head lice cause a pruritic scalp rash that can occasionally become secondarily infected. This infestation is not associated with systemic disease; however, the condition is associated with a great deal of anxiety, embarrassment, lost work, and lost school time and significant medical costs (visits to providers, medication costs, etc.).[34]

Lice are obligate ectoparasites, spending their entire life on humans. They are six-legged, wingless insects measuring 1–4 mm in length. They have gray-white, translucent, elongated bodies, which become red when engorged with blood. Lice crawl and climb; they are incapable of jumping, hopping or flight.[33,34] Species of lice infesting humans are *Pediculus humanus capitis* (head louse) (Fig 5), *Pediculus humanus corporis* (body louse), and *Phthirus pubis* (the "crab" or pubic louse). The body louse can be associated with transmission of infectious diseases (*Rickettsia*, *Bartonella*, and *Borrelia* species).[35] Pubic lice are transmitted by close physical contact and infestation is a sexually transmitted disease. Although most cases of eyebrow and eyelash infestation in children are probably nonsexual, the presence of this finding requires exclusion of possible sexual abuse. The remainder of this section will focus on head lice infestation, which is the most common of these infestations seen in the pediatric setting.

FIG 5. Head louse.

Head lice are almost exclusively transmitted by close contact and rarely by fomites. They are not associated with transmission of infectious disease (as opposed to body lice as above). Head lice lay their eggs (7–10 per day) on hair close to the scalp surface, primarily in the temple, occipital, and postauricular scalp areas.[33,35] The eggs are attached to hair by a proteinaceous cement secreted by the female louse. Eggs hatch in 7–10 days.[34,36] Lice pierce the surface of the skin for feeding, which begins within 1 minute after hatching.[37] They feed every 3–6 hours, injecting saliva which contains an anticoagulant, and sucking blood.[34,38] Once the nymphs undergo three molts and develop into adult lice over the subsequent 9–15 days, they mate, the female lays eggs and the cycle is repeated until eradication.[34,37,39] The life span of a louse is 3–4 weeks.[37] Lice die if away from the host head for more than 2 days.[33]

P. capitis is the most common form of lice to infest children, with the peak incidence seen in those 3–12 years of age. Spread is primarily through head-to-head contact, especially during play, although fomites (hair brushes, combs, barrettes, towels, and upholstery) may rarely contribute to transmission.[34,35] Girls, especially those with brown/red hair, are at increased risk due to longer hairstyles and more frequent head-to-head contact.[33,35] All socioeconomic groups can be affected, although African-American youth are less likely infested, possibly due to the shape of their hair shafts.[33,34] The spread of the infestation is enhanced with increased numbers of persons in the household.[35] There is no relationship of head lice infestation to level of hygiene.[39]

Head lice infestation elicits a hypersensitivity response to louse saliva which can take 2–6 weeks to manifest in the initial infestation.[39] Infested individuals may be asymptomatic, but often experience scalp pruritus accompanied by a dermatitis.[35] Scalp

excoriations are common. When the hair is wet, movement of head lice is impeded and identification and removal is enhanced.[33] Infested individuals usually carry less than 10 mature lice; thus, nits are typically more easily identified.[38] Occasionally, secondary skin infection, adenopathy (cervical and suboccipital) and rarely anemia (in extensive infestation) can occur.[33,35]

Clinical diagnosis of head lice infestation requires visual identification of viable nits on the hair shaft and/or live lice on the scalp. Wet combing using a nit comb is the most efficacious diagnostic method.[35,40] Nits are cemented to the hair shaft and not easily removed. Viable nits (intact operculum at the end of the nit case with viable embryo) are most likely to be found within 0.6 cm of the scalp[38] (Fig 6). Their presence can be confirmed by mounting hairs on a glass slide and viewing under the microscope. Differentiation from pseudonits, scale of psoriasis or seborrheic dermatitis, residue from hair care products, hair casts and piedra (an asymptomatic superficial fungal infection of the hair shaft) is necessary to prevent misdiagnosis.

Children and parents should be instructed on avoidance of sharing combs, brushes, barrettes, hats or other objects used to style/cover hair. Cleaning of bedding (by washing/laundering at water temperatures >140°F) and hair care items (in hot and soapy solution) belonging to infested individuals is recommended.[34,35] Affected individuals should be treated promptly to avoid disease transmission.[34] Detection and simultaneous treatment of disease in infested family members or other close contacts is crucial for successful eradication of the infestation. Cleaning and vacuuming of clothing, furniture, carpeting or other items in contact with the head of the infested person can be considered. Non-washable items can be placed in sealed plastic bags for two weeks.[33,38] Excessive cleaning/decontamination is not warranted.[38]

"No nit" school attendance policies have not been shown to be effective in reducing transmission of head lice and have no sound scientific basis.[38] The American Academy of Pediatrics, Canadian Pediatric Society, and the Public Medicine Environmental Group in the United Kingdom all discourage such policies because they are ineffective and result in missed school attendance for children and missed work attendance for parents. In most instances, infested individuals can remain in the classroom with instruction to avoid direct head contact with others. School officials should notify parents or guardians of classmates of the infested individual to check their children for infestation and treat as needed.[34]

FIG 6. Viable head louse nit (attached to hair shaft) with nymph and intact operculum.

Effective treatment (Table 1) of head lice infestation involves pediculicidal agents, mechanical removal, and possible environmental decontamination.[33] Use of pediculicidal agents should be confined to cases where viable nits or live lice are observed. Avoid application of conditioners to hair prior to use of topical pediculicides; these can coat hair and protect lice.[33] No topical pediculicide is 100% ovicidal; thus, reapplication 7–10 days after initial application is recommended.[33,37,38] Use of neurotoxic substances is associated with a potential increased risk for leukemia.[32,35]

Pyrethrins are derived from chrysanthemums and are stabilized by addition of piperonyl butoxide (e.g., RID®, Pronto®, and ClearLice®). These products are neurotoxic to lice but have very low mammalian toxicity. They are pediculicidal but not ovicidal; thus, the initial application should be followed by a repeat application 1 week later. Treatment should be applied to the scalp and rinsed out 10 min later. Resistance has been reported.[34] Common side effects are scalp burning and itching. Pyrethrins should be avoided by individuals allergic to plants belonging to the Compositae/Asteraceae family, including chrysanthemums and ragweed, allergic youth may develop dyspnea and wheezing.[39,40]

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TABLE 1. Medications for head lice treatment

	Medication	Administration	Resistance	FDA approval*	Comments
Neurotoxins	Pyrethrins w/piperonyl butoxide (OTC)	Topical application for 10 min to dry hair, repeat in 7 days	Yes	2 years	Avoid in individuals allergic to chrysanthemum and ragweed
	Permethrin 1% (OTC)	Topical application for 10 min to dry hair, repeat in 7 days	Yes	2 months	
	Lindane (Rx)	Topical application for 10 min, repeat in 7–10 days	Yes	Use with caution in patients < 50 kg	Banned in California, second-line treatment, avoid in infants, pregnant/breast-feeding women; AAP no longer recommends use as pediculicide
	Malathion 0.5% (Rx)	Topical application for 8–12 h, repeat in 7–10 days (if live lice noted)	Yes (not in United States)	6 years	Flammable
	Spinosad 0.9% (Rx)	Topical application for 10 min, repeat in 7–10 days if necessary	No	6 months	
	Topical ivermectin 0.5% (Rx)	Topical application for 10 min	No	6 months	
	Oral invermectin (Rx)	Initial dose of 200 μg/kg or 400 μg/kg, repeat in 10 days	No	Not approved	Avoid in children weighing <15 lbs, contraindicated in pregnant or breast-feeding women
Non-neurotoxins	Benzoyl alcohol 5% (Rx)	Topical application for 10 min, repeat in 7 days	No	6 months	

*Minimum age.

Permethrin 1% (Nix®) is the treatment of choice for head lice infestation. The cream rinse is applied to shampooed hair and rinsed off after 10 min.[33,34] Treatment can be repeated in 1 week. Synthetic pyrethroids, such as permethrin (Nix® and generics), have the same mechanism of action as pyrethrins, but are more environmentally stable and remain active for 2 weeks following application.[33] They have much less mammalian toxicity than treatment with certain other therapies such as malathion (an organophosphate) or lindane (an organochloride that is no longer recommended). They do not need to be avoided in individuals with plant allergies. Permethrin 5%, used for treatment of scabies, has been used off-label as a pediculicide in treatment-resistant cases. It can be applied to the scalp for several hours or overnight, then rinsed off. There are concerns regarding the reported decline in efficacy of permethrin due to rapid growth of documented worldwide resistance.[33,35,37,39] A shared site of action with dichlorodiphenyltrichloroethane (DDT) and widespread use are likely factors contributing to this loss of therapeutic efficacy.[37]

Lindane (γ-hexachlorocyclohexane), first registered in the 1950s, acts by causing neuronal hyperstimulation followed by paralysis and death of the louse. It has low ovicidal activity.[33,34] It is available as a shampoo which is rinsed out after a 10-min application. Repeat application is recommended in 7–10 days. Lindane was found to be insufficiently effective to warrant use for head lice infestation in a 1995 systemic review.[37] Banned in California since 2002 (due to concerns of contamination of waste water), lindane has a boxed warning of serious adverse events including death.[38] Seizures and other serious complications have been reported in children due to inappropriate use or intentional ingestion of the medication.[33,34] Resistance of head lice to lindane is widespread.[36] Some sources, including the American Academy of Pediatrics, recommend against use for treatment of head lice.[40] If used, it should be restricted to those who have failed or are unable to tolerate other approved therapies and should be used with extreme caution in infants, children, elderly adults, immunosuppressed individuals, those on anticonvulsants, pregnant females and nursing mothers.[33,38]

Malathion 0.5% (Ovide® and Prioderm®) is an organophosphate which irreversibly inhibits acetylcholinesterase. Lotion is applied to the hair and rinsed off 8–12 hours later. Malathion has high ovicidal activity but should be re-applied if live lice are noted 7–10 days after initial application. The product is malodorous and flammable; it can cause respiratory depression if ingested. Use of a hair dryer or curling iron after application should be avoided.[40] Skin irritation, including second-degree burns and eye irritation, can occur with use.[33,37] Safety has not been demonstrated in children under 6 years of

age.[37] Use should be reserved for those not responding/resistant to other topical products.[34] Its use may be considered during pregnancy but there is limited human data available.

Topical ivermectin 0.5% (Sklice®), a fermentation product of the soil-dwelling actinomycete *Streptomyces avermitilis*, exerts a neurotoxic effect on lice through binding to glutamate- and GABA-gated chloride ion channels. It is not directly ovicidal but is lethal to nymphs through paralysis of mouthparts.[40] It is approved for treatment of head lice in those 6 months of age and older. A single topical dose is effective in about 75% of patients. A single treatment with 1% topical ivermectin (10 min application) appears to provide a significantly higher cure of infestation and faster reduction of pruritus than a dose of oral ivermectin.[41] Reported side effects to topical ivermectin include conjunctivitis, eye irritation, and a burning sensation upon application. Resistance has not been reported. It is classified as category C for use during pregnancy.[40]

Spinosad 0.9% topical suspension (Natroba®) is a pediculicidal agent based on compounds produced by the actinomycete *Saccharopolyspora spinose.*[40] The mechanism of action involves binding to nicotinic acetylcholine receptors and antagonizing GABA receptors, causing insect paralysis and death. It appears to have ovicidal activity and is highly efficacious as a single dose.[37] Side effects include erythema, application site and ocular irritation. Resistance has not been reported. Spinosad® is classified as category B for use during pregnancy.[40]

Benzyl alcohol 5% (Ulesfia®) is non-neurotoxic chemical and kills head lice by asphyxiation.[36,37] It is approved for children 6 months of age and older. It has no ovicidal activity. Treatment consists of two 10 min applications 1 week apart.[37] Allergic reactions and skin irritation, pruritus, erythema, and eye irritation are reported side effects.[36] It is classified as category B for use during pregnancy.[40]

Dimethacones, synthetic silicone oils, in a single application have been found to be safe and effective in treatment of head lice. Studies have demonstrated superior efficacy to permethrin.[35] Given its physical mode of action (seals the breathing pores, or spiracles, of head lice), resistance is unlikely to develop.[36] Dimethicones are flammable so avoidance of exposure to heat sources during treatment is essential.[35]

Oxyphthirine is a non-flammable patented meta-emulsion that has a mechanical action that asphyxiates head lice. A single application has been demonstrated to have high efficacy and ability to remove attached nits.[36]

The combination of isopropyl myristate 50% and ST-cyclomethicone 50% is approved in Canada for treatment of head lice in children 4 years of age and older. This non-insecticidal product acts by dissolving the wax covering on the exoskeleton of the louse, leading to dehydration and death.[40] It is applied for 10 min to dry scalp, followed by a repeat application 1 week later. Erythema and pruritus are the only reported side effects.[38]

Home remedies such as mayonnaise, olive oil, margarine, and petroleum jelly have not been demonstrated to be safe and effective.[34,40] Treatments such as head shaving are unnecessarily traumatic and the use of gasoline and kerosene are unsupported and can result in skin/eye irritation and burns.[33,36] Small studies have noted some pediculicidal activity of essential oils.[38]

Oral trimethoprim-sulfamethoxazole, through its ability to kill symbiotic bacteria in the gut of the louse or perhaps through direct toxic effects on lice, has been cited in observational studies as a potential head lice treatment. This is not an FDA-approved pediculicide and potential for severe allergic reactions and promoting bacterial resistance limit its use in this setting.[33,34]

Oral ivermectin (Stromectal®), a macrocyclic lactone antihelminth used to treat onchocerciasis, strongyloidiasis, filiariasis and scabies, has shown to be an effective pediculicidal agent. It acts by interrupting neurotransmission in invertebrates.[32] It has the potential to block neural transmission once it passes the blood-brain barrier; thus, it should be used with caution in young children. Treatment consists of an initial oral dose of 200 μg/kg (and in some studies 400 μg/kg) which is repeated in 10 days.[32] Off-label use should not be administered to children weighing less than 15 pounds. Its use is contraindicated in pregnant or breastfeeding women.[39] Use of oral ivermectin should be considered in individuals with head lice infestation who have failed topical pediculicide treatment.[32]

The investigational drug abametapir acts by inhibiting metalloproteinase enzymes needed for egg development and survival of hatched lice. In unpublished studies, it has been found to exert pediculicidal and ovicidal activity.[40]

Combing with use of fine-tooth "nit combs" (e.g., Lice Comb® and Licemeister®) in conjunction with use of a pediculicidal or ovicidal agent can be performed

for esthetic reasons, to decrease diagnostic confusion or to remove emerging nits which survived medication treatment.[33,34] Combing is most effective when performed on wet hair after shampooing. Vinegar and products containing formic acid may loosen nits and enhance the process of nit removal. This process can be repeated every 1–3 days for up to 2 weeks.[34,39] Repeated wet combing has been reported to be more effective than a single pediculicide application. This method can be used in cases where there is a high level of concern regarding exposure to chemical substances (e.g., pregnant and nursing mother, infants, patients with open scalp wounds, and patients with asthma).[35]

Drug resistance has been reported with lindane, pyrethroids, permethrin, and malathion.[34,40] Pyrethroid resistance results from an amino acid mutation in neuronal sodium channels that alters the sensitivity of the head louse nervous system to the medication.[32,35] Widespread use of DDT in the mid-20th century may have contributed to the emergence of knockdown resistance (kdr) mutations in head lice.[37] While overall prevalence of resistance is rising worldwide, this does not appear to be as large of a problem in North America.[38] Many treatment "failures" can be attributed to misdiagnosis and noncompliance with treatment recommendations, incorrect treatment application or failure to treat all potentially infested contacts, acquisition of new infestation following treatment or other resistance mechanisms (e.g., cuticular modification resulting in reduced insecticide penetration).[34,35,37,38] Persistent pruritus for as long as 1–2 weeks following treatment does not represent a "failure" and can be managed with topical corticosteroids and oral antihistamines.[38] Re-treatment of persistent lice infestations should be undertaken with a product not used for the initial treatment.[37]

Candidiasis

Candida comprises the genus that causes the most frequent fungal infections in humans. This genus contains a wide range of yeasts that reproduce via budding, often by forming long chains called pseudohyphae.[42] Candida exists as an infective yeast form known as blastoconidia with invasive forms of hyphae and pseudohyphae. There are at least 15 distinct species of Candida, the most common of which is *Candida albicans*, which comprises about 50% of *Candida* infections, and is found normally colonized on the skin, mouth, intestinal tract, and vagina.[42–44] Another 40% of invasive disease is caused by *Candida tropicalis*, *Candida glabrata*, *Candida guilliermondii*, *Candida lusitaniae*, *Candida parapsilosis*, *Candida dubliniensis*, and *Candida krusei.*[43] Some species are more common in various clinical settings such as *C. glabrata* which is seen in patients with hematological malignancies. While *C. albicans* remains the most common cause of systemic candidiasis, studies of very low birth weight infants have shown *C. parapsilosis* to be an emerging threat with varying antifungal susceptibilities to common treatments.[42–44]

Candida infection remains a very serious threat to immunocompromised hosts. The organism can affect nearly all organs and tissues in the body, causing both localized and systemic infections.[42,43] T-lymphocyte mediated immunity is the primary defense mechanism against *Candida* infections. This is accomplished via phagocytosis and destruction of yeast organisms via macrophages and neutrophils. Normal cutaneous bacterial flora provides host protection by competing for nutrients with yeast, by taking over epithelial adhesion sites, and by producing metabolic byproducts toxic to yeast.[45] When there is environmental stimuli at the site of infection, *Candida* changes from the budding yeast form to hyphal and germ tube forms that produce proteolytic enzymes against keratin, collagen, laminin, fibronectin, and immunoglobulins including IgA. Hyphal invasion of the corneal layer of the eye enables the yeast to penetrate into deeper tissues.[45–49] Person to person transmission is rare and the incubation period is unknown.

Candida does not require the use of special fungal culture media to grow. Testing for susceptibilities is not necessary unless a species other than *albicans* is suspected; *C. krusei* and *C. glabrata* are not susceptible to fluconazole.[42,43]

The main classes of antifungal medications include polyenes (nystatin and amphotericin B), azoles (fluconazole, itraconazole, voriconazole, posaconazole, isavuconazole, miconazole, and clotrimazole—which are notably all fungistatic against *Candida* species), echinocandins, and flucytosine.[42]

Oral Thrush

Oral candidiasis, also known as oral thrush, is most commonly encountered in the newborn period where infants can orally acquire this infection *in utero* or postnatally *via* passage through the vaginal canal.[43] A very mild form of disease is common in infants but can also be seen in patients with poorly controlled

diabetes and the immunocompromised, including very low birth weight infants. Approximately, 20–40% of healthy children will develop thrush within the first year of life.[44,45] The prevalence of oral candidiasis is much higher in bottle-fed versus breast-fed infants.[42,43,45] Patients with asthma who are on daily inhaled corticosteroids can also develop this condition. Infants may present with pain, fussiness, or poor feeding, both often are asymptomatic.[42,45] Older children may present with a sensation of cottony or dry mouth, loss of taste, or may be asymptomatic. Lesions have a curdled milk appearance and typically appears as an adherent white plaque seen on the soft palate, uvula, tongue, or buccal mucosa. Scraping of these plaques can reveal erythema and bleeding at the base of the plaque which aids in differentiating candida infection from milk on the tongue.[45]

The differential diagnosis includes infections with organisms causing similar-appearing lesions (viral or bacterial), or trauma. The diagnosis is primarily made clinically but can be made also by scraping the lesions and using 10–20% potassium hydroxide (KOH) suspension to evaluate for yeast with or without pseudohyphae present. Other testing includes gram stain, calcofluor white, or fluorescent antibody stains.[43] Oral candidiasis in immunocompetent hosts is typically treated with oral nystatin suspension, applying 50,000 units to each cheek 4 times daily for 7–14 days, continuing for 2–3 days after resolution of the lesions. Nystatin pastilles (200,000 units) can also be used by giving 1–2 pastilles four times daily for 7–14 days.[44] Nystatin can be given via syringe directly into the mouth. Another option is oral fluconazole 6 mg/kg on day 1 followed by 3 mg/kg once daily for 7–14 days. Nystatin has a 29–80% cure rate. Fluconazole is associated with faster clearance and increased clinical cure rates, with the rates of side effects and relapse being similar. However, as the course of oral thrush is fairly benign, nystatin remains first-line therapy.[50–53]

For older children with mild disease, defined as less than 50% mucosal involvement and absence of deep, erosive lesions, treatment should commence with topical nystatin 400,000–600,000 units swished and held in the mouth as long as tolerated and then swallowed four times daily. Nystatin 200,000 units in lozenge form can be used four times daily as well.[54] A clotrimazole lozenge (10 mg) taken five to six times per day can be used as well. Lozenges are contraindicated in patients under 4 years of age due to the risk of choking. For moderate-to-severe disease in older patients, fluconazole 6 mg/kg per day orally for 7–14 days can be used.[44] Other, less commonly used treatments include clotrimazole 10 mg in troche form, five times per day applied directly to lesions (note these cannot be used in infants), and miconazole 50 mg in mucoadhesive buccal tablet form applied to the mucosal surface daily for 7–14 days. Gentian violet (0.5% or 1%) applied once to twice daily has been used in the past and is known to be effective. It can stain both lips and clothing. Gentian violet rarely causes irritation and ulceration, it is not recommended.[55]

For treatment failures, consider sources of re-infection or re-exposure as well as unusual species of *Candida*. Patients should be advised to sterilize or decolonize any items that are placed in infants mouths, such as pacifiers and bottles, and these items should be boiled after each use. A candidal infection of the breast in lactating women should also be addressed and treated.[54] Topical miconazole or clotrimazole is initially used to treat the lactating woman. Before each feeding, visible residual medication should be removed and the antifungal medication should be re-applied after each feed.

Dermatitis (Diaper and Intertrigo)

Candidal diaper dermatitis and intertrigo are the most common forms of candidiasis seen in the pediatric population. Intertrigo is an inflammatory condition of two closely opposed skin surfaces.[56] Intertriginous regions of the body include the axillae, scrotum, inguinal folds, intergluteal folds, inframmamary folds, neck folds in infants, webbed space between toes and fingers, as well as abdominal folds, particularly pannus, in obese individuals.[57] While candida diaper dermatitis is seen almost exclusively in diapered infants, intertrigo can be seen frequently in older children and adults.[45,46] This disease has a predilection for dark, moist areas of the body. It comes about due to a disruption of the normal skin flora in those with an impaired immune system, following treatment with a systemic antibiotics, topical, or oral steroids. Obesity with excessive sweating is a risk factor.[45] Chronic occlusion of the skin by wet diapers is a leading risk factor in infants and if left untreated for more than 3 days, secondary infection from microorganisms, including candida, may occur.[58] Of babies with diaper dermatitis, up to one-half are due to candida with peak incidence seen at 3–4 months of age. The rash may

occur at any age if incontinence is present.[59] Infection typically starts at the perianal area and then extends to the perineum and inguinal creases.

The rash appears as beefy erythematous plaques with scalloped borders and classic satellite lesions. Maceration is present especially in the intertriginous areas.[45] Plaques may have a fine peripheral scaling present along with pustules. These pustules, along with the satellite papules, easily rupture and develop a red base with a surrounding collarette of epidermis.[60] The rash is sometimes pruritic and can be painful if accompanied by significant ulceration. Typically plaques are confluent as opposed to primary irritant diaper dermatitis that usually spares skin folds. The differential diagnosis include tinea cruris, contact dermatitis, atopic dermatitis, inverse psoriasis, seborrheic dermatitis, erythrasma, bacterial intertrigo, herpes simplex infection, and Langerhans cell histiocytosis,. Histocytosis should always be considered in the instance of recalcitrant diaper dermatitis.

Diagnosis is largely based on the appearance and distribution with the presence of satellite lesions being a key finding. If there is clinical uncertainty, confirmation with KOH microscopy or a culture of the skin scrapings can be performed.[58–61]

Little evidence actually exists to guide optimal therapy for candida dermatitis.[61] The goal of treatment is threefold—first, to treat the active infection with topical medication, second to initiate skin care with drying agents, and finally to address any predisposing factors to reduce the risk of recurrence. Topical antifungals are typically polyene or azole medications. The polyene nystatin is used frequently. Azoles include miconazole, clotrimazole, naftifine, ketoconazole, econazole, and ciclopirox.[62,63] Ciclopirox has strong activity against both gram-positive and gram-negative bacteria that often are found along with *Candida* in intertriginous locations.[60,62] Nystatin is considered first-line therapy in either ointment or cream form, 100,000 units/g, applying 2–4 times per day for up to 14 days. Clotrimazole 1% cream 2–4 times per day for 14 days can also be used. Clotrimazole has been shown to be superior to nystatin with reduction in both symptom scores and global assessment of diaper dermatitis among infants, but cure was achieved regardless of treatment used.[62] Nystatin is effective and the least expensive modality, so it remains the treatment of choice. Topical antifungal medications are usually well tolerated with minimal adverse reactions—allergic contact dermatitis, irritation at the site of administration, pruritus, and erythema.[62] Systemic treatment has an excellent success rate of 80–100%, but is warranted solely in the most severe cases, such as widespread intertrigo with multiple site involvement, significant ulceration, and/or numerous areas of exudate and pustules.[60,64] For such moderate-to-severe disease, fluconazole orally 6 mg/kg once, then 3 mg/kg daily, or itraconazole 5–10 mg/kg per day divided twice daily for 7–14 days can be used. Treatment with the antifungal medication may be extended for up to 6 weeks or until the infection is resolved.[44] If not cleared within this time frame, one should consider an alternative diagnosis. Side effects of azole medications are uncommon and include diarrhea, nausea, abdominal pain, headache, and morbilliform rash.[64] An asymptomatic, mild elevation in transaminases may be seen in 3–4% of patients.[65] Adjunctive low-dose topical corticosteroids may help with burning, pain, and pruritus. However, one should avoid combination preparations of medications (e.g., clotrimazole 1% with betamethasone dipropionate) due to risk of skin atrophy from higher potency steroid.[61,62] One safe, well studied combination preparation includes iodoquinol 1% with hydrocortisone 2%.[66] Recurrent or recalcitrant cases should raise the possibility of immunodeficiency, including HIV infection. Endocrine abnormalities or malnutrition may be contributing factors.[56]

To reduce the risk of candidiasis the skin should be kept as clean and dry as possible. Preventative measures include changing wet diapers frequently in infants to reduce skin exposure to moisture. Diapered infants should be given "diaper-free" periods of time when possible. One should avoid occlusive clothing and promote aeration of intertriginous areas. Fungal infections often follow courses of antibacterial medications. To avoid this, it is important to use antibiotics only when necessary. Intermittent use of topical antifungals has not been confirmed to be beneficial in studies. Use of powders such as cornstarch or talcum powder create a significant respiratory risk if accidentally aspirated and should be avoided.[67]

Perleche (Angular Cheilitis)

Angular cheilitis, also known as "perleche" which comes from the French word "lecher" meaning "to lick," is a painful fissuring at the corners of the mouth and is worsened secondary to frequent lip licking.[68,69] Concomitant atopic dermatitis may predispose to this,

but it is also noted in patients with other systemic diseases, including lichen planus and various nutritional deficiencies. Predisposing factors are similar to those mentioned previously but include systemic immune suppression as well as local irritation and increased moisture.[69] Perleche is a clinical diagnosis, with microbiological confirmation rarely needed. Treatment is commonly accomplished with topical clotrimazole 1% applied twice daily for 14 days. Topical 1% hydrocortisone is also typically applied twice daily with the antifungal agent. Miconazole 2% applied twice daily for 1–3 weeks can also be used. An emollient cream such as zinc oxide or petrolatum to keep the lips moisturized should be employed to discourage frequent lip licking. In those with recurrent or difficult to treat cheilitis, one should obtain bacterial or fungal cultures from the involved areas.

Vulvovaginitis

Vulvovaginitis will occur in 75% of women at some point in their lives.[44] *Candida* vaginitis can be seen in the sexually active adolescent, but is rarely seen in prepubertal girls.[45,46] Clinical history includes vaginal soreness, burning, irritation, and often times dyspareunia. Patients will usually report creamy curd-like vaginal discharge that can sometimes be watery.[45,46] The vaginal discharge is typically odorless, in contrast to the abnormal, fish-like odor noted in bacterial vaginosis. On examination, vulvar erythema and swelling may be seen. Of note, infection with *C. glabrata* may only present with erythema and no discharge.[70] Differential diagnosis includes contact dermatitis, infection with bacteria such as with group A streptococci, and trauma. Poor or excessive hygiene and chemical irritants are the most common causes of vaginitis in prepubertal children.

Candida vaginitis can be seen in the sexually active adolescent, but is rarely seen in prepubertal girls.

Medications are used primarily in girls aged 12 years and older. Treatments include intravaginal topical clotrimazole 1% cream, inserting 1 applicator intravaginally at night for 7–14 days. A 2% cream applied nightly for 3 consecutive nights can also be used. Clotrimazole applied externally twice daily for 7 days can combat itching and irritation.[71] Miconazole 2% applied nightly for 7 days is also effective. No one treatment has been found to be superior to the others.[44] All of the above have superior efficacy to nystatin which is not recommended in the treatment of candida vulvovaginitis. Refractory or recurrent cases should receive oral antifungal therapy with single-dose fluconazole 150 mg, itraconazole 200 mg daily for 3–7 days, or ketoconazole 200–400 mg once daily until infection has resolved. Of note, ketoconazole has serious risks for hepatotoxicity and drug interactions and is infrequently used.[64,72] For severe, acute illness, fluconazole 150 mg should be taken every 72 h for 2–3 doses.[44] In the case of recurrent infections, 10–14 days of induction therapy with a topical agent above followed by fluconazole 150 mg weekly for 6 months should be considered.[44] *C. glabrata* is resistant to oral azole therapies. One should consider use of intravaginal boric acid gelatin capsule 600 mg daily for 14 days or nystatin 100,000 units as an intravaginal suppository daily for 14 days. A third-line treatment includes flucytosine cream alone or in combination with 3% amphotericin B cream daily for 14 days.[70]

Dermatophytoses

Superficial fungal infections are some of the most common infectious conditions throughout the world.[73] The dermatophytes are molds that can invade the stratum corneum of the skin or other keratinized tissues derived from epidermis, such as hair and nails. Organisms most commonly affect the scalp, feet, groin, and nails.

The three dermatophyte genera include *Trichophyton*, *Microsporum*, and *Epidermophyton*. Most of the dermatophyte species are parasitic and can cause disease in either humans or animals, often being adapted to a single or narrow range of host species. The dermatophytes are referred to as *anthropophilic*, *zoophilic*, or *geophilic*, depending on whether their primary source is a human, animal, or soil, respectively.[73]

The distribution and transmission of these fungi are generally dependent on the source of the infection.[74] Zoophilic dermatophyte fungi are animal pathogens that uncommonly cause human infection. These pathogens can elicit severe, suppurative inflammatory responses in humans. *Microsporum canis* is most prevalent zoophilic dermatophyte throughout the world, in both temperate regions and some tropical

regions. Anthropophilic dermatophyte infections chiefly cause infections of glabrous skin, skin that does not contain hair follicles, such as that over the palms and soles. *Trichophyton rubrum*, the most common dermatophyte pathogen in the world, causes tinea pedis or, in intertriginous areas of the body, tinea cruris. Other pathogens may penetrate the hair shaft, with *Trichophyton tonsurans* being the most common cause of scalp infection. Less commonly, *Microsporum* spp. may be involved. *Epidermophyton* spp. invade the intertriginous skin. Infections of the nail plate, tinea unguium, commonly occur in concert with tinea pedis. Typically, *T. rubrum* and *Trichophyton mentagrophytes* are involved. Dermatophyte infections are generally noninflammatory, easily transmissible, and readily treated with antifungal agents.

Infection of the scalp, tinea capitis, occurs mainly in children, usually between the ages of 4 and 14 years with a peak occurrence at 3–7 years. It is an infrequent infection in postpubertal individuals.[75] Tinea pedis principally occurs in adolescents or young adults. Foot infections occasionally occur in young children, but in this age group the nails may be involved without associated skin infection. Organisms infect glabrous skin largely through contact with infected desquamated skin scales. For example, tinea pedis may follow exposure in locker or shower rooms where numbers of individuals share common facilities. The geographic distribution of dermatophytes that cause tinea capitis in children is somewhat restricted, often found within defined endemic areas. Infection is acquired most often by close contact with infected hair or skin epithelial cells deposited on surfaces such as chairs, hats, combs, or brushes. Anthropophilic fungi can readily spread to other contacts, and one may need to screen classmates or family members of children with such infections.[76] Zoophilic infections do not usually spread from child to child, although family members exposed to the same source of infection may develop scalp disease. The frequency and severity of infections by each of these pathogens is likely impacted by the genetic susceptibility of the host and the virulence of the fungus.[76]

Diagnosis is typically made by history and physical examination. Laboratory diagnosis of superficial fungal infections depends on the examination and culture of scrapings or clippings from lesions. The edge of skin lesions and infected nails should be sampled. In the case of infected hairs, remove broken stubs with forceps. Material should be allowed to soften in

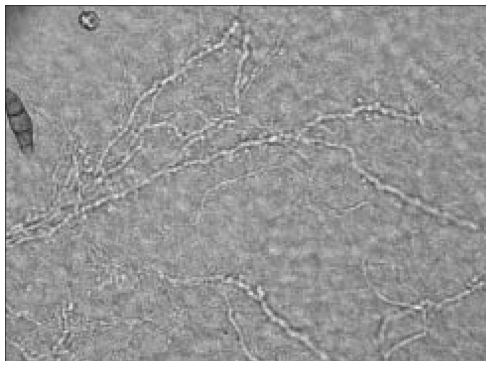

FIG 7. Skin scraping and KOH mount showing branching fungal hyphae in dermatophyte infection.

10–20% potassium hydroxide (KOH) before being examined under the microscope. Branching fungal hyphae appear in skin scales or clippings[77] (Fig 7). Most dermatologists will document infections with cultures, while primary care providers typically reserve cultures for unclear or unresponsive cases. Material may also be cultured at room temperature, usually on Sabouraud's agar containing antibiotics, and can be identified within 2 weeks.

A Wood's light examination may be helpful in some cases. Light rays with a wavelength greater than 365 nm are produced when ultraviolet light is projected through a Wood's filter.[76,77] With a Wood's light, hair in some individuals with tinea capitis, but not the skin, will fluoresce a blue-green color if infected with *M. canis* or *M. audouinii* . *Trichophyton schoenleinii* produces a paler green fluorescence of infected hair. No other dermatophytes that infect hair produce fluorescence. Except for pityriasis (tinea) versicolor, which produces a pale white-yellow fluorescence, skin dermatophyte infections do not fluoresce. Erythrasma, an infection caused by the bacterium *Corynebacterium minutissimum*, may fluoresce coral-red. For best results, the examination should be performed in a dark room with a high-intensity instrument.

Tinea Capitis

Tinea capitis is a disease caused by superficial fungal infection of the skin of the scalp with a propensity for attacking hair shafts and follicles. This is almost exclusively a disease of childhood. It is particularly common in African-American children. Clinical signs may be subtle with this infection and the presentation may vary.[75,78] Some children will present with a

diffuse scale, similar to dandruff. The infection is often accompanied by itching. There is minimal hair loss in this variant and it can be confused with dandruff, seborrhea, or atopic dermatitis. Another variant presents with circular patches and associated alopecia. Scalp infection may present as black-dots (hairs broken off at follicular orifices that appear a few millimeters above the skin surface) within patches of hair loss, representative of an infection within the hair shaft (an endothrix infection, typically caused by *T. tonsurans* or *Trichophyton violaceum*). Lymphadenopathy may be noted. Some children may present with widespread scattered pustular lesions. An aggressive host inflammatory response in some individuals may result in a kerion, a boggy, localized mass, often peppered with pustules. The pustules noted in a kerion are not a sign of secondary bacterial infection, although this may occur under crusts. A child with a kerion may have fever, pain, and lymphadenopathy. Alopecia and permanent scarring may follow.

The differential diagnosis of tinea capitis includes seborrhea, psoriasis, atopic dermatitis, and alopecia areata. A kerion can be mistaken for impetigo, furunculosis, or a bacterial abscess frequently resulting in inappropriate medical or surgical treatment.

Topical therapies are ineffective in tinea capitis.

Topical therapies are ineffective in tinea capitis. Oral griseofulvin for a minimum of 6–12 weeks remains first-line treatment because of its safety profile and no requirement for blood tests[79,80] (Table 2). Absorption of griseofulvin is improved when given with fatty foods. Treatment may need to be extended beyond 3 months in recalcitrant cases. Cultures may be useful both to identify species and to document cure (with therapy administered for a month beyond the first negative culture). Treatment with itraconazole may be helpful in cases resistant to griseofulvin therapy. In some studies, fluconazole, administered either daily for 3–6 weeks or weekly for an 8–12 week treatment course, has been shown to be effective in the treatment of tinea capitis. Oral terbinafine has an equivalent efficacy and safety profile to griseofulvin and enables a shorter treatment duration.[81] While both are effective, oral terbinafine is more effective against infections caused by *T. tonsurans* and griseofulvin more effective in *M. canis* infections. Adjunctive treatment with an antifungal shampoo (ketoconazole, selenium sulfide, povidone-iodine, or ciclopirox) 2–3 times a week for both the patient and family contacts is recommended to reduce the number of spores. All children with tinea capitis should be reevaluated monthly.

TABLE 2. Recommended dosing regimens for pediatric tinea capitis (including kerion*)

Medication	Recommended dosing	Monitoring	Notes
Griseofulvin	• 20–25 mg/kg once daily (microsized) for 6–12 weeks, or 10–15 mg/kg once daily (ultramicrosized) for 6–12 weeks. • Take with milk or fatty foods to enhance absorption.	No monitoring generally necessary in normal healthy children	• Preferred therapy. • Superior for *Microsporum* infections. • Available in tablets or liquid.
Terbinafine	• Use terbinafine for only 2–4 weeks. • 62.5 mg/day (<20 kg). • 125 mg/day (20–40 kg). • 250 mg/day (>40 kg).	• Discuss risk-benefit profile with individual patients/family, advise as to signs/symptoms of hepatotoxicity. • Obtain baseline LFTs and repeat after 1 month of treatment.	• Superior for *Trichophyton* infections. • Available in tablets or granules.
Fluconazole	6 mg/kg/day for 3–6 weeks, or 6 mg/kg once weekly for 8–12 weeks.	Periodic liver function tests (AST/ALT, alkaline phosphatase), renal function tests, electrolytes, CBC.	Available in tablets or liquid.
Itraconazole	3–5 mg/kg/day for 4–6 weeks, or 5 mg/kg daily for 1 week of each month for 2–3 months.	Baseline LFTs and repeat after 1 month of treatment.	Available in tablets or liquid.

2.5% selenium sulfide shampoo or 2% ketoconazole shampoo should be used concomitantly 2–3 times/week to prevent recurrences.

***Abbreviations*: ALT, alanine aminotransferase; AST, aspartate aminotransferase; CBC, complete blood count; LFTs, liver function tests. (Adapted with permission from Bolognia et al. (eds). *Dermatology*. 3rd edition. London: Mosby; 2012 and Bradley et al. *Nelson's Pediatric Antimicrobial Therapy*. 23rd edition. Elk Grove Village, IL: American Academy of Pediatrics; 2017.)**

***For kerion, treat concurrently with prednisone (1–2 mg/kg/day for 1–2 weeks).**

Tinea Corporis

Tinea corporis often presents as an itchy, elevated, dry, scaly papule or plaque, mildly erythematous, which spreads centrifugally to form an annular lesion (ringworm). The advancing, scaly border may have red, raised papules or vesicles. The central area becomes brown or hypopigmented and less scaly as the active border progresses outward (Fig 8). The amount of scale can vary. Sometimes plaques may spread over large areas or present with pustules. Lesions typically occur in exposed areas, but can appear anywhere.[77,82] At times, a Majocchi granuloma develops as organisms (typically *T. rubrum*) penetrate along hair follicles to the level of the dermis producing follicular-based pustules or nodules. This may be propagated by the use of corticosteroids on the initial lesions. A kerion-like presentation with a robust inflammatory response, tinea profunda, can lead to boggy, inflamed cutaneous plaques that may develop secondary bacterial abscesses.[77]

At times, tinea corporis can be mistaken for other cutaneous conditions. Granuloma annulare may confused with tinea corporis. The differential diagnosis also includes atopic or nummular dermatitis, and psoriasis, but these all generally tend to be more diffusely distributed. Pityriasis rosea, fixed drug eruptions, and erythema migrans may mimic tinea corporis. Tinea incognito occurs when topical steroids are mistakenly applied to fungal infections.[77,83] The resultant, localized inflammatory response is diminished and can give a false impression that the rash has improved. The infection loses its characteristic findings only to return in a different manner when steroid therapy is stopped. The affected area may have expanded, and develop diffuse scale and erythema with papules or pustules. It may be pruritic and have a brownish coloration. Hyphae can be seen with KOH microscopy when scaling returns.

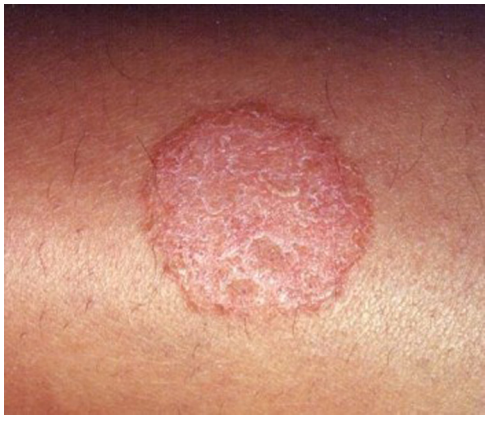

FIG 8. Tinea corporis.

Topical antifungal agents are the treatment of choice for tinea corporis.[78,84] There are many formulations that may be used. Creams should be applied to the affected area and the surrounding normal skin approximately 1–2 cm beyond the lesion. Applications should continue for a week beyond clinical cure.[84,86] The imidazoles have broad antifungal activity and have been core in the treatment of these infections. A 2–6 week treatment course with these formulations is safe and effective in eradicating dermatophyte infections. Naftifine and terbinafine (allylamines) and butenafine (benzylamine) are fungicidal and possess excellent activity against dermatophytes, may be applied once daily, use shorter treatment durations, and commonly have higher cure rates when compared to fungistatic azoles.[84] Nystatin has no efficacy against dermatophytes. Topical corticosteroids are not recommended.[86]

Tinea Pedis

Tine pedis, or athlete's foot, typically presents in adolescents and is rare among children. The prevalence of infection increase with age. Infection is more common among males. Predisposition to infection includes increased foot sweating, wearing of occlusive footwear, and exposure to communal areas (locker rooms and pools). Host factors that can enhance infections include broken skin, maceration of the skin and immunosuppression. Approximately, one-third of patients with tinea pedis have a concomitant nail infection.[85] Tinea should always be considered in the differential diagnosis of children and adolescents with foot dermatitis.

Tinea pedis commonly presents with involvement of the most lateral interdigital spaces extending medially. The web can become dry, scaly and fissured or white, macerated, and moist (Fig 9). Pruritus is the most severe and a very common complaint. An ulcerative subtype can present with more severe erosions and ulcers within the web spaces. Hyperkeratosis of the plantar and lateral aspect of the foot may sometimes be seen in the so-called moccasin type of infection. Fissures can make it painful when walking. The dorsum of the foot is spared. Less commonly, vesicles and blisters on an erythematous base may be seen on the plantar surface of the foot. The "two feet-one hand

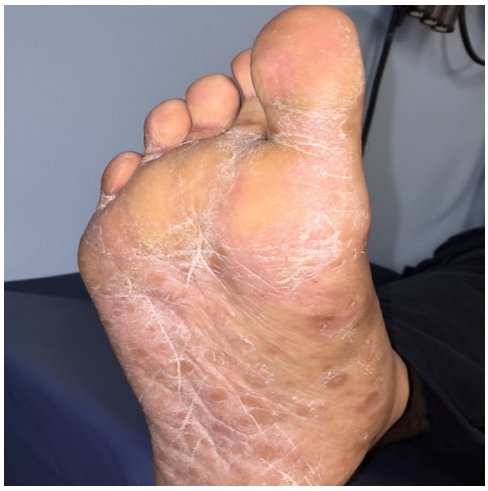

FIG 9. Dry, scaly tinea pedis (in an adolescent male with HIV-1 infection; underlying brown macular rash represents healing lesions of secondary syphilis).

syndrome" may be encountered where dermatophytes infect both feet and one hand. Dermatophytes initiate damage to the stratum corneum and can predispose to secondary bacterial infection. Subclinical tinea disease is also possible.[86]

An "id" (dermatophytid) reaction is a type of secondary immunological reaction caused by a local inflammatory infection at a distant site.[85] The most common cause is a superficial fungal infection, especially tinea pedis. Symmetric, pruritic papules are observed on the hands and sides of fingers and may last for weeks. Eradication of the triggering fungal infection results in a cure.

Superficial fungal foot infections can be confused with dyshidrotic eczema, contact dermatitis, psoriasis, and pitted keratolysis. Bacterial or candidal infection at times can also mimic tinea pedis.

Topical antifungals are effective in treating tinea pedis. Treatment duration ranges from 1 to 6 weeks according to the type of medication.[84,85] Hyperkeratotic tinea pedis requires at least 4 weeks of topical treatment applied to the entire sole and the interdigital areas. Topical imidazole agents are effective and include clotrimazole, miconazole, and ketoconazole. Terbinafine cream has been shown to be effective in persons with interdigital infection after only 1 week of treatment. Tolnaftate, ciclopirox, and butenafine are also effective. Nystatin is not recommended. If infection is extensive, fails to respond to topical therapy, or if a hyperkeratotic infection is widespread, oral therapy with antifungal medications should be utilized (terbinafine for 2 weeks, itraconazole for 1 week, or fluconazole for 2–6 weeks). Any associated toenail infections require longer treatment courses.

Measures to prevent tinea pedis include practicing good personal hygiene (regular washing of the feet with thorough drying), wearing breathable cotton socks, avoiding tight-fitting footwear, wearing sandals, keeping toenails short, and not sharing socks, slippers, shoes, and towels with infected persons.

Tinea Unguium (Onychomycosis)

Onychomycosis is a general term that refers to a fungal infection of the fingernails or toenails. Tinea unguium is a term that specifically implies dermatophyte infection of the nails. Incidence increases with age and is rare in prepubertal children. The lower incidence in children has been attributed to faster nail growth, smaller surface area for invasion, less nail trauma, lower incidence of tinea pedis, and less time spent in environments prone to infected fomites such as locker rooms. The majority of prepubertal children with onychomycosis have a first-degree relative with onychomycosis and/or tinea pedis. Nail infection commonly occurs with tinea pedis.[86]

The fungal elements in onychomycosis occur mostly in the deeper portions of the nail plate and in the hyperkeratotic nail bed rather than on the surface of the nail plate. Infection more commonly affects the toenails rather than fingernails. Infection is rarely symmetric. The nail eventually becomes "soft" and disfigured with subungual debris and nail discoloration. Infection is generally asymptomatic, but some may complain of itching in the surrounding skin. In the most common clinical presentation of tinea unguium, distal lateral subungual onychomycosis, the fungus invades the keratin along the distal and lateral aspect of the nail.[73] It presents with yellow or white discoloration in the corner of a nail fold, gradually spreads along the nail, and extends toward the proximal nail fold. The nail may thicken, break off and expose the nail bed. In another presentation, proximal subungual infection begins with proximal discoloration and spreads distally. A less common superficial form has focal, irregular white, soft areas on the nail plate that can spread to encompass the entire nail.

Griseofulvin often is used for the treatment of tinea unguium in children. Alternatives include terbinafine, itraconazole, and fluconazole.[79,84] The duration of griseofulvin therapy is prolonged (6–12 months), and

recurrences are common. Terbinafine and the azoles can be administered for shorter durations or administered as pulse regimens. They are not FDA-approved for the treatment of pediatric onychomycosis. Topical antifungal agents generally are ineffective because they do not penetrate to the lowest portions of the nail bed where new nail growth occurs. Surgical removal of the nail is not recommended because it can lead to irreversible damage to the nail bed.

Tinea Cruris

Tinea of the groin, or jock itch, tends to occur in warm weather, after excessive sweating, and the wearing of wet clothing.[75,82] Tinea cruris is mainly a disease of adolescent males and young men, rarely occurring in children. The infection is typically unilateral and starts with well-defined scaling and irritation affecting the crural fold. The rash usually involves the anterior aspect of the thigh, less commonly the scrotum. The leading edge extending onto the thighs is conspicuous and initially half-moon shaped. Papules and pustules may be seen at times. The infection may spread to the anal cleft. As with tinea pedis, there may be clustering of cases of tinea cruris in institutionalized or sport groups. Frequently the toe webs are often infected in patients with tinea cruris.

Tinea cruris is mainly a disease of adolescent males and young men, rarely occurring in children.

Erythrasma (caused by the bacterium, *Corynebacterium minutissimum*) of the groin on occasion may be confused with tinea cruris. It also causes a localized rash with itching. However, here the leading edge is less prominent than in tinea cruris and the rash is covered with fine wrinkles. The rash fluoresces a brilliant coral-red with the Wood's light.[75] An intertriginous rash may present with an erythematous, moist plaque resembling tinea infection. Obesity and moisture within the crural fold are risk factors for this inflammatory process that can become secondarily infected. Candidiasis of the groin may also mimic tinea cruris, but a central clue to the presence of *Candida* is the appearance of small satellite pustules beyond the free margin of the rash. Flexural psoriasis causes a vivid red, uniformly scaling rash in the groin, but there is ordinarily another site with typical psoriatic plaques.

Tinea of the groin responds to many of the topical antifungal creams that treat tinea corporis. Topical medications should be applied for 10–14 days. Naftifine, terbinafine, and butenafine require a shorter duration of treatment when compared with fungistatic azoles (clotrimazole, econazole, ketoconazole, oxiconazole, miconazole, and sulconazole).[79] Oral antifungal therapy is generally reserved for cases unresponsive to topical agents or can be used along with topical agents in severe cases. Effective medications are fluconazole 200 mg daily for 7 days or 150 mg once a week for 4 weeks, terbinafine 250 mg once daily for 14 days, or itraconazole 200 mg/day for 7 days. Griseofulvin 500 mg daily for 4–6 weeks is also effective.[84] Moist intertriginous lesions may be contaminated with other fungi (e.g., Candida) or bacteria. Such concomitant infections must also be addressed. Preventive measures include keeping infected areas clean and dry. Cotton underwear is preferred for men and women. Boxer shorts are preferred to briefs. The lessening of perspiration and enhancement of evaporation from the crural area are necessary prophylactic measures.

Pityriasis (Tinea) Versicolor

Pityriasis versicolor is a common superficial fungal disorder of the skin characterized by multiple round to oval sharply demarcated macules and patchy, thin scaly papules and plaques distributed over the upper portions of the trunk, proximal arms, and occasionally the neck or face (Fig 10). It is caused by the yeast forms of the dimorphic fungus *Malassezia furfur*.[86] The majority of cases present in adolescents, possibly in relation to the lipophilic nature of the organism and the sebum-rich environment of the affected regions. Sun exposure accentuates differences in skin pigmentation between affected and unaffected areas. Lesions may be hypopigmented or hyperpigmented depending on the patient's complexion and history of sun exposure. Risk factors include oily skin, excessive sweating, warm temperatures, high humidity, and immunosuppression. An equal prevalence between the sexes has been noted. A KOH preparation of scrapings will reveal short fungal hyphae and spore clusters ("spaghetti and meatballs"). Since *M. furfur* is part of the normal skin flora, cultures are not helpful.

The differential diagnosis of pityriasis versicolor includes vitiligo, pityriasis alba, postinflammatory

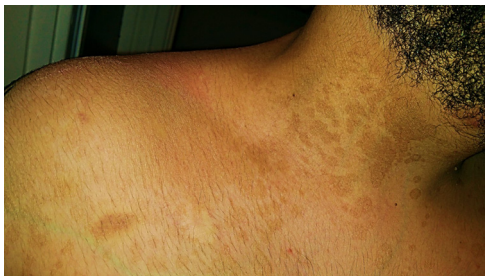

FIG 10. Pityriasis (tinea) versicolor.

hypopigmentation or hyperpigmentation, pityriasis rosea, and psoriasis. Clinical features can generally differentiate these disorders.

The most appropriate therapy for pityriasis versicolor is the application of a topical azole (e.g., clotrimazole, econazole, or miconazole creams) or terbinafine cream for 4 weeks.[86] Pityriasis versicolor can be treated with daily topical selenium sulfide 2.5% shampoo, left on affected areas for 10 min before rinsing off and repeated 5 times weekly for 4–6 weeks. One might also continue treatment 2–3 times a week for prevention. Systemic therapy with a single dose of either oral fluconazole or itraconazole should be considered for widespread or refractory disease.[79,84] Neither oral terbinafine nor griseofulvin is effective. Because the yeast is a skin commensal, recurrence is common and repeat topical antifungal treatment may be required. Pigmentary changes may take months to clear, even after eradication of the fungus.[79,84]

Endemic Mycoses

The endemic mycoses are a group of dimorphic fungi that occasionally cause skin and soft tissue infections in children. They are typically found in mycelial form at room temperature and on growth media, while exhibiting their yeast form at body temperature. This article will cover blastomycosis, histoplasmosis, and coccidioidomycosis. Inhalation of spores followed by pulmonary infection is the primary route of exposure and pathogenesis for all three organisms, but this article will cover only the skin and soft tissue manifestations of the disease. There are also regionally important fungi, not addressed in this article including *Cryptococcus gattii*, *Penicillium marnefei*, and *Sporothrix schenckii*. If history, location, or failure to achieve a clinical response occurs for a particular patient, then it may be necessary to consider these other fungi.

Blastomycosis

Blastomycosis is the syndrome caused by *Blastomyces dermatitidis*. Blastomycosis was originally described in Chicago in the late 1890s, and labeled "Chicago disease." Further knowledge gained from outbreaks expanded its region of endemicity to the southeastern United States. It is now known to have a world-wide distribution. In North America, the regions bordering the Great Lakes and the Ohio and Mississippi River valleys, the so-called balstomycosis belt, are the endemic regions.[87] All age groups are susceptible to infection with blastomyces, but only about 10% of cases occur in children.[88] Infection typically occurs after spores are inhaled, however, direct inoculation via dog bite has been reported.[89] Human to human transmission is not typical, though there are case reports of neonatal blastomycosis thought to be due to disseminated maternal disease with congenital infection.[90] Two neonates developed respiratory distress due to pulmonary infection 3 weeks after birth and both succumbed to the infection. Both mothers had cutaneous lesions caused by blastomyces.

Once inside the lung, the fungal spores transition to yeast forms. Only half of infected children are initially symptomatic. The constellation of symptoms is similar to typical bacterial pneumonia, and lobar consolidation is seen in these cases. Patients may respond to antibiotics initially, but symptoms will recur.[91] From the lung, the fungus spreads locally and disseminates. Blastomyces can disseminate anywhere in the body including bone and skin via hematogenous routes. Dissemination is more frequent in children, and is seen in up to 50% of children with blastomyces infection.[92] The classic verrucous skin lesions that are described in adults are not typical for infection in children. When pustular lesions or lesions with purulent drainage are found on the skin of children, one must suspect a deeper infection, such as an underlying osteomyelitis. No clinical syndrome is pathognomonic for blastomycosis, so an unequivocal diagnosis must be made by recovering organisms from infected tissue. Visualizing the characteristic yeast form leads to a presumptive diagnosis. In addition, blastomyces is easily found in bronchial washings in those children with lung abnormalities. Serologic tests that target the yeast phase antigen can also be performed. However, these cross-react with *Histoplasma capsulatum* and are non-specific. A commercial test for detecting blastomyces antigen in urine, blood and other fluids is also

available. However, the test cross-reacts with *H. capsulatum*.[93] Given the likelihood of disseminated disease in children, expert consultation should be considered in treatment of these patients. Mild-to-moderate, localized disease could be treated with itraconazole. Moderate-to-severe disease, including disseminated disease requires treatment with amphotericin. Use of fluconazole is not recommended, and there is no information regarding the effectiveness of newer azoles.[93]

Histoplasmosis

Histoplasmosis is the most common endemic fungal infection in humans.[94] Its area of endemicity is typically in the regions surrounding the Ohio, Mississippi, and St. Lawrence rivers. There is a strong association between the presence of bird and bat guano in soil and the presence of *H. capsulatum.* Birds are not infected by the fungus, and attempts to isolate *H. capsulatum* have not been successful. Bats can carry the fungus in their gastrointestinal tracts and may shed the fungus. In areas where avians roost, the fungus is found most often where the guano is decaying and mixed with soil.[94] As with the other endemic fungi, infection almost universally occurs after inhalation of spores. Totally, 95% of infections are self-limited. The incubation period depends on the size of the inoculum but is typically 1–3 weeks. Infections in children are often asymptomatic. When symptomatic, pulmonary infections result in non-specific symptoms of fever, and cough.[94] Dissemination is more likely to occur in patients with impaired cellular immunity and in infants under 1 year of age. Residual findings of old granulomatous disease in the lung parenchyma and calcifications in the hilar and mediastinal lymph nodes are typical. There are also rare case reports of apparent direct inoculation of the fungus from a penetrating outdoor injury resulting in primary, localized infections. These case reports have failed to provide sufficient evidence to exclude subclinical dissemination of histoplasmosis.[95]

Skin and soft tissue manifestations of histoplasmosis are not common, and are not specific to histoplasma infection. They typically occur in patients with impaired cellular immunity, including patients with HIV/AIDS, primary immunodeficiency disorders, transplant patients, and patients receiving chemotherapy for malignancies of the reticuloendothelial system.[96] Skin lesions have a diverse appearance, including pustules, folliculitis, papules, ulcerations, and rosacea-like eruptions and are non-specific. Oral ulcerations, including tongue ulcerations, have been described.[97] Diagnosis of histoplasmosis in these lesions can be made by microscopic examination of tissue specimens or skin biopsy. Skin manifestations of histoplasmosis should prompt an investigation for disseminated infection. In disseminated histoplasmosis, cultures can be useful in making the diagnosis. Bone marrow and fungal blood cultures can be helpful. Fungal cultures do not yield rapid results. Antigen studies are commercially available and can be performed on a variety of body fluids. Their sensitivity is highest in severe pulmonary disease and disseminated infection. They do cross-react with other fungal antigens and may not be as specific. Antibodies require 4–8 weeks to develop and may not be useful in patients with acute, disseminated disease. Antibody assays have lower sensitivity among immunocompromised patients.[95]

Histoplasmosis is the most common endemic fungal infection in humans.

Antifungal treatments are often unnecessary for self-limited pulmonary disease, and is almost never warranted for children where previous infection is suspected such as old granulomatous calcifications noted on chest radiographs. Skin and soft tissue manifestations should prompt concern for disseminated disease, especially in immunosuppressed patients. Expert consultation with an infectious disease specialist is necessary for these cases. Mild-moderate disease can be treated with itraconazole. Moderate and severe infections, and those in immunocompromised patients are treated with amphotericin.[95]

Coccidioidomycosis

Coccidioidomycosis is a common regional infection caused by two clinically indistinguishable fungal species, *Coccidioides immitis* and *Coccidioides posadasii*. It is colloquially known as San Joaquin Fever or Valley fever. Coccidioides is exclusively found in the western hemisphere, within 40° latitude of either side of the the equator. In the United States, the area of endemicity is limited to southern California, Arizona,

Nevada, and New Mexico. There are an estimated 150,000 infections each year. Spores become airborne during wind and dust storms, or due to activities like construction and farming. Their small size enables them to reach human alveoli with inhalation, and cause disease. There are also case reports of direct cutaneous inoculation by contaminated objects.[98] An individual's susceptibility to primary infection is not affected by age, sex, or racial background. However, there are a variety of factors that affect the likelihood of disseminated or severe disease. Neonates are particularly vulnerable to dissemination from inhaled coccidioidomycosis. There are a few case reports of maternal-fetal transmission, though there doesn't appear to be a significant risk of congenital infection.[99] Immunocompromised patients, including AIDS patients, transplant patients, pregnant women in the third trimester, and those who use TNF-α inhibitors and similar drugs are also at increased risk of disseminated disease. Filipinos and African Americans are also at a higher risk of disseminated disease.[100] The typical incubation period following exposure is 1–4 weeks.

Outside of the neonatal period, dissemination occurs less frequently in children compared to adults. Extrapulmonary spread can occur anywhere in the body, but bone and soft tissue lesions are most frequent. The most common skin manifestation of disseminated coccidioidomycosis is verrucous granuloma.[101] This is a non-specific chronic ulceration, usually on the nasolabial fold. Chronic osteomyelitis can occur as well. A total of 60% of cases involve only a single bone. The bones most commonly involved include vertebrae, tibia, metatarsals, skull and metacarpals. When vertebrae are involved, the disc is usually spared. Chronic osteomyelitis may result in inflammation of the overlying skin and draining sinus tracts.[98]

The organism is easily detected by microscopic examination and culture of purulent material. The yeast form of the organism does not pick up Gram stain. Enzyme immunoassays should be used to detect early disease. However, IgG antibodies sometimes take several months to appear and serologic testing may have diminished sensitivity. In the setting of suspected disseminated disease, diagnosis should be made by histopathologic verification of yeast in the tissue. Imaging can be useful in identifying chronic osteomyelitis in disseminated disease, demonstrating non-specific, lytic lesions in infected bones.[102]

Conclusion

Children and adolescents with either ectoparasitic infestations or mucocutaneous fungal infections are often seen by primary care clinicians. Understanding the epidemiology, transmission, and clinical manifestations associated with these conditions is important to guide proper management. While laboratory studies can be very helpful in the management of these conditions, diagnoses are made predominantly by obtaining a careful history and performing a thorough physical examination. To aid in clinical diagnosis, the reader is encouraged to view the excellent collection of infectious disease images at the American Academy of Pediatrics' Red Book Online® site at https://redbook.solutions.aap.org/visual-library.aspx. Appropriate diagnosis will guide the clinician toward effective management strategies. Finally, efforts should be made to provide patients and families with measures to prevent these conditions.

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