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Skin and Soft-tissue Infections

Skin and soft-tissue infections (SSTIs) are very common in children, and during the past decade the incidence has been increasing dramatically, especially infections secondary to methicillin-resistant Staphylococcus aureus (MRSA). Every clinician commonly encounters cellulitis, abscesses, and lymphadenitis. It is important to have a clear understanding of the most likely etiologic agent and appropriate antibiotic therapy. Ultrasound has changed the approach to soft-tissue infections, enabling differentiation of cellulitis from an abscess and recognition of retained foreign bodies. The authors review common SSTIs, pathophysiology, clinical presentation, and management, with an emphasis on current standards and optimal therapy.

— Ann M. Dietrich, MD, Editor

Epidemiology

Skin and soft-tissue infections (SSTIs) are common in children, resulting in numerous emergency and primary care visits. Skin infections range from superficial infections, such as impetigo, to more serious infections involving subcutaneous tissue, including cellulitis and skin abscesses.

The incidence of SSTIs has increased in the past decade, and this increase has paralleled the rising rates of methicillin-resistant *Staphylococcus aureus* (MRSA) infections.¹ In particular, the incidence of skin and soft-tissue abscesses is increasing.² *S. aureus* is the most commonly identified cause of SSTI,³⁻⁵ with MRSA becoming increasingly widespread, which has presented additional challenges for the management of infection.

Skin and soft-tissue infections occur in children of all ages; however, impetigo is most commonly found in children younger than 5-6 years of age. While abscesses can occur at any age, infants and toddlers manifest an increased incidence of these infections. Skin infections are more likely to occur in the summer and in warm climates.

Skin and soft-tissue infections in children typically have a favorable prognosis; however, significant morbidity and occasional mortality can occur in complicated cases or in children with immunodeficiency or other risk factors.

Pathophysiology

Skin and soft-tissue infections result from the penetration of bacteria through the skin barrier, often at the site of pre-existing skin breakdown or trauma. Alternatively, cellulitis can develop from hematogenous dissemination of a bloodstream infection, as is typical of facial cellulitis caused by *Streptococcus pneumoniae* or *Haemophilus influenzae*.

The dermis is a superficial layer of skin and functions as a barrier against bacterial pathogens. The dermis is involved in infections such as impetigo and erysipelas. Beneath the dermis lies subcutaneous tissue comprised of fat and connective tissue. Abscesses and cellulitis frequently involve subcutaneous tissue. Fascia separates subcutaneous tissue from underlying muscle.

Executive Summary

- CA-MRSA is now the most common pathogen in skin abscesses, occurring in more than 50% of abscesses.
- Specific exposures can introduce unusual pathogens, such as *Pseudomonas folliculitis* from hot tubs; *Mycobacterium* infections from contaminated nail salons; *Pasteurella multocida* and *Capnocytophaga canimorsus* from cat and dog bites; *Eikenella corrodens* from human bites; *Aeromonas hydrophila*, *Vibrio vulnificus*, and *Mycobacterium marinum* from aquatic exposure; and *Clostridium* from penetrating trauma.
- Children with atopic dermatitis can develop an impetiginous secondary infection, manifesting as crusting, weeping, fissuring, and/or pustules in affected areas.

The skin is colonized with normal flora, of which coagulase-negative staphylococci predominate. The normal flora help to prevent colonization with pathogenic bacteria, such as *S. aureus*. When the skin integrity is compromised, bacteria that may be transiently present on skin (*S. aureus* and group A Streptococcus) can gain entry and cause infection. Children with atopic dermatitis are frequently colonized with *S. aureus* on the skin due to the persistent disruption of the skin barrier.⁶ Group A Streptococcus may colonize the oropharynx; likewise, *S. aureus* can colonize the nasal or rectal mucosa, and serve as a reservoir for these pathogenic bacteria.

Risk factors for SSTI include both host and bacterial pathogenic factors. Colonization with MRSA, particularly rectal colonization, is a risk factor for recurrent MRSA abscesses.^{7,8} However, the process by which MRSA colonization causes disease is unclear, as a majority of colonized children do not develop MRSA infection. Skin conditions such as eczema predispose children to infection through compromised skin barriers. Other skin infections can become secondarily infected, such as varicella zoster or tinea pedis. Certain types of immunodeficiency leave affected children susceptible to SSTIs, which can become invasive. (See Table 1.)

Etiology

Staphylococcus. The etiology for SSTIs is not always possible to determine, as the isolation of an organism is more difficult in non-purulent infections such as cellulitis. When

Table 1. Epidemiology and Risk Factors for SSTIs

Infection	Risk Factors
Impetigo	Young children, eczema
Folliculitis	Moist skin, shaving, hot tub exposure
Abscess	All ages, common in toddlers MRSA carrier is risk
Cellulitis	Breaks in skin
Erysipelas	Breaks in skin
Paronychia	Nail biting
Deep tissue infections	Impaired immunity

an organism is able to be isolated, *S. aureus* is the most frequently identified pathogen.³⁻⁵ *S. aureus* has been implicated in various types of infections such as furuncles, abscesses, cellulitis, deep soft-tissue infection, and other invasive infections.

The prevalence of community-associated MRSA (CA-MRSA) continues to rise,^{1,9} although hospital-acquired MRSA cases appear to be decreasing.¹⁰ CA-MRSA is now the most common pathogen in skin abscesses, occurring in more than 50% of abscesses.^{3,5,11-13} CA-MRSA has a greater tendency to cause abscesses than methicillin-sensitive *S. aureus* (MSSA).¹¹

Coagulase-negative staphylococcus can occasionally cause folliculitis, and may be present in polymicrobial abscesses, along with more pathogenic bacteria such as *S. aureus*.

Streptococcus. Streptococcus species are also important skin and soft-tissue pathogens and remain a major cause of non-purulent

infections such as cellulitis and erysipelas.¹⁴ Group A Streptococcus is a frequent skin and soft-tissue pathogen; however, other beta-hemolytic streptococci (Groups B, C, F, G) are implicated also. Erysipelas is classically a group A Streptococcus infection, as is perianal dermatitis. Facial cellulitis can be caused by *S. pneumoniae* or *Haemophilus influenzae*, although their prevalence has decreased with the use of vaccinations. In neonates, group B Streptococcus should always be considered as a potential pathogen.

Other Bacterial Pathogens.

While the majority of bacterial skin infections are caused by Staphylococcus and Streptococcus, numerous other bacteria can cause SSTIs. Abscesses can be polymicrobial, particularly in the perioral or perirectal areas. Gram-negative bacteria and anaerobes are often present in abscesses in the perirectal or perineal areas. Paronychia infections often involve oral flora.

Specific exposures can introduce unusual pathogens, such as *Pseudomonas folliculitis* from hot tubs; *Mycobacterium* infections from contaminated nail salons; *Pasteurella multocida* and *Capnocytophaga canimorsus* from cat and dog bites; *Eikenella corrodens* from human bites; *Aeromonas hydrophila*, *Vibrio vulnificus*, and *Mycobacterium marinum* from aquatic exposure; and *Clostridium* from penetrating trauma. Infections in immunocompromised patients can involve other pathogens, including *Pseudomonas*.

Clinical Features

Children with SSTIs typically present with a painful skin lesion; however, the immediate reason for seeking medical attention is sometimes fever or systemic symptoms. When approaching a child with a suspected SSTI, important historical features to elicit include systemic symptoms such as fever, lethargy, irritability, or decreased oral intake; onset and progression of skin lesion(s); recent antibiotic use; past medical history, including prior skin infections; and risk factors for MRSA, including prior infection, household contacts, or health care utilization, particularly hospitalizations or chronic care facilities.

The physical exam should focus on the general appearance and overall evaluation of the child, in addition to the skin exam. While systemic involvement is infrequent with SSTIs, fever and tachycardia can be signs of systemic infection.

For any SSTI, attention should also be directed to looking for complications of the infection. Lymphangitis appears as red streaking progressing from the site of the skin infection, and can spread to regional lymph nodes. Lymphadenitis can be found with SSTIs, and nodes should be assessed for tenderness or fluctuance suggesting infection. SSTI near a joint should prompt joint examination looking for signs of septic arthritis, such as swelling, pain, and decreased range of motion. Extensive spread of infection, ecchymosis or skin

Table 2. Clinical Features of SSTIs

Infection	Clinical Features
Impetigo	Bullous: Superficial “honey crusted” lesions on an erythematous base Non-bullous: Bullae filled with clear or purulent fluid
Folliculitis	Multiple small, erythematous papules or pustules around hair follicles Often in moist areas, buttocks, shaved sites, extremities, and scalp
Furuncle	Purulent collection around hair follicle
Abscess	Tender, nodular, erythematous lesions Often on buttock, thigh, axillae All ages; common in toddlers
Cellulitis	Warm, tender, erythematous, and indurated skin The area of erythema and induration is less clearly demarcated than in erysipelas
Erysipelas	Skin erythema, warmth, and tenderness, sharply demarcated margins; skin is raised and shiny Often in lower extremity after cuts
Lymphadenitis	Tender, enlarged, fluctuant lymph node, often following skin trauma
Paronychia	Infection around nail bed — purulence, swelling, tenderness

necrosis, crepitance, pain out of proportion to the extent of visible infection, pain with passive muscle stretch, or a toxic-appearing child are all signs of a possible deep tissue infection.

Types of Skin Infection

Impetigo. Impetigo is a superficial skin infection, often at a site of trauma that has disrupted the skin barrier. Impetigo is distinguished by “honey crusted” lesions on an erythematous base, typically with little pain or surrounding erythema. Lymphadenopathy may be present, but systemic signs are not

usually seen. Impetigo can be bullous or non-bullous. Non-bullous impetigo, which is more common, is most frequently caused by *Staphylococcus aureus*, or less commonly by *Streptococcus pyogenes* in older children. Lesions are often located on the face. Bullous impetigo is characterized by bullae filled with clear or purulent fluid, which result from local production of toxin by *S. aureus*. Bullous impetigo is more common on the trunk. Another variation of superficial staphylococcal infection is pustulosis, consisting of small pustules, frequently in the diaper area. Impetigo can progress to

Table 3. Differential Diagnosis for Common SSTIs

Infection	Differential
Impetigo	Herpes simplex Burn Scabies Tinea pedis
Cellulitis	Contact dermatitis Insect bite reaction DVT Myofasciitis
Abscess	Cyst Myiasis
Paronychia	Herpetic whitlow

cellulitis, although most cases resolve uneventfully within two weeks with treatment.

Children with atopic dermatitis can develop an impetiginous secondary infection, manifesting as crusting, weeping, fissuring, and/or pustules in affected areas.

Erysipelas. Erysipelas is a bacterial infection of the dermis. Like cellulitis, the affected area is erythematous, warm, and tender; however, it can be distinguished by sharply demarcated margins and the affected area will be raised and shiny. Erysipelas also involves superficial lymphatics and can spread, causing lymphangitis. Fever and systemic symptoms are frequent and onset is acute. This infection occurs most frequently on the lower extremities.

Cellulitis. Cellulitis can be similar in appearance to erysipelas, but involves the entire dermis and subcutaneous fat. On exam, the area will appear warm, tender, erythematous, and indurated. The area of erythema and induration is less clearly demarcated than in erysipelas. Systemic signs are usually not present early in the course. Potential etiologies include beta-hemolytic *Streptococcus* (particularly *Streptococcus pyogenes*) as well as *Staphylococcus aureus*. The presence of abscess should be excluded, and the physician should distinguish between purulent cellulitis and non-purulent cellulitis.¹⁵

Folliculitis. Folliculitis is a superficial infection around hair follicles characterized by multiple small erythematous papules or pustules. Frequently involved areas include moist areas such as the buttocks, shaved sites, extremities, and the scalp.

Cutaneous Abscesses. Cutaneous abscesses are localized collections of purulent fluid within the dermis and/or subcutaneous tissue. Small abscesses of hair follicles are termed furuncles, and carbuncles are confluences of infected hair follicles. Abscesses appear as tender, nodular, erythematous lesions. Abscesses are frequently located on the buttock,¹⁶ thigh, or axilla.¹⁷ Detection of an abscess within surrounding cellulitis can be difficult, as both can present with erythema, warmth, tenderness, and induration. In this setting, identification of an abscess is essential so that the fluid collection can be drained. The presence of fluctuance or purulent drainage suggests an abscess, although detection of fluctuance can be difficult.¹⁷ Ultrasound is a valuable adjunct for the delineation of fluid collections.

Paronychia. Paronychia is inflammation or infection of the lateral nail fold, resulting from trauma such as nail biting. The nail fold will appear erythematous, swollen, warm, and tender.

See Table 2 for a summary of the common clinical features for SSTIs.

Differential Diagnosis

A number of conditions can have an appearance similar to bacterial SSTIs. Vesicular or crusting lesions can resemble impetigo, including viral infections (herpes simplex and varicella zoster), burns, scabies, or fungal infections such as tinea pedis. These conditions can also develop secondary impetigo. Contact dermatitis can resemble impetigo, with bullae, erythema, and crusting; however, pruritis will be present and is a distinguishing feature.

The differential diagnosis for cellulitis includes other conditions causing swelling and erythema, such as venous thrombosis, an insect sting, and insect bite hypersensitivity. Reactions to insect stings and bites begin rapidly and are often accompanied by pruritis, whereas cellulitis typically takes 24-48 hours to develop. Cellulitis should be distinguished from fasciitis and deep tissue infection.

Infection of an existing cyst (e.g., epidermal inclusion cyst) presents like an abscess. In returning travelers, myiasis should be considered in the differential for skin lesions. (See Table 3.)

Diagnostic Studies

Wound Cultures. Skin infection with purulent drainage should be cultured if antibiotics will be used. Culture is becoming increasingly critical in this era of CA-MRSA as resistance increases to antibiotic agents, including clindamycin.^{9,13} Needle aspiration or a swab of purulent drainage should be sent for culture in high-risk patients or situations in which the diagnosis is unclear to guide antibiotic management. Aspiration of cellulitis has a low yield, but should be considered if the infection has not responded to appropriate empiric treatment or the patient has significant underlying medical conditions.⁴ Aspirate taken from the area of greatest inflammation rather than the leading edge provides a better yield.

Blood Work. Laboratory testing is not routinely indicated for SSTIs; however, testing may be indicated

if bacteremia is suspected, as in the case of an ill-appearing child. Blood culture should be performed whenever bacteremia is suspected. CBC and inflammatory markers (ESR and CRP) may be helpful when complicated infection is suspected, for example deep tissue infection, septic arthritis, or osteomyelitis. An elevated white blood cell count (WBC) with left shift in a febrile child has some predictive value for bacteremia.¹⁸ Cellulitis of the face (e.g., periorbital or buccal) has a high rate of associated bacteremia.

Imaging. Ultrasound has been shown to be a useful tool in detecting drainable fluid collections. In a recent study, bedside ultrasound showed a higher sensitivity and specificity for detecting a drainable fluid collection than clinical judgment alone.¹⁹ (See Figures 1 and 2.) Ultrasound can also help to identify foreign bodies, which might not be visible upon radiography. (See Figures 3 and 4.) Ultrasound may also be used to evaluate lymph nodes and assist with early diagnosis of lymphadenitis and recognition of an abscess within a lymph node. (See Figure 5.)

Plain radiographs are not routinely indicated. Plain radiographs cannot exclude deep infections such as fasciitis, and their use should not delay surgical evaluation and antibiotic treatment if deep tissue infection is suspected on physical exam.

Management

Folliculitis can be treated with warm compresses applied several times a day. If folliculitis fails to resolve spontaneously or with application of warm compresses and attention to proper hygiene, topical antibiotics should be used. For non-pseudomonal folliculitis, use topical mupirocin 2% TID for 7 to 10 days. A small furuncle can also be adequately treated by application of warm compresses to promote drainage.²⁰

Impetigo, both bullous and non-bullous, can be treated with topical antibiotics. Topical antibiotics improve cure rates over skin

Figure 1. Cellulitis

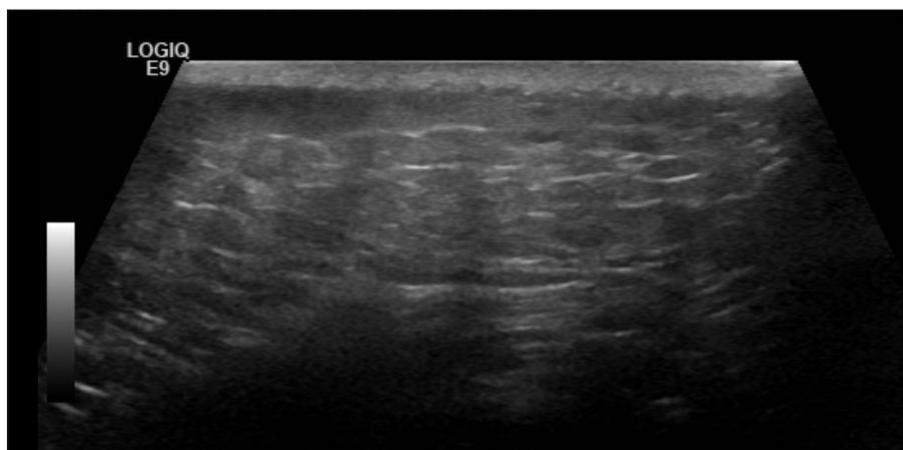
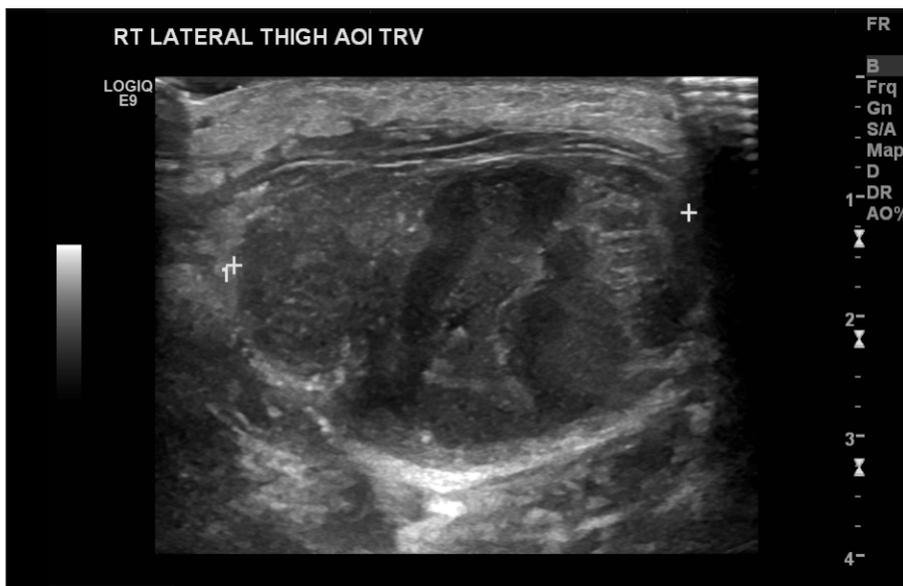


Figure 2. Thigh Abscess



cleansing. Uncomplicated impetigo should be treated with topical mupirocin 2% TID for 7 to 10 days.²¹ In the case of extensive impetigo, or if underlying cellulitis is present, oral antibiotics should be given.

Cellulitis and erysipelas infections can typically be treated with outpatient oral antibiotics. Erysipelas can be treated with penicillin or amoxicillin; however, if the infection cannot be distinguished from cellulitis, the antibiotic choice should also cover *Staphylococcus aureus*.

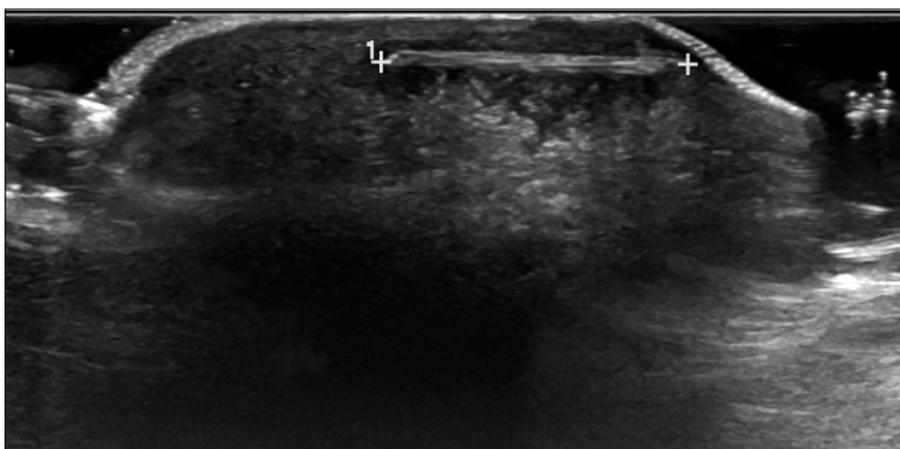
Non-purulent cellulitis is frequently caused by beta-hemolytic

Streptococcus. Treatment should include coverage for beta-hemolytic Streptococcus and MSSA, such as with a first-generation cephalosporin (cephalexin). Empiric treatment for CA-MRSA should be considered as well, as many cases of cellulitis are now caused by CA-MRSA.⁴ Clindamycin is a good first-line choice for cellulitis due to adequate coverage of all common pathogens: MRSA, MSSA, and beta-hemolytic Streptococcus. In geographic areas with a low prevalence of CA-MRSA, cephalexin alone may still be an effective course of treatment.

Figure 3. Abscess Foreign Body



Figure 4. Foreign Body Foot



Purulent cellulitis should be treated for suspected MRSA with clindamycin or trimethoprim-sulfamethoxazole (TMP-SMX); coverage for beta-hemolytic *Streptococcus* is not necessary.¹⁵ Treatment for 7 to 10 days is usually adequate.

Paronychia. Small superficial infections can be treated with warm compresses. Incision and drainage and oral antibiotics should be used for deep lesions/paronychia abscesses. Amoxicillin/clavulanic acid or clindamycin should be used for coverage of anaerobic oral flora.²¹

Abscess. Primary treatment for a skin/soft-tissue abscess is incision and drainage.¹⁵ Controversy exists over the use of packing and oral antibiotics after incision and drainage. In adults, a randomized trial has shown that incision and drainage without packing does not increase the risk of treatment failure.²² Only one small randomized trial of packing versus no packing has been conducted in children. It concluded that packing is not necessary in superficial skin abscesses.²³

Several recent pediatric studies have evaluated the use of antibiotics

for abscesses in addition to incision and drainage. In adults, studies have shown that most abscesses will resolve without antibiotic treatment,^{3,24,25} although retrospective studies did show a small benefit of antibiotic therapy.^{26,27} Antibiotics may prevent the development of new lesions in the short term.²⁵ In children, use of TMP-SMX prevented development of new lesions but did not improve outcomes for abscesses treated by incision and drainage.¹⁶ Two other pediatric studies saw favorable results with incision and drainage, even when the prescribed antibiotic was not effective against the isolated organism.^{28,29}

Antibiotics are indicated if an abscess cannot be adequately drained, multiple concurrent lesions are present, significant cellulitis is present (e.g., > 5 cm), or when the child is febrile.¹⁵ Antibiotics should also be considered for children younger than 1 year of age; there have been higher treatment failure rates without antibiotic use.²⁸ A 7- to 10-day course is standard, although a 5-day course has been effective in adults.¹⁵

Incision and drainage of an abscess in a child may require sedation. After application of a topical anesthetic and infiltration with lidocaine, the abscess should be incised with a #11 blade along the length of the abscess following skin lines. Smaller incisions can be used for cosmetic reasons. Use a hemostat to break up loculations, then irrigate with saline. If warranted, pack the abscess. Contraindications to incision and drainage include abscesses in the middle third of the face (due to the risk for septic thrombosis) or paronychia associated with herpetic whitlow.¹⁸

A potential alternative to incision is the application of anesthetic cream to aid in abscess drainage. A significant number of abscesses will spontaneously drain after topical application of lidocaine cream covered with an occlusive dressing.³⁰ In a trial of this method, several patients did not require further intervention after spontaneous

Figure 5. Lymphadenitis



Table 4. Antibiotics (from Red Book 2012 and IDSA Guidelines) 7-10 Days

Oral Antibiotics	Dose (mg/kg/day)	Adult/max dose (per day)	Frequency	MSSA	MRSA	Group A Strep
Clindamycin	30-40	0.9-1.8 g	Q8 hours	+	+	++ res 1-2% (Red Book p. 676)
Doxycycline (age ≥ 8 years)	2-4	200 mg	Q12-q day	+	+	-
Trimethoprim-Sulfamethoxazole**	8-12 tmp	320 mg tmp	Q12 hours	++	++	-
Dicloxacillin	12-25	0.5-1 g	Q6 hours	++	-	++
Cephalexin	25-50	1-2 g	Q6 or q12 hours	++	-	++
Amoxicillin-clavulanate (7:1)*	25-45 amox	1750 mg	Q12	++	-	++

++ good coverage
 + some resistance reported, prevalence may vary by community
 - no coverage
 * Recommended for treatment of infection associated with human or animal bite wounds. Covers *Eikenella* and anaerobes. Red Book p. 206.
 **Trimethoprim-sulfamethoxazole is not FDA approved for treatment of *Staphylococcus aureus* infections.

abscess drainage, and fewer patients required sedation. However, this study did not look at treatment failure rates.³⁰ Further study is needed to determine if expression of a spontaneously draining abscess is comparable to incision and drainage.

Oral Antibiotics. Penicillinase-resistant penicillins (e.g.,

dicloxacillin) and first-generation cephalosporins (cephalexin) cover *S. pyogenes* and MSSA for impetigo and cellulitis; however, they should not be used as a first-line agent for SSTI if the community prevalence of MRSA is significant. Dosing for either medication in 25 mg/kg/day PO divided q6 hours.²⁰

Clindamycin has been studied in children and is effective for treatment of SSTI^{31,32} and invasive staphylococcal infections.¹⁵ If community resistance to clindamycin is low, clindamycin is a good first-line agent for cellulitis, with good coverage of Streptococcus and most MRSA and MSSA. Resistance is increasing,

however.^{9,13} Soft-tissue and abscess penetration is excellent. Oral dosing for clindamycin is 30 mg/kg/day divided TID.¹⁵ One limitation in the use of clindamycin is its poor palatability.³³ The most common side effect is diarrhea, and clindamycin has been associated with development of *Clostridium difficile* colitis.³⁴

TMP-SMX is an appealing option for treatment of MRSA-related SSTIs, as nearly all isolates are susceptible in vitro. TMP-SMX is not FDA approved for treatment of *S. aureus* infections; however, it has been used successfully in adults for cellulitis and other SSTIs.^{27,35,36} Its efficacy in clinical practice is not well established in children. A retrospective study showed slightly higher failure rates for TMP-SMX-treated SSTI than clindamycin.³⁷ TMP-SMX should not be used alone for non-purulent cellulitis, as it lacks streptococcal coverage, but may be used in combination with a beta-lactam antibiotic. Common side effects include nausea. Although rare, Stevens Johnson syndrome can occur with TMP-SMX use. Patients with G6PD deficiency should not be prescribed TMP-SMX.

Tetracyclines such as doxycycline are effective for uncomplicated SSTIs. Doxycycline can be used to treat SSTIs caused by CA-MRSA, although inducible resistance has been reported. Tetracyclines should not be used in children younger than the age of 8 years due to the possibility of tooth enamel discoloration.

Linezolid is effective for treating MRSA SSTIs, but is rarely used as a first-line agent due to its high cost. Oral availability is 100%. Dosing is 600 mg PO BID for patients who are 12 years of age or older, or 30 mg/kg/day divided q8 hours for patients younger than 12 years of age.¹⁵

Hygiene. Patients should be instructed on proper hygiene measures to prevent the spread of MRSA and other infections. Infected draining areas should be kept covered with clean dressings, and hand washing after contact with the infected area should be encouraged. To

prevent spread among members of a household, patients should not share items such as towels, and surfaces in the home that contact skin frequently should be cleaned. Prophylactic antibiotics are not indicated for family members; antibiotics should only be used for an active infection.¹⁵

Complications

Bacteremia. Aside from facial cellulitis with *Streptococcus pneumoniae* or *Haemophilus influenzae*, which are almost always associated with bacteremia, the incidence of bacteremia secondary to cellulitis or cutaneous abscess is low. Fasciitis is often associated with bacteremia.

Lymphatic Involvement. SSTIs, particularly cellulitis, can spread via lymphatics. Lymphangitis is recognized by streaking erythema from the area of infection, and often progresses rapidly. Infection of local lymph nodes can occur without evidence of lymphangitis. Treatment is with IV antibiotics.

Deep Tissue Infections. Cellulitis can progress to osteomyelitis, septic arthritis, and necrotizing fasciitis.²¹ Necrotizing soft-tissue infection is characterized by rapid progression of infection with tissue necrosis, and can involve subcutaneous tissue and superficial fascia, or deep fascia and muscle. Immediate surgical intervention and antibiotic therapy is necessary to control the infection and remove necrotic tissue. Immunocompromised children are at higher risk for necrotizing infections; however, these infections can also develop in normal hosts following skin trauma or varicella infection.

Venous Thrombosis. Thrombophlebitis can complicate cellulitis, particularly if other risk factors for thrombosis are present. A rare complication of SSTI of the central face is cavernous sinus thrombosis, which presents with orbital pain, proptosis, and oculomotor nerve palsies.

Toxic Shock Syndrome/Toxin-mediated Disease. Staphylococcus and Streptococcus can both produce

exotoxins, which can cause a spectrum of illness ranging from rash alone to shock. Patients are usually febrile, have a diffuse erythrodermal rash, and may have multi-organ system dysfunction/failure, including circulatory collapse. Often the focus of infection is not identified; however, toxic shock syndrome can result from an identified SSTI. Treatment is with antibiotics and supportive therapy in an appropriate inpatient setting.

Post-streptococcal Glomerulonephritis. Certain strains of *Streptococcus pyogenes* can lead to glomerulonephritis, which presents an average of 2-3 weeks after skin infection. Antibiotics do not prevent post-streptococcal glomerulonephritis. Skin infection with *S. pyogenes* does not cause rheumatic fever.

Disposition

Indications for Admission. Most infections can be managed as an outpatient unless bacteremia or deep tissue infection is suspected. Children younger than 1 year of age or with a compromised immune system should be considered for admission unless treating a superficial skin infection.¹⁸ Signs of infection warranting admission include an ill- or toxic-appearing child, fever greater than 39°C with leukocytosis or extensive infection, and facial cellulitis with fever.¹⁸ Lymphangitis or rapid spread of infection are also indications for admission.

Parenteral antibiotic selection depends on the extent of infection. If bacteremia is suspected, vancomycin should be used. For children without bacteremia, appropriate choices include clindamycin 40 mg/kg/day divided q6-8 hours or linezolid 600 mg IV/PO BID for patients 12 years of age or older, or 30 mg/kg/day divided q8 hours for patients younger than 12 years of age.¹⁵

Neonates. Neonates require hospitalization and IV antibiotics for most SSTIs. Omphalitis and mastitis can occur in the first few weeks of life. Antibiotic therapy should cover *S. aureus*, *S. pyogenes*, and group B streptococci for neonatal SSTIs.

Assessing Therapeutic Response. Follow-up at approximately 48 hours is appropriate to assess the response to initial therapy. If incision and drainage was performed, packing should be removed and replaced if necessary. Persistent fluid collections warrant drainage, and antibiotics should be considered. Most abscesses heal within 1-2 weeks. SSTIs such as cellulitis treated with appropriate antimicrobial therapy should show signs of improvement within 24-48 hours. If cellulitis has not improved or has progressed, the antibiotic choice should be re-evaluated. If an antibiotic lacking activity against CA-MRSA was used initially, it should be changed to an antibiotic effective against CA-MRSA, such as clindamycin, TMP-SMX, or linezolid. Culture data and susceptibilities should be used to select an appropriate antibiotic whenever available.

Prognosis

Uncomplicated SSTIs in children have a good prognosis. However, recurrent infections are problematic, particularly in children colonized with CA-MRSA. Decolonization regimens involving dilute bleach baths and nasal mupirocin have been shown to have benefits for some patients with recurrent MRSA infections.¹⁵

Summary

Skin and soft-tissue infections are common in children. Diagnosis can typically be made on physical exam alone. Primary treatment includes topical antibiotics for superficial infection, oral antibiotic for cellulitis, and incision and drainage for abscesses, resulting in resolution without sequelae in the majority of cases.

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pediatric skin and soft-tissue infections. *Pediatrics* 2011;128(3):e479-e487.

CME Questions

1. Which pathogen is now the most common cause of skin abscesses?
 - A. coagulase-negative *S. aureus*
 - B. methicillin-sensitive *S. aureus*
 - C. methicillin-resistant *S. aureus*
 - D. *S. pyogenes*
2. A 10-month-old has a 3 cm erythematous patch on the upper thigh with several large, flaccid bullae. The most likely diagnosis is:
 - A. contact dermatitis
 - B. impetigo
 - C. herpes simplex
 - D. varicella
3. A child presents with erythema and swelling after an insect sting. What would be most suggestive of cellulitis rather than a local reaction?
 - A. erythema, warmth, and swelling
 - B. pruritus
 - C. swelling started within hours of the sting and persisted for 48 hours
 - D. onset of swelling started more than 24 hours after sting
4. Which of the following is *not true* regarding the use of imaging studies for skin and soft-tissue infections?
 - A. Ultrasound can be useful in detecting drainable fluid collections/abscesses.
 - B. Use of plain radiographs should not delay surgical intervention for patients with suspected deep tissue infection.
 - C. Ultrasound can identify foreign bodies that may not be visible on plain radiographs.
 - D. Plain radiographs easily and reliably identify necrotizing fasciitis.
5. Which of the following is a contraindication for incision and drainage of an abscess?
 - A. child will not cooperate
 - B. paronychia infection associated with herpetic whitlow
 - C. multiple abscesses
 - D. large abscess
6. A 10-year-old female with no PMH presents with a 4 cm abscess on the buttock with no surrounding cellulitis. Which option would be the best initial treatment?
 - A. incision and drainage (with or without packing), no antibiotics, and follow-up in 48 hours
 - B. incision and drainage, discharge with oral clindamycin, no follow-up
 - C. warm compresses and treatment with trimethoprim-sulfamethoxazole
 - D. incision and drainage, packing must be used, follow-up in 48 hours to remove packing

Pediatric Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in pediatric patients presenting to the emergency department;
- describe the epidemiology, etiology, pathophysiology, historical and examination findings associated with conditions in pediatric patients presenting to the emergency department;
- formulate a differential diagnosis and perform necessary diagnostic tests;
- apply up-to-date therapeutic techniques to address conditions discussed in the publication;
- discuss any discharge or follow-up instructions with patients.

CME Instructions

HERE ARE THE STEPS YOU NEED TO TAKE TO EARN CREDIT FOR THIS ACTIVITY:

1. Read and study the activity, using the provided references for further research.
2. Log on to www.cmecity.com to take a post-test; tests can be taken after each issue or collectively at the end of the semester. *First-time users will have to register on the site using the 8-digit subscriber number printed on their mailing label, invoice, or renewal notice.*
3. Pass the online tests with a score of 100%; you will be allowed to answer the questions as many times as needed to achieve a score of 100%.
4. After successfully completing the last test of the semester, your browser will be automatically directed to the activity evaluation form, which you will submit online.
5. **Once the completed evaluation is received, a credit letter will be e-mailed to you instantly.**

7. A patient is seen 48 hours after incision and drainage of a buttock abscess. On exam, the child is afebrile and well-appearing. There is a 1.5 cm area of fluctuance and erythema, with minimal surrounding erythema. The child was discharged on clindamycin and the wound culture grew MRSA sensitive to clindamycin. The best treatment at this time would be:
- continued observation and current antibiotic therapy
 - change antibiotic to trimethoprim-sulfamethoxazole
 - repeat incision and drainage
 - hospital admission for IV antibiotics
8. A 4-year-old female has sharply demarcated erythema extending approximately 5 cm from a small abrasion on her leg. She is afebrile and otherwise healthy. Which antibiotic would be appropriate for initial treatment?
- clindamycin
 - tetracycline
 - doxycycline
 - topical mupirocin
 - vancomycin
9. A previously healthy 3-year-old presents with spreading erythema and induration around the site of an insect bite on the forearm. There is a small pustule at the site and erythematous streaking extending up the arm. Which antibiotic would be appropriate for initial treatment?
- clindamycin PO
 - vancomycin IV
 - ampicillin/sulbactam IV
 - doxycycline
10. An 18-month-old male presents to the ED with a 3 cm abscess on the thigh. The total area of erythema is 4 cm. The patient is febrile to 101°F, but is well-appearing, well hydrated, and has no PMH. After incision and drainage of the abscess, which is an appropriate disposition?
- discharge with trimethoprim-sulfamethoxazole
 - discharge with cephalexin
 - discharge with doxycycline
 - admit and start IV antibiotics
 - discharge without antibiotics

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Pediatric Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Skin and Soft-tissue Infections

Epidemiology and Risk Factors for SSTIs

Infection	Risk Factors
Impetigo	Young children, eczema
Folliculitis	Moist skin, shaving, hot tub exposure
Abscess	All ages, common in toddlers MRSA carrier is risk
Cellulitis	Breaks in skin
Erysipelas	Breaks in skin
Paronychia	Nail biting
Deep tissue infections	Impaired immunity

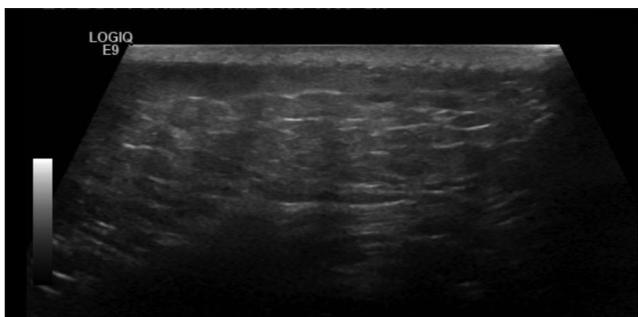
Clinical Features of SSTIs

Infection	Clinical Features
Impetigo	Bullous: Superficial “honey crusted” lesions on an erythematous base Non-bullous: Bullae filled with clear or purulent fluid
Folliculitis	Multiple small, erythematous papules or pustules around hair follicles Often in moist areas, buttocks, shaved sites, extremities, and scalp
Furuncle	Purulent collection around hair follicle
Abscess	Tender, nodular, erythematous lesions Often on buttock, thigh, axillae All ages; common in toddlers
Cellulitis	Warm, tender, erythematous, and indurated skin The area of erythema and induration is less clearly demarcated than in erysipelas
Erysipelas	Skin erythema, warmth, and tenderness, sharply demarcated margins; skin is raised and shiny Often in lower extremity after cuts
Lymphadenitis	Tender, enlarged, fluctuant lymph node, often following skin trauma
Paronychia	Infection around nail bed – purulence, swelling, tenderness

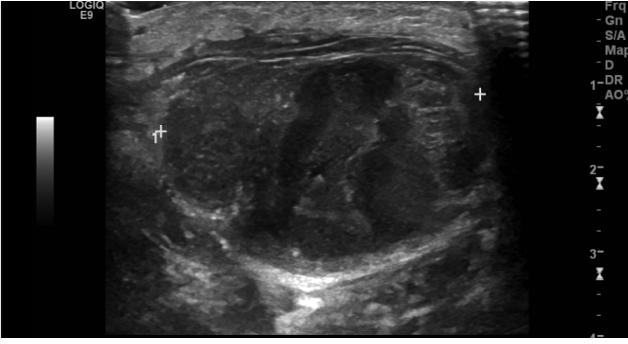
Differential Diagnosis for Common SSTIs

Infection	Differential
Impetigo	Herpes simplex Burn Scabies Tinea pedis
Cellulitis	Contact dermatitis Insect bite reaction DVT Myofasciitis
Abscess	Cyst Myiasis
Paronychia	Herpetic whitlow

Cellulitis



Thigh Abscess



Abscess Foreign Body



Lymphadenitis



Antibiotics (from Red Book 2012 and IDSA Guidelines) 7-10 Days

Oral Antibiotics	Dose (mg/kg/day)	Adult/max dose (per day)	Frequency	MSSA	MRSA	Group A Strep
Clindamycin	30-40	0.9-1.8 g	Q8 hours	+	+	++ res 1-2% (Red Book p. 676)
Doxycycline (age ≥ 8 years)	2-4	200 mg	Q12-q day	+	+	-
Trimethoprim-Sulfamethoxazole**	8-12 tmp	320 mg tmp	Q12 hours	++	++	-
Dicloxacillin	12-25	0.5-1 g	Q6 hours	++	-	++
Cephalexin	25-50	1-2 g	Q6 or q12 hours	++	-	++
Amoxicillin-clavulanate (7:1)*	25-45 amox	1750 mg	Q12	++	-	++

++ good coverage

+ some resistance reported, prevalence may vary by community

- no coverage

* Recommended for treatment of infection associated with human or animal bite wounds. Covers *Eikenella* and anaerobes. Red Book p. 206.

**Trimethoprim-sulfamethoxazole is not FDA approved for treatment of *Staphylococcus aureus* infections.

Supplement to *Pediatric Emergency Medicine Reports*, August 2013: "Skin and Soft-tissue Infections." Authors: Evan J. Weiner, MD, FAAP, FACEP, FAAEM, Interim Director, Department of Emergency Medicine, PEM Fellowship Director, St. Christopher's Hospital for Children, Assistant Professor of Pediatrics and Emergency Medicine, Drexel University College of Medicine, Philadelphia, PA; and Kathryn Kasmire, MD, Pediatrics Resident, St. Christopher's Hospital for Children, Philadelphia, PA. *Pediatric Emergency Medicine Reports' Rapid Access Guidelines*. Copyright © 2013 AHC Media, a division of Thompson Media Group, LLC, Atlanta, GA. **Interim Editorial Director:** Lee Landenberger. **Editor-in-Chief:** Ann Dietrich, MD, FAAP, FACEP. **Executive Editor:** Shelly Morrow Mark. **Managing Editor:** Leslie Hamlin. For customer service, call: 1-800-688-2421. This is an educational publication designed to present scientific information and opinion to health care professionals. It does not provide advice regarding medical diagnosis or treatment for any individual case. Not intended for use by the layman.