SOFT TISSUE INFECTIONS

ANATOMY AND DEFINITIONS:

- Cellulitis → infection of the deep dermis and subcutaneous fat
- Erysipelas → more superficial skin infection, involving upper dermis with PROMINENT LYMPHATIC INVOLVEMENT
- Folliculitis → infection of the hair follicle
- Skin abscesses → collection of pus within dermis and deeper skin tissues
    - Furuncle → single, deep nodules involving the hair follicle
    - Carbuncle → formed by multiple interconnecting furuncles

Table 147-1 Risk Factors for Infections with Methicillin-Resistant *Staphylococcus aureus* (MRSA)
Health care–associated risk factors
Previous antibiotic use, antibiotic use in the last month*
Residence in a long-term care facility
Contact with a health care worker or nursing home resident
Residence in a long-term care facility
Diabetes mellitus
Hospitalization
Admission to an intensive care unit
IV drug use
Invasive indwelling devices
Hemodialysis or peritoneal dialysis
Mechanical ventilation with endotracheal tube or tracheostomy tube
Nasogastric tube
Gastrostomy tube
Foley catheter
Total parenteral nutrition or enteral feeding
Surgical procedures
Immunosuppression
Chronic illness
Previous isolation of MRSA*
Community-acquired risk factors
Children in day care centers
Household contacts with proven community-acquired MRSA
Pacific Islanders
Competitive athletes
Homeless youth
Native Americans
Men who have sex with men
Jail inmates
Military recruits
Report of a suspected spider bite*

CELLULITIS AND ERYSIPELAS:

EPIDEMIOLOGY:

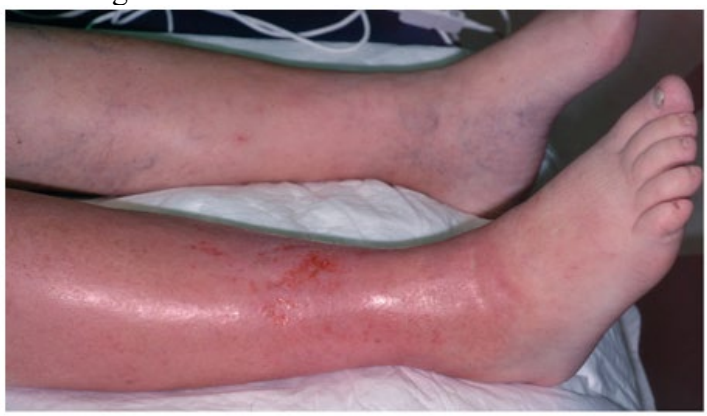
- Risk factors for cellulitis and erysipelas are:
    - Immunocompromise
    - Peripheral vascular disease
    - LYMPHOEDEMA (Odds Ratio 71.2)
    - SKIN BREAKDOWN/SITE OF ENTRY (OR 23.8)
    - VENOUS INSUFFICIENCY (OR 2.9)
    - LEG OEDEMA
    - OBESITY

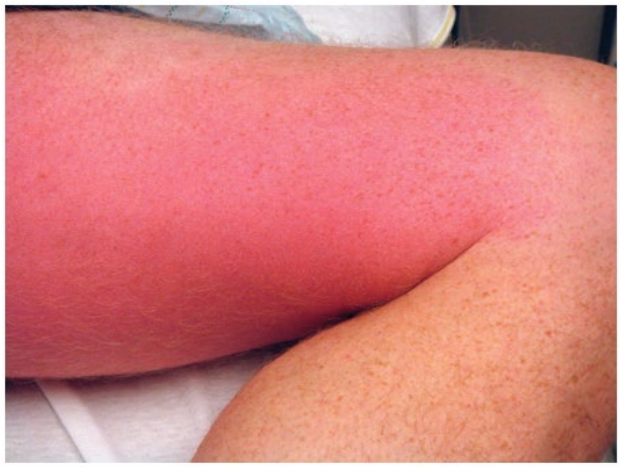
MICROBIOLOGY:

- About 80% of cellulitis cases are caused by GRAM POSITIVE BACTERIA
    - Most common pathogens are Beta-haemolytic streptococcus, Staph aureus and gram-negative aerobic bacilli

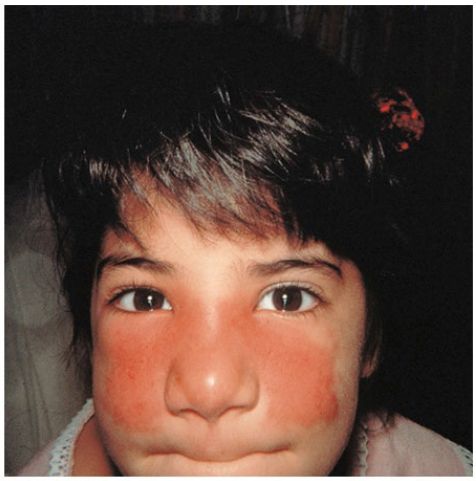
CLINICAL FEATURES:

- CELLULITIS:
    - Affected skin is tender, warm, erythematous and swollen, typically without a sharp demarcation → purely local inflammation is much more common
    - Systemic signs of fever, leukocytosis, and bacteraemia are more typical in the immunosuppressed
    - Recurrent episodes of cellulitis can lead to → impaired lymphatic drainage, permanent swelling, dermal fibrosis and epidermal thickening





- ERYSIPELAS:
    - Onset of symptoms usually abrupt with fever, chills, malaise and nausea representing prodromal phase
    - As infection progresses → affected skin becomes indurated with distinct demarcation and PEAU D'ORANGE skin puckering
    - "Butterfly" pattern over face



- Lymphatic inflammatory changes, known as TOXIC STRIATIONS and local lymphadenopathy
- If purpura, bullae and small areas of necrosis present → search for possible necrotizing soft tissue infection is warranted

- DIAGNOSIS IS CLINICAL:
  - Blood cultures are positive in only 5% cases
  - Despite this, if there are signs of systemic toxicity, extensive skin involvement, underlying comorbidities, recurrent episodes or in circumstances such as bites → culture of pus, bullae or blood are recommended
  - Routine imaging NOT NECESSARY UNLESS OSTEOMYELITIS OR NECROTISING SOFT TISSUE INFECTION IS SUSPECTED

Table 147-3 Differential Diagnosis of Cellulitis and Erysipelas

Infectious Disorders	Noninfectious Disorders
Necrotizing soft tissue infection	Deep vein thrombosis
Herpes zoster	Superficial thrombophlebitis
Bursitis	Insect stings
Osteomyelitis	Contact dermatitis
Toxic shock syndrome	Gouty arthritis
	Drug reactions
	Malignancy

TREATMENT:

- GENERAL MEASURES:
  - Elevation of affected area
  - Antibiotics
  - Treatment of underlying conditions (tinea pedia, lymphoedema, chronic venous insufficiency)

Mild early cellulitis and erysipelas

To cover *Staphylococcus aureus* and *Streptococcus pyogenes*, use:

di/flucloxacillin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for 7 to 10 days.

If *S. pyogenes* is confirmed, or suspected due to clinical presentation (see above) or local disease patterns (eg in Indigenous communities in central and northern Australia), use:

1 **phenoxymethylpenicillin 500 mg (child: 10 mg/kg up to 500 mg) orally, 6-hourly for 10 days**

OR

1 **procaine penicillin 1.5 g (child: 50 mg/kg up to 1.5 g) IM, daily for at least 3 days.**

Cephalexin can be used for patients with penicillin hypersensitivity (excluding immediate hypersensitivity, see Table 2.2), and is a useful alternative to di/flucloxacillin in children due to better tolerability, and palatability of the liquid formulation. Use:

cephalexin 500 mg (child: 12.5 mg/kg up to 500 mg) orally, 6-hourly for 7 to 10 days.

For patients with immediate penicillin hypersensitivity (see Table 2.2), use:

clindamycin 450 mg (child: 10 mg/kg up to 450 mg) orally, 8-hourly for 7 to 10 days.

Severe cellulitis

If patient has significant systemic features or is not responding to oral therapy after 48 hours, commence IV therapy.

To treat infection with either streptococci or staphylococci, use initially:

di/flucloxacillin 2 g (child: 50 mg/kg up to 2 g) IV, 6-hourly.

For patients hypersensitive to penicillin (excluding immediate hypersensitivity, see Table 2.2), use initially:

cephazolin 2 g (child: 50 mg/kg up to 2 g) IV, 8-hourly.

For patients with immediate penicillin hypersensitivity (see Table 2.2), use initially:

1 **clindamycin 450 mg (child: 10 mg/kg up to 450 mg) IV or orally, 8-hourly**

OR

1 **lincomycin 600 mg (child: 15 mg/kg up to 600 mg) IV, 8-hourly**

OR

2 **vancomycin 1.5 g (child less than 12 years: 30 mg/kg up to 1.5 g) IV, 12-hourly (adjust initial dosage for renal function and monitor blood concentrations, see Dosing and monitoring of vancomycin; slow infusion required).**

Where home-based intravenous antimicrobial therapy is practical, for initial therapy in carefully selected patients, use:

1 **cephazolin 2 g IV, 12-hourly**

OR THE COMBINATION OF

1 **cephazolin 2 g IV, daily**

PLUS

probenecid 1 g orally, daily.

- Consider surgical consultation in patients with bullae, crepitus and pain out of proportion to examination or in those with rapidly progressive erythema with signs of systemic toxicity

CUTANEOUS ABSCESSES, FURUNCLES AND CARBUNCLES:

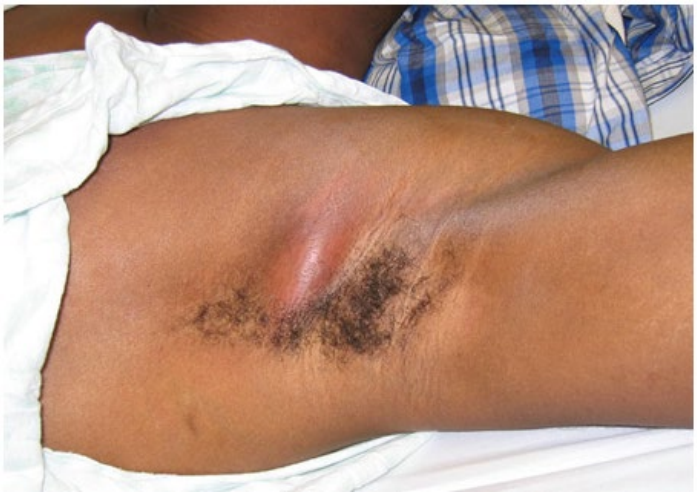
FURUNCLES AND CARBUNCLES INVOLVE THE EPIDERMIA AND ABSCESSES INVOLVE THE DEEPER SOFT TISSUE AND CAN DEVELOP IN OTHERWISE HEALTHY PATIENTS WITH NO RISK FACTORS

PATHOPHYSIOLOGY:

- Skin abscesses typically begin as localized superficial cellulitis with loculation and subsequent walling-off of cellular debris/leukocytes
- Infection can be caused by one or multiple pathogens that typically include skin flora or organisms from adjacent mucous membranes
- Any process causing a breach in the skin barrier heightens the risk for skin abscess:
    - Foreign bodies, bites, IVDU, abrasions/lacerations
- Other risk factors → diabetes, immunologic disorders

CLINICAL FEATURES:

- Skin abscesses are fluctuant, tender and erythematous nodules with surrounding erythema



- Signs of systemic toxicity are rare in simple abscesses

DIAGNOSIS:

- Diagnosis is clinical
- BEDSIDE ULTRASOUND is an invaluable tool for distinguishing abscess from cellulitis and also useful in identifying a foreign body

TREATMENT:

- It is best to drain large abscesses or those in deep areas in OT
- Specialist areas include HAND (PALMS), SOLES OR NASOLABIAL FOLDS and usually require specialist involvement for drainage
- Can perform I&D for simple furuncles, carbuncles and skin abscesses in ED
- ANTIBIOTICS ARE GENERALLY UNNECESSARY AFTER I&D IN THOSE WITHOUT SURROUNDING CELLULITIS (especially in healthy patients)
    - It is reasonable to prescribed antibiotics for patients with multiple lesions, extensive surrounding cellulitis, immunosuppression or systemic infection

<u>NECROTISING SOFT TISSUE INFECTIONS:</u>

- A spectrum of illnesses characterised by fulminant, extensive soft tissue necrosis, systemic toxicity and high mortality
- RISK FACTORS:

    - Advances age
    - Diabetes
    - Alcoholism
    - Peripheral vascular disease
    - Heart disease
    - Renal failure
    - HIV
    - NSAID use
    - Decubitus ulcers
    - Chornic skin infection
    - IVDU
    - Immunosuppression
- Various classifications

Table 147-6 Classification of Necrotizing Soft Tissue Infection

Classification Factor	Comment
Anatomic location	Fournier gangrene of perineum/scrotum
Depth of infection	Necrotizing adipositis (most common), fasciitis, myositis
Microbial cause	Type 1: Polymicrobial (most common)
	Type II: Monomicrobial (*Staphylococcus*, *Streptococcus*, *Clostridia* species and methicillin-resistant *S. aureus*)
	Type III: *Vibrio vulnificus**

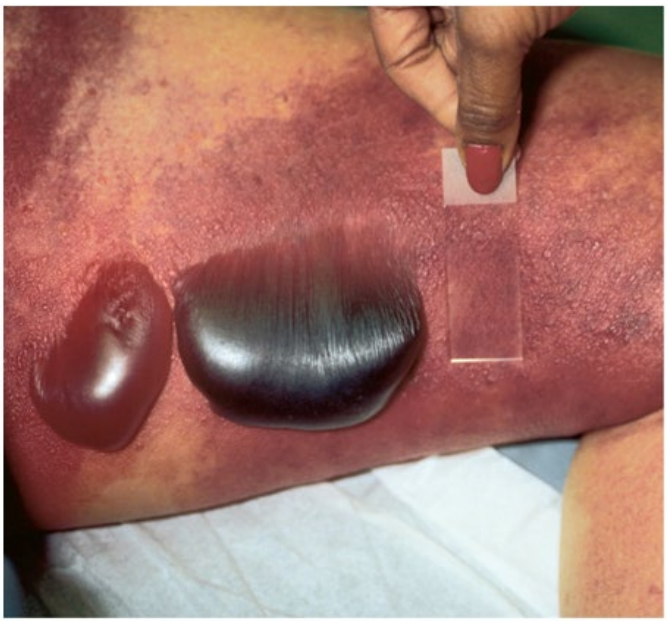
- Mortality rate remains 25-35%
    - Bacteraemia reported in 25-30% cases and is a strong predictor or mortality
- Other patient factors that predict mortality:
    - IVDU
    - Age <1, >60
    - Comorbid conditions (esp cancer, CRF, CHF)
    - Positive blood cultures
    - Trunk or perineal involvement
    - Delay in diagnosis

PATHOPHYSIOLOGY:

- Rapid necrotizing process gengins with direct invasion of subcutaneous tissue from external trauma (IVDU, surgical incision, abscess, insect bite) OR from direct spread from a perforated viscous (usually colon, rectum or anus)
- Spontaneous development is rare
- Bacteria proliferate and invade subcutaneous tissue/deep fascia leading to release of exotoxins that lead to tissue ischaemia, liquefaction necrosis and systemic toxicity
- INFECTION CAN SPREAD AS RAPIDLY AS 2.5CM PER HOUR
- Tissue ischaemia produced in all such infections impedes immune system destruction of bacteria and prevents adequate delivery of antibiotics
    - Antibiotics are therefore rarely effective and immediate surgical intervention remains the cornerstone of successful management

CLINICAL FEATURES:

- PAIN OUT OF PROPORTION TO PHYSICAL FINDINGS → perhaps the most important feature to make the diagnosis early
- Classically patients have tissue pain, anxiety and diaphoresis
- About 10-40% of the time, patients report trauma or break in skin 48 hours prior
- Painful area may show BRAWNY OEDEMA AND CREPITUS as a result of GAS PRODUCTION BY BACTERIA
- In one study, in 50% cases, the only signs were erythema, tenderness or marked oedema beyond the area of redness (crepitus only present in 13-31%)
- Systemic manifestations include a low-grade fever with tachycardia out of proportion to the fever
- BULLAE MAY BE PRESENT



DIAGNOSIS:

- Diagnosis is CLINICAL
- X-ray may show subcutaneous gas but CT is more sensitive and can demonstrate fascial thickening and oedema with deep tissue collection and gas formation (no additional benefit with IV contrast). MRI has best sensitivity but usually relates to delay to diagnosis/treatment
-

TREATMENT:

- Begin aggressive fluid resuscitation immediately and avoid vasoconstrictors if possible to maintain optimal perfusion to already ischaemic tissue
- IV antibiotics reflect emergence of MRSA and decline of clostridial infections

Empirical therapy

For empirical therapy, where the diagnosis is uncertain and until tissue and blood culture results are available, use initially:

meropenem 1 g (child: 25 mg/kg up to 1 g) IV, 8-hourly

PLUS EITHER

1 **clindamycin 600 mg (child: 15 mg/kg up to 600 mg) IV, 8-hourly**

OR

1 **lincomycin 600 mg (child: 15 mg/kg up to 600 mg) IV, 8-hourly.**

Clindamycin or lincomycin is recommended to reduce bacterial toxin production, but there is no clinical evidence to support this. Penicillin is commonly added but is theoretically unnecessary. Consider the use of IV immunoglobulin if *Streptococcus pyogenes* necrotising fasciitis is suspected (see below)—seek expert advice.

Streptococcus pyogenes necrotising fasciitis

For *Streptococcus pyogenes* necrotising fasciitis, in addition to surgical debridement, use:

benzylpenicillin 1.8 g (child: 45 mg/kg up to 1.8 g) IV, 4-hourly

PLUS EITHER

1 **clindamycin 600 mg (child: 15 mg/kg up to 600 mg) IV, 8-hourly**

OR

1 **lincomycin 600 mg (child: 15 mg/kg up to 600 mg) IV, 8-hourly**

PLUS (consider after expert advice)

normal immunoglobulin (adult and child) 0.4 to 2 g/kg IV, for 1 or 2 doses during the first 72 hours.

For patients hypersensitive to penicillin (excluding immediate hypersensitivity, see Table 2.2), **substitute** for benzylpenicillin:

cephazolin 2 g (child: 50 mg/kg up to 2 g) IV, 8-hourly.

Clostridial infection

Clostridial infection varies from mild cellulitis to overwhelming myonecrosis (gas gangrene). The basis of treatment is surgical debridement of necrotic tissue, resuscitation and antibiotic therapy. In severe infections, hyperbaric oxygen should be considered if available. The diagnosis of gas gangrene is a clinical one. Neither the isolation of clostridia nor the presence of gas in tissue is diagnostic of the condition.

For clostridial infection with or without myositis/myonecrosis (gas gangrene), use:

benzylpenicillin 2.4 g (child: 60 mg/kg up to 2.4 g) IV, 4-hourly.

For patients with penicillin hypersensitivity, use:

metronidazole 500 mg (child: 12.5 mg/kg up to 500 mg) IV, 8-hourly.

- EARLY SURGICAL CONSULTATION FOR ALL SUSPECTED CASES REMAINS THE GOLD STANDARD
    - Fundamental therapy remains operative exploration and surgical debridement
    - Mortality skyrockets if debridement is delayed beyond 24 hours
- Provide tetanus prophylaxis
- Controversies → hyperbaric, IV IG

OTHER SOFT TISSUE INFECTIONS:

FOLLICULITIS:

- Inflammation of hair follicles related to infection, irritation or physical injury → typically involves superficial infection with Staph aureus most often of apocrine areas of upper back, chest, buttocks, hips and axilla but can occur in any hair-bearing region of the body

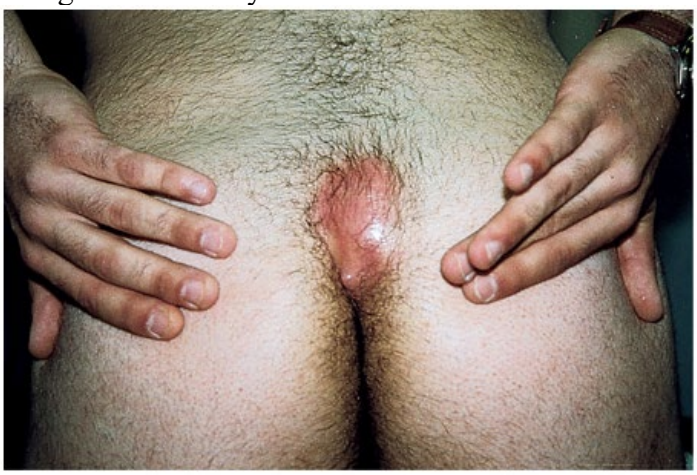
- Classically clusters of pruritic, erythematous lesions that are usually <5mm diameter
- Treated with twice-daily cleansing with mild hand soap, warm compresses usually sufficient → may need to add topical bacitracin, polymixin B
    - Oral antibiotics for more extensive or painful cases

HIDRADENITIS SUPPURATIVA:

- A recurrent, suppurative and scarring disease of the apocrine glands, especially those of African descent
- Neither a disease of poor hygiene or contagious

PILONIDAL ABSCESS:

- Located along superior gluteal fold
- Causative organisms usually are skin flora



- Treatment is incision and drainage with care to remove excess hair → definitive treatment consists of wide surgical incision and healing by secondary intention

INFECTED SEBACEOUS CYST:

- Occur diffusely throughout the body → once infection occurs, abscess formation is common
- I&D is appropriate treatment

BARTHOLIN GLAND ABSCESS:

- Common in women of reproductive age
    - An abscess in a perimenopausal woman requires gynaecological follow up to exclude carcinoma
    - The Bartholin glands area pair of organs located in the labia minora in the 4 and 8 O'clock positions that usually provide moisture for the vagina
    - Abscess development begins with cyst formation when these ducts become blocked → pain and vulvar discomfort occurs with infection
- Definitive treatment is marsupialisation of glands by gynaecology