Common Skin Infections
Nathaniel C. Cevasco
Kenneth J. Tomecki

Bacterial infections

Humans are natural hosts for many bacterial species that colonize the skin as normal flora. *Staphylococcus aureus* and *Streptococcus pyogenes* are infrequent resident flora, but they account for a wide variety of bacterial pyodermas. Predisposing factors to infection include minor trauma, preexisting skin disease, poor hygiene, and, rarely, impaired host immunity.

Impetigo

*Definition and Etiology*

Impetigo is a superficial skin infection usually caused by *S. aureus* and occasionally by *S. pyogenes*.

*Prevalence and Risk Factors*

Impetigo affects approximately 1% of children.

*Pathophysiology and Natural History*

*S. aureus* produces a number of cellular and extracellular products, including exotoxins and coagulase, that contribute to the pathogenicity of impetigo, especially when coupled with preexisting tissue injury. Impetigo commonly occurs on the face (especially around the nares) or extremities after trauma.

*Signs and Symptoms*

Two clinical types of impetigo exist: nonbullous and bullous. The nonbullous type is more common and typically occurs on the face and extremities, initially with vesicles or pustules on reddened skin. The vesicles or pustules eventually rupture to leave the characteristic honey-colored (yellow-brown) crust (*Fig. 1*). Bullous impetigo, almost exclusively caused by *S. aureus*, exhibits flaccid bullae with clear yellow fluid that rupture and leave a golden-yellow crust.

*Diagnosis*

Diagnosis is by clinical presentation and confirmation by culture.¹
Treatment

For most patients with impetigo, topical treatment is adequate, either with bacitracin (Polysporin) or mupirocin (Bactroban), applied twice daily for 7 to 10 days. Systemic therapy may be necessary for patients with extensive disease (Table 1).² ³

Table 1: Treatment of Impetigo

<table>
<thead>
<tr>
<th>Topical</th>
<th>Systemic</th>
<th>Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First-Line Treatment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mupirocin bid for 7-10 days</td>
<td>Dicloxacillin</td>
<td>250-500 mg PO qid for 5-7 days</td>
</tr>
<tr>
<td></td>
<td>Amoxicillin plus clavulanic acid;</td>
<td>25 mg/kg PO tid; 250-500 mg PO qid for 10 days</td>
</tr>
<tr>
<td></td>
<td>cephalaxin</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Clavulanic acid</td>
<td></td>
</tr>
<tr>
<td><strong>Second-Line Treatment</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>(Penicillin allergy)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Azithromycin</td>
<td>500 mg PO × 1, then 250 mg PO daily for 4 days</td>
</tr>
<tr>
<td></td>
<td>Clindamycin</td>
<td>15 mg/kg/day PO tid for 10 days</td>
</tr>
<tr>
<td></td>
<td>Erythromycin</td>
<td>250-500 mg PO qid for 5-7 days</td>
</tr>
</tbody>
</table>

Folliculitis, Furunculosis, and Carbunculosis

**Definition and Etiology**

Folliculitis is a superficial infection of the hair follicles characterized by erythematous, follicular-based papules and pustules. Furuncles are deeper infections of the hair follicle characterized by inflammatory nodules with pustular drainage, which can coalesce to form larger draining nodules (carbuncles).

**Pathophysiology and Natural History**

*S. aureus* is the usual pathogen, although exposure to *Pseudomonas aeruginosa* in hot tubs or swimming pools can lead to folliculitis. In general, folliculitis is a self-limited entity. Occasionally, a pustule enlarges to form a tender, red nodule (furuncle) that becomes painful and fluctuant after several days. Rupture often occurs, with discharge of pus and necrotic material. With rupture, the pain subsides and the redness and edema diminish.

**Signs and Symptoms**

Folliculitis is generally asymptomatic, but it may be pruritic or even painful. Commonly affected areas are the beard, posterior neck, occipital scalp, and axillae (Fig. 2). Often a continuum of folliculitis, furunculosis (furuncles), arises in hair-bearing areas as tender, erythematous, fluctuant nodules that rupture with purulent discharge (Fig. 3). Carbuncles are larger and deeper inflammatory nodules, often with purulent drainage (Fig. 4), and commonly occur on the nape of the neck, back, or thighs. Carbuncles are often tender and painful and occasionally accompanied by fever and malaise.¹ ² ³

**Diagnosis**
Diagnosis is by clinical presentation and confirmation by culture.

**Treatment**

Topical treatment with clindamycin 1% or erythromycin 2%, applied two or three times a day to affected areas, coupled with an antibacterial wash or soap, is adequate for most patients with folliculitis. Systemic antistaphylococcal antibiotics are usually necessary for furuncles and carbuncles, especially when cellulitis or constitutional symptoms are present. Small furuncles can be treated with warm compresses three or four times a day for 15 to 20 minutes, but larger furuncles and carbuncles often warrant incision and drainage. If methicillin-resistant *S. aureus* (MRSA) is implicated or suspected, vancomycin (1-2 g IV daily in divided doses) is indicated coupled with culture confirmation. Antimicrobial therapy should be continued until inflammation has regressed or altered depending on culture results. Treatment is summarized in Table 2.

Table 2: Treatment of Folliculitis, Furunculosis, and Carbunculosis

<table>
<thead>
<tr>
<th>Folliculitis</th>
<th>Furunculosis/Carbunculosis</th>
<th>Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td>First-Line Treatment</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Topical clindamycin/ erythromycin bid</td>
<td>Incision and drainage</td>
<td>bid</td>
</tr>
<tr>
<td></td>
<td>Dicloxacillin</td>
<td>250-500 mg PO qid for 5-7 days</td>
</tr>
<tr>
<td></td>
<td>Amoxicillin plus calvulanic acid; cephalexin</td>
<td></td>
</tr>
<tr>
<td>Antibiotic wash (e.g. chlorhexidine) bid</td>
<td>Clavulanic acid;</td>
<td>bid</td>
</tr>
<tr>
<td></td>
<td>Warm compresses</td>
<td>tid</td>
</tr>
<tr>
<td>Second-Line Treatment (MRSA)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doxycycline (2-8 weeks depending on severity)</td>
<td>Doxycycline</td>
<td>100 mg PO bid (2-8 weeks depending on severity)</td>
</tr>
<tr>
<td></td>
<td>Vancomycin</td>
<td>1-2 g IV daily in divided doses for 7 days</td>
</tr>
</tbody>
</table>

**Ecthyma**

**Definition and Etiology**

Ecthyma is a cutaneous infection characterized by thickly crusted erosions or ulcerations. Ecthyma is usually a consequence of neglected impetigo and often follows impetigo occluded by footwear or clothing.

**Prevalence and Risk Factors**

Ecthyma typically occurs in homeless persons and soldiers based in hot and humid climates.

**Pathophysiology and Natural History**

*S. aureus* or *S. pyogenes* is the usual pathogen of ecthyma. Untreated staphylococcal or streptococcal impetigo can
extend more deeply, penetrating the dermis, producing a shallow crusted ulcer. Ecthyma can evolve from a primary pyoderma, in a pre-existing dermatosis, or at the site of trauma.

**Signs and Symptoms**

Infection begins with vesicles and bullae that progress to punched-out ulcerations with an adherent crust, which heals with scarring. The most common site of infection is the legs.

**Diagnosis**

Diagnosis is by clinical presentation and confirmation by culture.3,4

**Treatment**

Treatment is summarized in Table 3.3

Table 3: Treatment of Ecthyma

<table>
<thead>
<tr>
<th>Topical</th>
<th>Dosing</th>
<th>Systemic</th>
<th>Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First-Line Treatment</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Warm compresses</td>
<td>qid</td>
<td>Dicloxacillin</td>
<td>250-500 mg PO qid for 5-7 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Amoxicillin plus clavulanic acid</td>
<td>25 mg/kg PO tid</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clavulanic acid</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cephalexin</td>
<td>40-50 mg/kg/day PO for 10 days</td>
</tr>
<tr>
<td><strong>Second-Line Treatment (Penicillin Allergy)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Azithromycin</td>
<td>500 mg PO × 1, then 250 mg PO daily for 4 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Clindamycin</td>
<td>15 mg/kg/day PO tid for 10 days</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Erythromycin</td>
<td>250-500 mg PO qid for 5-7 days</td>
</tr>
</tbody>
</table>

**Erysipelas and Cellulitis**

**Definition and Etiology**

Erysipelas is a superficial cutaneous infection of the skin involving dermal lymphatic vessels. Cellulitis is a deeper process that extends to the subcutis.

**Prevalence and Risk Factors**

Erysipelas has a predilection for young children and the elderly. Lymphedema, venous stasis, web intertrigo, diabetes mellitus, trauma, alcoholism, and obesity are risk factors in the adult patient.3,4

**Pathophysiology and Natural History**
Group A β-hemolytic streptococcus is the most common pathogen responsible for erysipelas, and *S. aureus* is by far the most common pathogen for cellulitis. *S. pyogenes* produces enzymes that promote infection with systemic manifestations, such as fever and chills, tachycardia, and hypotension. Left untreated, cellulitic skin can become bullous and necrotic, and an abscess or fasciitis, or both, can occur.

**Signs and Symptoms**

Classically, erysipelas is a tender, well-defined, erythematous, indurated plaque on the face or legs (Fig. 5). Cellulitis is a warm, tender, erythematous, and edematous plaque with ill-defined borders that expands rapidly. Cellulitis is often accompanied by constitutional symptoms, regional lymphadenopathy, and occasionally bacteremia (Fig. 6).3-4

**Diagnosis**

Diagnosis is by clinical presentation and confirmation by culture (if clinically indicated, i.e., bullae or abscess formation).

**Treatment**

Penicillin (250-500 mg, qid × 7-10 days) is the treatment of choice for erysipelas; parenteral therapy may be necessary for extensive or facial disease. An oral antistaphylococcal antibiotic is the treatment of choice for cellulitis; parenteral therapy is warranted for patients with extensive disease or with systemic symptoms as well as for immunocompromised patients. Good hygiene, warm compresses three or four times a day for 15 to 20 minutes, and elevation of the affected limb help to expedite healing. Treatment is summarized in Table 4.3

**Table 4: Treatment of Erysipelas and Cellulitis**

<table>
<thead>
<tr>
<th></th>
<th>Erysipelas</th>
<th>Dosing</th>
<th>Cellulitis</th>
<th>Dosing</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>First-Line Treatment</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Penicillin</td>
<td></td>
<td>500 mg PO qid for 10 days</td>
<td>Dicloxacillin</td>
<td>250-500 mg PO qid for 5-7 days</td>
</tr>
<tr>
<td>Dicloxacillin</td>
<td></td>
<td>500 mg PO qid for 5-7 days</td>
<td>Amoxicillin plus clavulanic acid</td>
<td>25 mg/kg PO tid</td>
</tr>
<tr>
<td>Warm compresses</td>
<td></td>
<td>tid</td>
<td>Warm compresses</td>
<td>tid</td>
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<tr>
<td><strong>Second-Line Treatment</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><em>Methicillin-Resistant Staphylococcus aureus</em></td>
<td></td>
<td></td>
<td></td>
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</tr>
<tr>
<td>Linezolid</td>
<td></td>
<td>600 mg PO bid for 7-14 days</td>
<td>Linezolid</td>
<td>600 mg PO bid for 7-14 days</td>
</tr>
<tr>
<td>Vancomycin</td>
<td></td>
<td>1-2 g IV daily in divided doses for 7 days</td>
<td>Vancomycin</td>
<td>1-2 g IV daily in divided doses for 7 days</td>
</tr>
<tr>
<td><strong>Penicillin Allergy</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Clindamycin</td>
<td></td>
<td>15 mg/kg/day PO tid for 10 days</td>
<td>Azithromycin</td>
<td>500 mg PO × 1, then 250 mg PO daily for 4 days</td>
</tr>
</tbody>
</table>
Necrotizing Fasciitis

**Definition and Etiology**

Necrotizing fasciitis is a rare infection of the subcutaneous tissues and fascia that eventually leads to necrosis. Predisposing factors include injuries to soft tissues, such as abdominal surgery, abrasions, surgical incisions, diabetes, alcoholism, cirrhosis, and intravenous drug abuse.\(^5\)\(^,\)\(^6\)

**Pathophysiology and Natural History**

*S. pyogenes* can be the sole pathogen responsible for necrotizing fasciitis, but most patients have a mixed infection with other aerobes (groups B and C streptococci, MRSA) and anaerobes (*Clostridium* spp).

**Signs and Symptoms**

Infection begins with warm, tender, reddened skin and inflammation that rapidly extends horizontally and vertically. Necrotizing fasciitis commonly occurs on the extremities, abdomen, or perineum or at operative wounds (Fig. 7). Within 48 to 72 hours, affected skin becomes dusky, and bullae form, followed by necrosis and gangrene, often with crepitus. Without prompt treatment, fever, systemic toxicity, organ failure, and shock can occur, often followed by death. Computed tomography (CT) or magnetic resonance imaging (MRI) can help to delineate the extent of infection. Biopsy for histology, Gram stain, and tissue culture help to identify the causative organism(s).\(^5\)\(^,\)\(^6\)

**Diagnosis**

Diagnosis is by clinical presentation; CT or MRI; skin biopsy for pathology, Gram stain, and tissue culture; culture of fluid from bullae or fluctuant plaques; and blood cultures.

**Treatment**

Necrotizing fasciitis is a surgical emergency requiring prompt surgical debridement, fasciotomy, and, occasionally, amputation of the affected extremity to prevent progression to myonecrosis. Treatment with parenteral antibiotics (usually gentamicin and clindamycin) is mandatory. Even with treatment, mortality approaches 70%.

Fungal and yeast infections

**Dermatophytosis**

**Definition and Etiology**

*Dermatophytosis* implies infection with fungi, organisms with high affinity for keratinized tissue, such as the skin, nails, and hair. *Trichophyton rubrum* is the most common dermatophyte worldwide.

**Pathophysiology and Natural History**
Three fungal genera—*Trichophyton*, *Microsporum*, and *Epidermophyton*—account for the vast majority of infections. Fungal reservoirs for these organisms include soil, animals, and infected humans.

**Signs and Symptoms**

Tinea pedis (athlete's foot) is the most common fungal infection in humans in North America and Europe. Affected skin is usually pruritic, with scaling plaques on the soles, extending to the lateral aspects of the feet and interdigital spaces (Fig. 8), often with maceration.

Tinea cruris (jock itch) occurs in the groin and on the upper, inner thighs and buttocks as scaling annular plaques (Fig. 9); disease is more common in men and typically spares the scrotum.

Tinea capitis, or fungal infection of the scalp, is most common in children. It is characterized by scaly, erythematous skin, often with hair loss. Tinea capitis can resemble seborrheic dermatitis. Kerion celsi is an inflammatory form of tinea capitis, characterized by boggy nodules, usually with hair loss and regional lymphadenopathy.

Tinea corporis (body), faciei (face), and manuum (hands) represent infections of different sites, each invariably with annular scaly plaques. Tinea unguium (onychomycosis) is fungal nail disease, characterized by thickened yellow nails and subungual debris (Fig. 10).

Potassium hydroxide preparation or culture help to establish the diagnosis for all forms of fungal infections.

**Diagnosis**

Diagnosis is by clinical presentation, KOH examination, and fungal culture.

**Treatment**

For most patients, topical treatment with terbinafine (Lamisil), clotrimazole (Lotrimin, Mycelex), or econazole (Spectazole) cream is adequate when applied twice daily for 6 to 8 weeks. For onychomycosis, tinea capitis, and extensive dermatophyte disease, systemic treatment is often necessary: itraconazole (Sporanox) or terbinafine (Lamisil) for nail disease, and griseofulvin or fluconazole for scalp or extensive dermatophyte disease. Treatment is summarized in Table 5.

**Table 5: Treatment of Dermatophytosis**

<table>
<thead>
<tr>
<th>Limited Disease</th>
<th>Onychomycosis</th>
<th>Tinea Capitis, Extensive Dermatophyte Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Terbinafine bid for 4 weeks</td>
<td>Itraconazole 3-5 /kg/day PO for 4-6 weeks</td>
<td>Griseofulvin 20-25 /kg/day PO for 8 weeks*</td>
</tr>
<tr>
<td>Clotrimazole bid for 4 weeks</td>
<td>Terbinafine 3-6 /kg/day PO for 4-8 weeks</td>
<td>Fluconazole 6 /kg/day PO for 20 days</td>
</tr>
<tr>
<td>Econazole bid for 4 weeks</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Pediatric dose for microsize form.

**Candidiasis**

www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/dermatology/common-skin-infections/
Definition and Etiology

*Candidiasis* refers to a diverse group of infections caused by *Candida albicans* or by other members of the genus *Candida*. These organisms typically infect the skin, nails, mucous membranes, and gastrointestinal tract, but they also cause systemic disease.

Prevalence and Risk Factors

Infection is common in immunocompromised patients, diabetics, the elderly, and patients receiving antibiotics.

Pathophysiology and Natural History

*Candida albicans* accounts for 70% to 80% of all candidal infections. *C. albicans* commonly resides on skin and mucosal surfaces. Alterations in the host environment can lead to its proliferation and subsequent skin disease.

Signs and Symptoms

Candidal intertrigo is a specific infection of the skin folds (axillae, groin), characterized by reddened plaques, often with satellite pustules (Fig. 11). Thrush is oropharyngeal candidiasis, characterized by white nonadherent plaques on the tongue and buccal mucosa. Paronychia is an acute or chronic infection of the nail characterized by tender, edematous, and erythematous nail folds, often with purulent discharge (Fig. 12); this disease is common in diabetics. Angular cheilitis is the presence of fissures and reddened scaly skin at the corner of the mouth, which often occurs in diabetics and in those who drool or chronically lick their lips (Fig. 13).

Candidal vulvovaginitis is an acute inflammation of the perineum characterized by itchy, reddish, scaly skin and mucosa; creamy discharge; and peripheral pustules. The counterpart in men is balanitis, characterized by shiny reddish plaques on the glans penis, which can affect the scrotum. Balanitis occurs almost exclusively in uncircumcised men.²

Diagnosis

Diagnosis is by clinical presentation, KOH examination, and fungal culture.

Treatment

For candidal intertrigo and balanitis, topical antifungal agents such as clotrimazole, terbinafine, or econazole cream, applied twice daily for 6 to 8 weeks, is usually curative when coupled with aeration and compresses. For thrush, the treatment is nystatin suspension or clotrimazole troches four to six times daily until symptoms resolve. Systemic antifungal drugs, such as fluconazole 100 to 200 mg/day or itraconazole 100 to 200 mg/day, for 5 to 10 days may be necessary for severe or extensive disease. For paronychia, treatment consists of aeration and a topical antifungal agent such as terbinafine, clotrimazole, or econazole for 2 to 3 months; occasionally, oral antistaphylococcal antibiotics are needed, coupled with incision and drainage for secondary bacterial infection. Cheilitis resolves with aeration, application of a topical antifungal agent, and discontinuation of any aggravating factors. A single 150-mg dose of fluconazole, coupled with aeration, is usually effective for vulvovaginitis.¹⁰ Treatment is summarized in Box 1.

Box 1: Treatment for Candidiasis

Intertrigo or Balanitis

- Terbinafine bid for 4 weeks
- Clotrimazole bid for 4 weeks
Paronychia

- Terbinafine bid for 2-3 months
- Clotrimazole bid for 2-3 months
- Econazole bid for 2-3 months
- Minimize wet work

Oral Candidiasis

- Nystatin suspension/clotrimazole troches, 5 × daily
- Fluconazole 100-200 mg/day PO for 5-10 days
- Itraconzazole 100-200 mg/day PO for 5-10 days

Vulvovaginal Candidiasis

- Fluconazole 150 mg PO × 1 dose
- Aeration

Tinea (Pityriasis) Versicolor

Definition and Etiology

Tinea versicolor is a common opportunistic superficial infection of the skin caused by the ubiquitous yeast *Malassezia furfur.*

Prevalence and Risk Factors

Prevalence is high in hot, humid climates. Purported risk factors include oral contraceptive use, heredity, systemic corticosteroid use, Cushing's disease, immunosuppression, hyperhidrosis, and malnutrition.

Pathophysiology and Natural History

*M. furfur* may filter the rays of the sun and also produces phenolic compounds that inhibit tyrosinase, which can produce hypopigmentation in many patients.

Signs and Symptoms

Infection produces discrete and confluent, fine scaly, well-demarcated, hypopigmented or hyperpigmented plaques on the chest, back, arms, and neck (Fig. 14). Pruritus is mild or absent.

Diagnosis

Diagnosis is by clinical presentation. Potassium hydroxide preparation exhibits short hyphae and spores with a spaghetti-and-meatballs appearance.


**Treatment**

Selenium sulfide shampoo (2.5%) or ketoconazole shampoo is the mainstay of treatment, applied to the affected areas and the scalp daily for 3 to 5 days, then once a month thereafter. Alternatively, a variety of topical antifungal agents, including terbinafine, clotrimazole, or econazole cream, applied twice daily for 6 to 8 weeks, constitute adequate treatment, especially for limited disease. Systemic therapy may be necessary for patients with extensive disease or frequent recurrences, or for whom topical agents have failed. Treatment is summarized in Box 2.

**Box 2: Treatment of Tinea (Pityriasis) Versicolor**

**Mild Disease**

- Selenium sulfide shampoo for 3-5 days, then once monthly thereafter
- Ketoconazole shampoo/cream for 3-5 days, then once monthly thereafter
- Econazole cream bid for 6-8 weeks, then once monthly thereafter

**Extensive or Recurrent Disease**

- Ketoconazole 200 mg PO daily for 7 days
- Itraconazole 200-400 mg PO daily for 3-7 days
- Fluconazole 400 mg PO × 1

**Viral infections**

**Herpes Simplex**

*Definition and Etiology*

Herpes simplex virus (HSV) infection is a painful, self-limited, often recurrent dermatitis, characterized by small grouped vesicles on an erythematous base. Disease is often mucocutaneous. HSV type 1 is usually associated with orofacial disease, and HSV type 2 is usually associated with genital infection.

*Prevalence and Risk Factors*

Eighty-five percent of the population has antibody evidence of HSV type 1 infection. HSV type 2 infection is responsible for 20% to 50% of genital ulcerations in sexually active persons.

*Pathophysiology and Natural History*

Disease follows implantation of the virus via direct contact at mucosal surfaces or on sites of abraded skin. After primary infection, the virus travels to the adjacent dorsal ganglia, where it remains dormant unless it is reactivated by psychological or physical stress, illness, trauma, menses, or sunlight.

*Signs and Symptoms*
Primary infection occurs most often in children, exhibiting vesicles and erosions on reddened buccal mucosa, the palate, tongue, or lips (acute herpetic gingivostomatitis). It is occasionally associated with fever, malaise, myalgias, and cervical adenopathy (Fig. 15). Herpes labialis (fever blisters or cold sores) appears as grouped vesicles on red denuded skin, usually the vermilion border of the lip; infection represents reactivated HSV. Primary genital infection is an erosive dermatitis on the external genitalia that occurs about 7 to 10 days after exposure; intact vesicles are rare. Recurrent genital disease is common (approximately 40% of affected patients). Prodromal symptoms of pain, burning, or itching can precede herpes labialis and genital herpes infections.

**Diagnosis**

Viral culture helps to confirm the diagnosis; direct fluorescent antibody (DFA) is a helpful but less-specific test. Serology is helpful only for primary infection. The Tzanck smear can be helpful in the rapid diagnosis of herpesviruses infections, but it is less sensitive than culture and DFA.

**Treatment**

Acyclovir remains the treatment of choice for HSV infection; newer antivirals, such as famciclovir and valacyclovir, are also effective. For recurrent infection (more than six episodes per year), suppressive treatment is warranted. Primary infection in immunosuppressed patients requires treatment with acyclovir 10 mg/kg every 8 hours for 7 days. Treatment is summarized in Table 6.

**Table 6: Treatment of Herpes Simplex**

<table>
<thead>
<tr>
<th>Indication</th>
<th>Acyclovir</th>
<th>Famciclovir</th>
<th>Valacyclovir</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary HSV</td>
<td>200 mg PO 5×/day or 400 mg PO tid for 10 days</td>
<td>500 mg PO bid or 250 mg PO tid for 7 days</td>
<td>1 g PO bid for 10 days</td>
</tr>
<tr>
<td>Recurrent HSV</td>
<td>400 mg PO tid for 5 days</td>
<td>750 mg PO bid for 1 day</td>
<td>2 g PO bid for 1 day</td>
</tr>
<tr>
<td>Suppression</td>
<td>400 mg PO bid</td>
<td>250 mg PO bid</td>
<td>1 g PO or 500 mg PO qd</td>
</tr>
</tbody>
</table>

HSV, herpes simplex virus.

**Herpes Zoster**

**Definition and Etiology**

Herpes zoster (shingles) is an acute, painful dermatomal dermatitis that affects approximately 10% to 20% of adults, often in the presence of immunosuppression.

**Pathophysiology and Natural History**

During the course of varicella, the virus travels from the skin and mucosal surfaces to the sensory ganglia, where it lies dormant for a patient’s lifetime. Reactivation often follows immunosuppression, emotional stress, trauma, and irradiation or surgical manipulation of the spine, producing a dermatomal dermatitis.

**Signs and Symptoms**

Herpes zoster is primarily a disease of adults and typically begins with pain and paresthesia in a dermatomal or bandlike
pattern followed by grouped vesicles within the dermatome several days later (Fig. 16). Occasionally, fever and malaise occur. The thoracic area accounts for more than half of all reported cases. When zoster involves the tip and side of the nose (cranial nerve V) nasociliary nerve involvement can occur (30%-40%). Most patients with zoster do well with only symptomatic treatment, but postherpetic neuralgia (continued dyesthesias and pain after resolution of skin disease) is common in the elderly. Disseminated zoster is uncommon and occurs primarily in immunocompromised patients.

**Diagnosis**

Diagnosis is by clinical presentation, viral culture, or direct fluorescent antibody.

**Treatment**

Zoster deserves treatment, with rest, analgesics, compresses applied to affected areas, and antiviral therapy, if possible, within 24 to 72 hours of disease onset. Disseminated and ophthalmic zoster warrants treatment with acyclovir 10 mg/kg intravenously every 8 hours for 7 days. Treatment is summarized in Table 7.

**Warts**

**Definition and Etiology**

Warts are common and benign epithelial growths caused by human papillomavirus (HPV).

**Prevalence and Risk Factors**

Warts affect approximately 10% of the population. Anogenital warts are a sexually transmitted infection, and partners can transfer the virus with high efficiency. Immunosuppressed patients are at increased risk for developing persistent HPV infection.

**Pathophysiology and Natural History**

HPV infection follows inoculation of the virus into the epidermis through direct contact, usually facilitated by a break in the skin. Maceration of the skin is an important predisposing factor, as suggested by the increased incidence of plantar warts in swimmers. After inoculation, a wart usually appears within 2 to 9 months. The rough surface of a wart can disrupt adjacent skin and enable inoculation of virus into adjacent sites, leading to the development and spread of new warts.

**Signs and Symptoms**

The common wart is the most common type: It is a hyperkeratotic, flesh-colored papule or plaque studded with small black dots (thrombosed capillaries) (Fig. 17). Other types of warts include flat warts (verruca plana), plantar warts, and condyloma acuminatum (venereal warts).

**Diagnosis**

The clinical appearance alone should suggest the diagnosis. Skin biopsy may be performed, if warranted.
Treatment

Therapy is variable and often challenging. Most modalities are destructive: cryosurgery, electrodesiccation, curettage, and application of various topical products such as trichloroacetic acid, salicylic acid, topical 5-fluorouracil, podophyllin, and canthacur. For stubborn warts, laser therapy or injection with candida antigen may be helpful. The immunomodulator imiquimod cream (Aldara) is a novel topical agent recently approved for treating condyloma acuminatum, and it might help with common warts as well, usually as adjunctive therapy. Sexual partners of patients with condyloma warrant examination, and women require gynecologic examination. Treatment is summarized in Box 3.

Box 3: Treatment of Warts

Destructive Methods

- Cryosurgery*
- Electrodesiccation
- Curettage
- Laser therapy

Chemotherapeutic Agents

- Podophyllin
- Canthacur
- 5-fluorouracil

Caustics and Acids

- Salicylic acid*
- Trichloracetic acid

Immunotherapies

- Imiquimod
- Candida antigen

*First line therapy

Prevention and Screening

For common warts, no approaches have been documented to prevent transmission. For genital warts (condyloma), the risk correlates with the number of sexual partners. A quadrivalent HPV vaccine (Gardasil) has been available since 2006, and this represents the newest approach to preventing genital HPV infection and ultimately cervical cancer in women. The vaccine is safe and 100% effective and is recommended for girls and women ages 9 to 26 years.

Molluscum Contagiosum

Definition and Etiology
Molluscum contagiosum is an infectious viral disease of the skin caused by the poxvirus.

**Prevalence and Risk Factors**

The prevalence is less than 5% in the United States. Infection is common in children, especially those with atopic dermatitis, sexually active adults, and patients with human immunodeficiency virus (HIV) infection. Transmission can occur via direct skin or mucous membrane contact, or via fomites.

**Pathophysiology and Natural History**

The disease follows direct contact with the virus, which replicates in the cytoplasm of cells and induces hyperplasia.

**Signs and Symptoms**

Molluscum are smooth pink, or flesh-colored, dome-shaped, umbilicated papules with a central keratotic plug (Fig. 18). Most patients have many papules, often in intertriginous sites, such as the axillae, popliteal fossae, and groin. They usually resolve spontaneously, but they often persist in immunocompromised patients.

**Diagnosis**

Diagnosis is by clinical presentation and by skin biopsy, if warranted.

**Treatment**

Treatment might not be necessary because the disease often resolves spontaneously in children. Treatment is comparable to the modalities outlined for warts; cryosurgery and curettage are perhaps the easiest and most definitive approaches. In children, canthacur, applied topically then washed off 2 to 6 hours later, is well tolerated, and is very effective.

**Summary**

- Impetigo is a superficial skin infection usually caused by *Staphylococcus aureus* and occasionally by *Streptococcus pyogenes*.
- Folliculitis is a superficial infection of the hair follicles characterized by erythematous, follicular-based papules and pustules.
- Ecthyma is a deep infection of the skin that resembles impetigo. Ecthyma is somewhat common in patients with poor hygiene or malnutrition.
- Erysipelas is a superficial streptococcal infection of the skin.
- Necrotizing fasciitis is a rare infection of the subcutaneous tissues and fascia that eventually leads to necrosis.
- Dermatophytosis implies infection with fungi, organisms with high affinity for keratinized tissue, such as the skin, nails, and hair. *Trichophyton rubrum* is the most common dermatophyte worldwide.
- Cutaneous candidiasis is a yeast infection caused primarily by *Candida albicans*.
- Tinea versicolor is a common superficial infection of the skin caused by the ubiquitous yeast *Malassezia furfur*.
- Herpes simplex virus infection is a painful, self-limited, often recurrent dermatitis, characterized by small grouped vesicles on an erythematous base.
- Herpes zoster (shingles) is an acute, painful dermatomal dermatitis that affects approximately 10% to 20% of adults, often in the presence of immunosuppression.
- Warts are common and benign epithelial growths caused by human papillomavirus.
Molluscum contagiosum is an infectious viral disease caused by the poxvirus.

References


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