Bacterial skin infections summary

MSS microbiology

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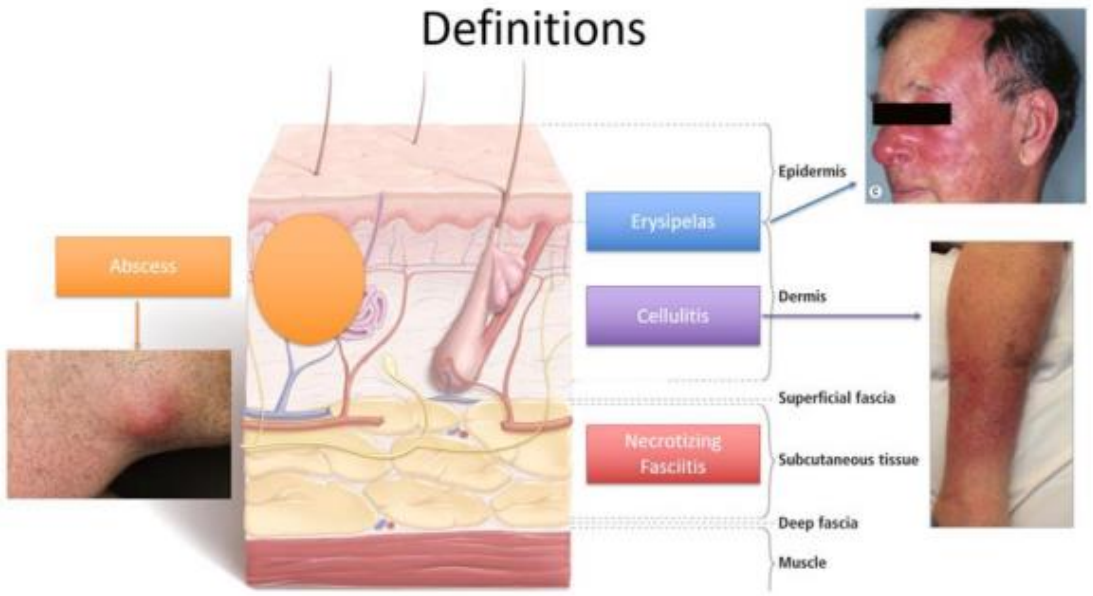
MSS microbiology is concerned with infections involving one or multiple layers of the following:

Layer	Infection
Epidermis	Impetigo, erysipelas
Dermis	Cellulitis
Fascia and subcutaneous tissue	Necrotizing fasciitis
Muscles	Myositis
Bones	Osteomyelitis



1. Impetigo Honey crust lesions 

Layer infected	Epidermis
Epidemiology	Infects children mainly (hygiene dependent)
Transmission	Direct contact, fomites (highly contagious) **Primary impetigo:** direct bacterial invasion of previously normal skin. **Secondary impetigo**: infection at sites of minor skin trauma.
Etiology	S. aureus mainly. Beta-hemolytic streptococci (primarily group A) account for a minority of cases.
Manifestations	**Non-bullous impetigo** (most common): papules that progress to vesicles surrounded by erythema, subsequently they become pustules that enlarge and rapidly break to form thick, adherent crusts with a characteristic golden appearance over approximately one week. **Bullous impetigo:** vesicles enlarge to form flaccid bullae with clear yellow fluid. **Ecthyma:** an ulcerative form of impetigo in which the lesions extend through the epidermis and deep into the dermis.
Diagnosis	Mainly **clinical** A Gram stain and culture of pus or exudate is recommended to identify the specific pathogen.
Treatment	Treatment of impetigo is important for reducing spread of infection, hastening the resolution of discomfort, and improving cosmetic appearance. You can treat empirically based on typical clinical manifestations. **Topical therapy:** Mupirocin and retapamulin are first-line treatments. Mupirocin is a mixture of several pseudomonic acids (depletes tRNA of bacteria → inhibits protein synthesis). For patients with numerous lesions, treat with oral abx.

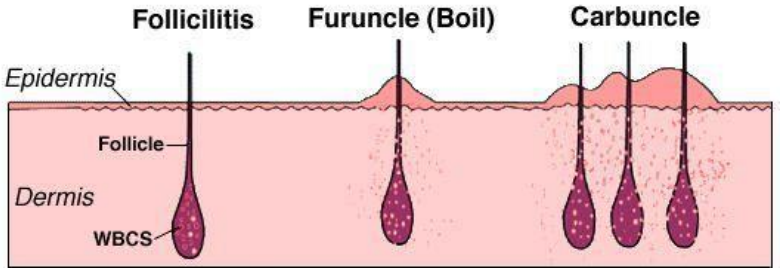
2. Folliculitis: inflammation of the superficial or deep portion of the hair follicle.

Layer infected	Hair follicles in the epidermal layer of the skin.
Etiology	S. aureus (most common) P. aeruginosa, associated with the use of unchlorinated hot tubs. (hot-tub folliculitis) Rarely, Candida and certain dermatophytes.
Manifestations	Pinpoint erythema around individual hair follicles. A small amount of purulence may be seen. This can be seen in an isolated body area or throughout the skin.
Diagnosis	Diagnosis is made clinically, but purulent material can be cultured.
Treatment	Often self-limited. Warm compresses or topical antibiotics can be considered.

3. Skin abscess: (infection that produces purulent material)

Layer infected	Dermis and deeper layers
Transmission	Bacteria are typically indigenous to the skin of the involved area.
Etiology	The most common organisms are S. aureus (MRSA being the most common in the US) and streptococci. Abscesses in the perineal region contain organisms found in the stool, commonly anaerobes or a combination of aerobes and anaerobes.
Manifestations	A skin abscess is round and feels firm and squishy due to the thick membrane around it and the liquid pus inside. It is usually painful, and the overlying skin is often red.
Diagnosis	Clinical Culture is recommended, primarily to identify MRSA.
Treatment	Incision and drainage (I&D). Antibiotics are unnecessary unless the patient has signs of systemic infection, cellulitis, multiple abscesses, immunocompromise, or a facial abscess in the area drained by the cavernous sinus. In these cases, empiric therapy should be started with a drug active against MRSA.
Differentials	Conditions resembling simple cutaneous abscesses include hidradenitis suppurativa and ruptured epidermal cysts. Hidradenitis suppurativa is a chronic inflammatory condition of the hair follicle and associated structures. It occurs near hair follicles where there are sweat glands, usually around the groin, buttocks, breasts and armpits.

When skin abscesses form around hair follicles, they're referred to as carbuncles and furuncles.



4. Erysipelas

Layer infected	Upper dermis and superficial lymphatics.
Epidemiology	Mostly occurs in young children and older adults.
Transmission	Bacterial entry via breaches in the skin barrier.
Etiology	Beta-hemolytic streptococci; group A strep. mainly
Manifestations	Acute onset of symptoms with systemic manifestations (fever, chills, severe malaise, and headache) before local inflammation occurs. **Nonpurulent** skin erythema, edema, and warmth. Nearly always unilateral, face and lower extremities are the most common sites of involvement. Classic descriptions of erysipelas note "butterfly" involvement of the face There's clear demarcation between involved and uninvolved tissue. There may be a raised, advancing border or erythema with central clearing.
Diagnosis	Clinical
Treatment	Empiric therapy against strep.

5. Cellulitis

Layer infected	Deeper dermis and subcutaneous fat
Epidemiology	Mostly in middle-aged and older adults. In nontropical regions, it has predilection for warmer months.
Transmission	Bacterial entry via breaches in the skin barrier.
Etiology	GAS (most common) S. aureus (including MRSA) is a notable but less common cause.
Manifestations	Erythema, edema, and warmth. With or without purulence. Petechiae, hemorrhage and superficial bullae can occur. Nearly always unilateral, and the lower extremities are the most common site of involvement. Patients with cellulitis tend to have a more indolent course with development of localized symptoms over a few days. (Remember that cellulitis is deeper than erysipelas) Systemic manifestations may be present.
Diagnosis	Mainly clinical Cultures of debrided material and blood cultures can be done in some cases
Treatment	Patients with nonpurulent cellulitis should be managed with empiric therapy against strep. And staph. : cefazolin IV, cephalexin oral. Deepening of erythema may be observed following initiation of antimicrobial therapy. This may be due to destruction of pathogens that release particles that enhance local inflammation and should not be mistaken for therapeutic failure. Symptoms improve within 24-48 hours of beginning antimicrobial therapy (severe cases may take up to 72 hours)

Predisposing factors associated with risk of cellulitis and/or skin abscess include:

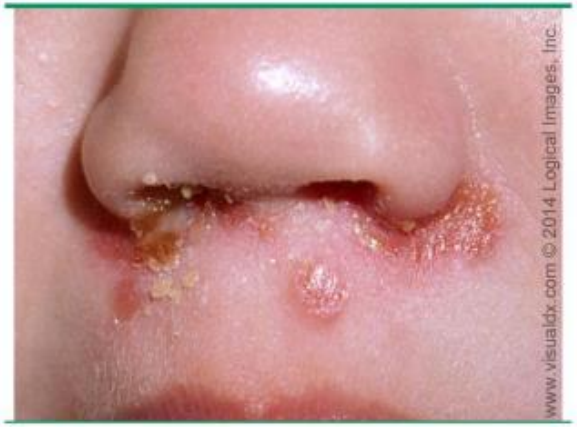
- Skin barrier disruption due to trauma (abrasion, penetrating wound, pressure ulcer, venous ulcer, insect bite, injection drug use)
- Skin inflammation (eczema, radiation therapy, psoriasis)
- Edema due to impaired lymphatic drainage or due to venous insufficiency
- Obesity
- Immunosuppression (diabetes or HIV infection)
- Skin breaks between the toes ("toe web intertrigo"); these may be clinically inapparent
- Pre-existing skin infection (such as tinea pedis, impetigo, varicella)

Cultures of debrided material and blood cultures (prior to addition of antibiotics) are warranted in the following circumstances:

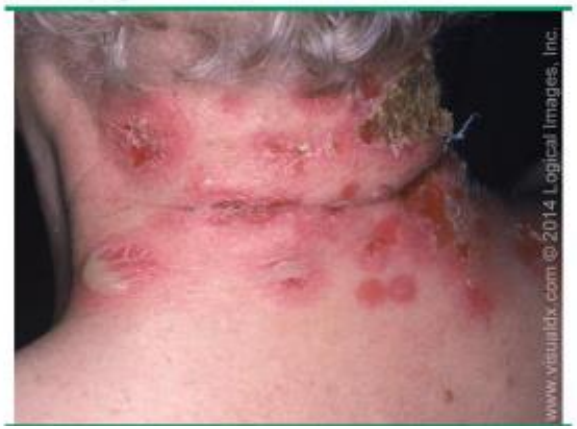
- Severe local infection
- Systemic signs of infection (e.g. fever)
- History of recurrent or multiple abscesses
- Failure of initial antibiotic therapy
- Extremes of age (young infants or older adults)
- Presence of underlying comorbidities (lymphedema, malignancy, neutropenia, immunodeficiency, splenectomy, diabetes)
- Special exposures (animal bite, water-associated injury)

Appendix

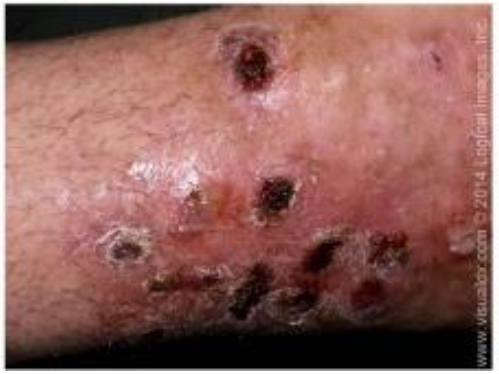
Nonbullous impetigo



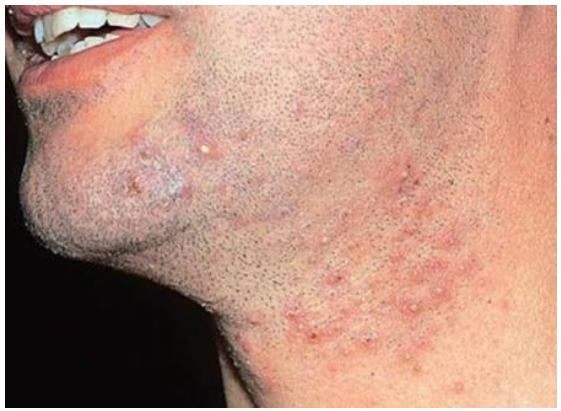
Bullous impetigo



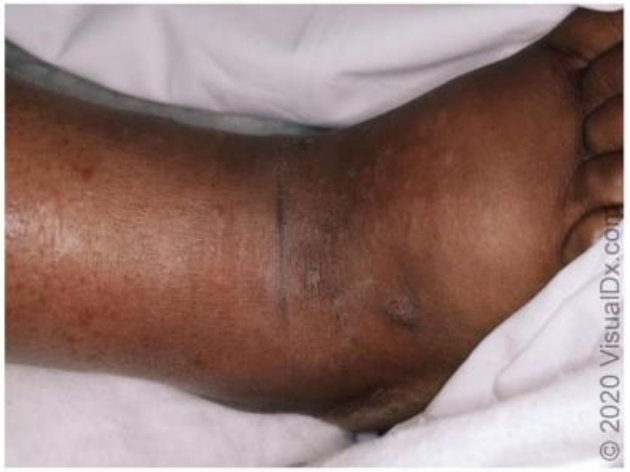
Ecthyma



Folliculitis

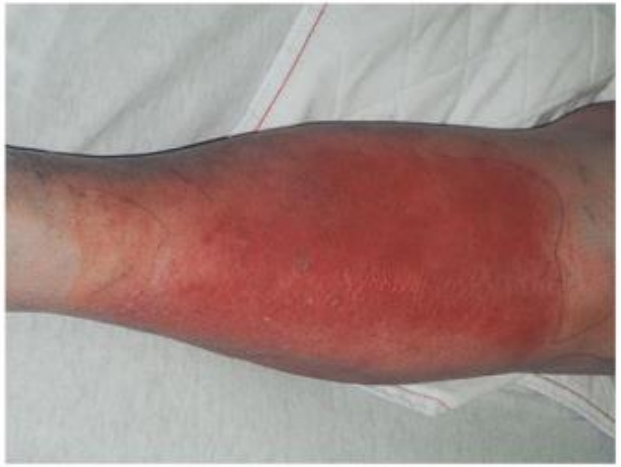


Cellulitis of the ankle



Edema and erythema around the ankle and on the dorsal foot.

Erysipelas of the leg



Erysipelas of the lower leg. The rash is intensely red, sharply demarcated, swollen, and indurated.