MSS System Bacterial Infections

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Introduction

- Intact skin is characterized by the presence of many defense mechanisms including:
 - The presence of highly keratinized surface which prevents direct microbial invasion
 - Continuous sloughing (shedding) of skin cells with microorganisms attached to them
 - Sebum decomposition by some microorganisms producing fatty acids that reduce skin pH
 - High salt concentration of sweat
 - Secretion of antimicrobial substances such as antimicrobial peptides and lysozymes
 - Presence of skin **normal flora** which competes with the growth of potential pathogens

Introduction

- Large number of **normal flora** occupies the skin in various locations
- It has been long established that the majority of organisms living on the skin are Gram +ve bacteria such as Staphylococcus, Micrococcus, Corynebacterium (diphtheroids), and Proprionibacterium
- However, the Human Microbiome Project has revealed that a large number of Gram –ve bacteria, such as *Pseudomonas* spp., can also reside on the skin

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Introduction

- Infections of the skin can result from microbial invasion from an external source
 - Invading microbes can gain access into deeper skin layers when its integrity is compromised even by repeated minor traumas
 - Microbial invasion can also occur along ducts of pilosebaceous and sweat glands
- Skin infection can also be caused by organisms reaching the skin through the **bloodstream** as part of a systemic disease
 - Blood-borne involvement is evidenced by rashes in many viral and bacterial infections, such as measles and secondary syphilis, or may yield more chronic granulomatous skin lesions in tuberculosis, and syphilis.
- Skin lesions remote from sites of infection can be produced by some bacterial toxins, such as the pyrogenic exotoxins of group A streptococci and Staphylococcus aureus. They can also result from immunologic responses to microbial antigens that have reached the skin

Skin Lesions Terminology

- Macule: a change in skin color without elevation or depression
- Papule: solid elevation of skin with no visible fluid
- Maculopapular rash: flat, red area on the skin that is covered with small confluent bumps
- Vesicle: elevated lesion filled with clear fluid (small blister)
- Bullous: large blisters (large vesicles)
- Pustule: elevated lesion filled with purulent fluid (pus)
- Cyst: raised encapsulated lesion containing solid or semisolid material
- Abscess: localized infection, which appears as an inflamed, fibrous encapsulated lesion enclosing a core of pus
- **Hematoma**: localized collection of blood outside the blood vessels (petechiae (<3mm), purpura (3-10mm), ecchymosis (>10mm))

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Folliculitis

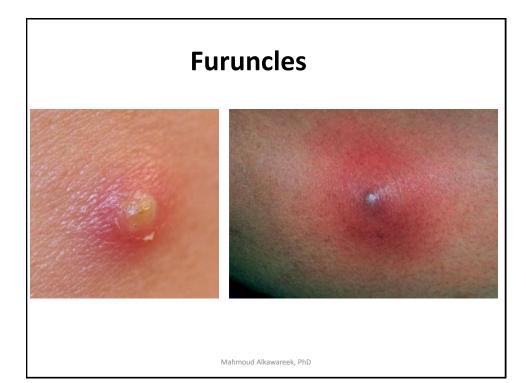
- Folliculitis is a minor infection of the hair follicles and is usually caused by Staphylococcus aureus.
- It is often associated with areas of friction and of sweat gland activity and is thus seen most frequently on the neck, face, axillae, and buttocks.
- Presents as papules or small pustules
- A more extensive form of folliculitis can also be caused by *Pseudomonas aeruginosa*, which is usually associated with the use of hot tubs and whirlpool baths.
 - Unless these facilities are thoroughly cleansed and adequately chlorinated, they can grow large numbers of Pseudomonads at their normal operating temperatures, causing extensive folliculitis on areas of the body that have been immersed.

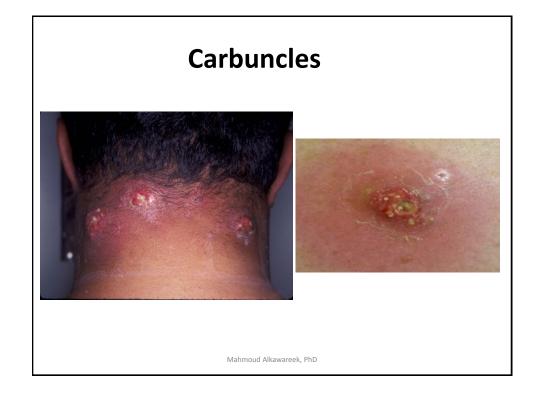
Folliculitis



Furuncles & Carbuncles

- A furuncle results when the inflammation of a single hair follicle (folliculitis) or sebaceous gland progresses into a larger, red and tender pustule or abscess.
- Furuncles are mainly caused by S. aureus
- Furuncles may be **solitary** or in **clusters (boils)** and they may constitute a troublesome **recurrent disease**
- Spread of infection to the dermis and subcutaneous tissues can result in a more extensive interconnected abscess called carbuncle
- Carbuncles are extremely painful and can give rise to systemic disease (especially in elderly patients)





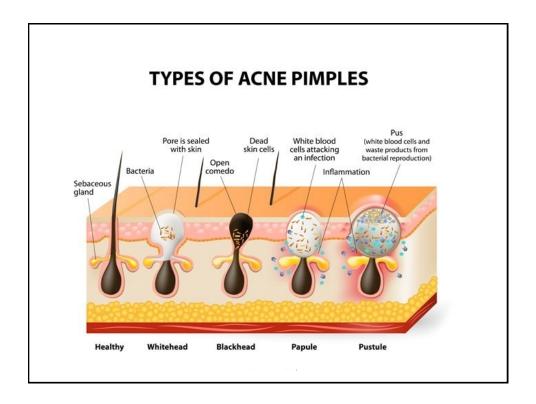
Treatment of Folliculitis, Furuncles and Carbuncles

- Folliculitis and individual furuncles usually heal on their own in about 2 weeks but are normally treated locally by measures designed to establish drainage without the use of antibiotics
- Chronic/recurrent furunculosis may require elimination of nasal carriage of S. aureus, which is sometimes the source of infection
- Antibiotics are not usually required unless surrounding cellulitis or carbuncles develop.
- Antibiotics that may be used include:
 - Topical antibiotics: mupirocin or Neomycin/polymyxin B/bacitracin ointment)
 - Oral antibiotics: β-lactamase resistant penicillins (e.g. oxacillin, cloxacillin, flucloxacillin...), first-generation cephalosporins (e.g. cephalexin) and clindamycin are most commonly used

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Acne Vulgaris

- Acne vulgaris also involve the inflammation of hair follicles and associated sebaceous glands
- The primary cause of acne vulgaris is the hormonal (male sex hormones) influences on sebum secretion that occur at puberty
- Comedo indicates whiteheads (closed) and blackheads (open) which are mild form of acne where hair follicles & sebaceous glands get plugged with sebum (& keratin)
- Propionibacterium acnes, the predominant anaerobe of normal skin, can multiply in the plugged ducts reaching high numbers
- Organic acids produced by this bacteria are believed to stimulate an inflammatory response and thus contribute to the disease process
- Inflammatory **papules and pustules** can then be formed which are considered a **moderate** form of the disease



Acne Vulgaris

- In severe cases, the plugged ducts rupture and sometimes get encysted in connective tissue forming nodules or cycts (cystic acne)
- Treatment is by frequent cleansing of the skin and the use of topical drying agents (benzoyl peroxide and salicylic acid)
- Treatment may also involve the use of oral antibiotics (most commonly tetracyclines, macrolides and clindamycin).
- Isotretinoin (Vit. A derivative) is used for severe & persistent cases; it inhibits sebum production, but has serious side effects (e.g. intestinal bleeding, damages the fetus)

Impetigo

- Impetigo, also termed pyoderma, is a common, sometimes epidemic skin infection that is primarily caused by group A β-hemolytic streptococci (i.e. Streptococcus pyogenes).
- The initial lesion is often a small vesicle that develops at the site of invasion and ruptures with superficial spread characterized by skin erosion and a serous exudate, which dries to produce a honey-colored crust.
- The exudate and crust contain numerous infecting streptococci which makes the disease highly contagious
- *S. aureus* may also produce **pustular impetigo** or contaminate the lesions caused by streptococci.
- Epidemic impetigo is most common in children and under conditions of heat, humidity, poor hygiene, and overcrowding

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Impetigo



Impetigo

- The infection is spread by direct contact with lesions or with nasal carriers and also by fomites such as shared clothing and towels.
- It is sometimes caused by **nephritogenic strains** of *S. pyogenes*, particularly in the tropics, and acute **glomerulonephritis may result**.
- However the strains causing impetigo differ from those causing strep throat infection and are not associated with rheumatic fever
- Treatment is usually with penicillins or erythromycin in addition to topical antibiotics or skin antiseptics to limit spread

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Bullous Impetigo

- Bullous impetigo is a distinct disease caused by strains of *S. aureus* that produce exfoliation toxins (exfoliative toxins A and B).
- It is most common in **small children**, but may occur at any age.
- The infection is characterized by large serumfilled bullae (large blisters) within the skin layers at the site of infection
- Minor infections are treated topically; however, bullous impetigo in infants is a serious disease that usually requires systemic antibiotic treatment

Bullous Impetigo



Erysipelas

- Erysipelas is a rapidly spreading infection of the deeper layers of the dermis that is almost always caused by group A streptococci
- It is associated with edema of the skin, welldemarcated erythema, pain, and systemic manifestations of infection including fever and lymphadenopathy
- Because the infection is intradermal, the streptococci cannot usually be isolated from the skin surfaces.
- The disease can progress to septicemia or local necrosis of skin. It requires immediate treatment with systemic antibiotics such as penicillins or erythromycin





Cellulitis

- Cellulitis is not a skin infection as such, but it can develop by extension from skin or wound infections. It involves an acute inflammation of the subcutaneous connective tissue (fat) in addition to the deep dermis layers
- It usually presents with swelling, erythema and pain and often with systemic signs and symptoms
- Unlike erysipelas, **erythema borders** in cellulitis are generally **not sharp**
- It can be caused by many pathogenic bacteria, but S. aureus and group A streptococci are most common.





Cellulitis

- Haemophilus influenzae type b is a cause in infants and children.
- Enteric Gram-negative rods, clostridia, and other anaerobes may also cause cellulitis as a complication of wound infections, particularly in immunocompromised hosts and individuals with uncontrolled diabetes.
- Treatment is with **systemic antibiotics** such as β -lactamase resistant penicillins (e.g. oxacillin), first-generation cephalosporins (e.g. cephalexin), macrolides, clindamycin or vancomycin

Scarlet Fever

- Scarlet fever usually follows a respiratory infection (or occasionally skin infection such as impetigo or cellulitis) caused by certain lysogenic strains of S. pyogenes
- These strains produce erythrogenic toxin which results in a sandpaper-like rash (most often on the neck, chest, elbows, and inner surfaces of the thighs) accompanied by high fever
- Mostly affects school-age children (5-15 years)
- Treatment by penicillins, first-generation cephalosporins or macrolides

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Scarlet Fever



Scalded Skin Syndrome (SSS)

- Caused by strains of S. aureus that produce exfoliation toxins similar to those in bullous impetigo but the toxin reaches the bloodstream and causes lesions remote from site of infection (can be thought of as systemic form of bullous impetigo)
- Causes erythema, bullous formation, and epidermal desquamation (leading to burned appearance) which can spread all over the body
- Removal of keratinized layer makes the patient vulnerable to secondary infections
- Most common in neonates and children less than 5 years old
- Immediate treatment with systemic antibiotics is required (β-lactamase resistant penicillins, clindamycin or vancomycin).

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Scalded Skin Syndrome (SSS)





Toxic Shock Syndrome (TSS)

- A potentially fatal illness caused by pyrogenic toxins (mainly TSST-1 and enterotoxin type B) produced by certain strains of S. aureus (and occasionally S. pyogenes strains)
- Most common in children and young women using intra-vaginal tampons
- Characterized by a skin rash followed by desquamation at a deeper level than in SSS.
- Also systemic symptoms: high fever, hypotension, vomiting, diarrhea, sore throat, and muscle pain
- May progress to severe shock associated with renal and hepatic damage.
- Treatment:
 - Removal of infection source
 - Combination antibiotic therapy (to cover both *S. aureus* and *S. pyogenes*): two or more of cephalosporins, penicillins, vancomycin and clindamycin

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Necrotizing Fasciitis

- A rapidly progressive infection of the fascia, with severe necrosis of the subcutaneous tissues.
- Also known as flesh-eating disease, hemolytic streptococcal gangrene...
- Usually a mixed infection including S. pyogenes and other bacteria such as S. aureus, Gram-ve bacilli, and anaerobes
- Treatment involve surgical debridement (or limb amputation) and use of combination IV antibiotics such as clindamycin, vancomycin, extended spectrum penicillins (e.g. piperacillin) and

Necrotizing Fasciitis



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Wound Infections

 Wounds are classified according to their of potential contamination (and thus risk of infection) into 4 classes:

Class	Description	Examples	Risk of Infection
I. Clean	Surgical wounds made under aseptic conditions No entry to Respiratory, gastrointestinal, biliary, and urinary tracts	Mastectomy Vascular surgery Hernia	< 2%
II. Clean- Contaminated	Surgical wounds involving respiratory, gastrointestinal, biliary, and urinary tracts Without gross spillage	Gastrostomy Small bowel resection	< 10%
III. Contaminated	Major break in aseptic technique Spillage from GI tract Nonpurulent infected tissues Fresh traumatic wounds	Diverticulitis Penetrating wounds	~ 20%
IV. Dirty	Purulent infected tissues Old (infected) traumatic wounds	Abscess I&D Perforated bowel	~ 40%

Wound Infections

- · Sources of Infection:
 - Endogenous: patient's normal flora
 - Exogenous: transmitted from other people, fomites or the environment
- Factors contributing to wound infection:
 - Class of wound
 - Inoculum size (contamination dose)
 - Microbial virulence
 - Wound condition: necrosis, edema, poor blood supply...
 - General health and nutritional status of the patient

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Wound Infections

• Etiologic agents:

Wounds			
Trauma	Clostridium		
	Enterobacteriaceae		
	Pseudomonas aeruginosa		
Surgical (clean)	Staphylococcus aureus		
	Enterobacteriaceae		
	Group A streptococci		
Surgical (dirty):	Staphylococcus aureus		
	Enterobacteriaceae		
	Anaerobes		
Burns	Pseudomonas aeruginosa	Candida albicans	
	Staphylococcus aureus		
	Enterobacteriaceae		

Wound Infections

- Treatment and Prevention:
 - Preoperative antibiotic prophylaxis for cleancontaminated and contaminated wounds
 - Most commonly used are 1st and 2nd generation cephalosporins, e.g. cefazolin (1st), cefotetan/cefoxitin (2nd)
 - Severe wound infections are almost always treated with a combination of:
 - · Surgical approaches:
 - Removal of necrotic tissue (surgical debridement)
 - Removal of contaminated foreign bodies (e.g. sutures)
 - Pus drainage
 - Antibiotics:
 - The choice of antibiotic depends on the probable (or known) infecting microorganism in addition to patient factors such as allergies, hepatic and renal function and severity of disease process