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Disease	Pathogen	Symptoms	DX	TX	Description
Impetigo	S. aureus, Beta-hemolytic streptococci (primarily group A, but occasionally C and G)	-**Nonbullous impetigo**: papule→ vesicle surrounded by erythema→ pustule enlarges and breaks down→ thick adherent crusts with golden appearance for one week. -**Bullous impetigo**: Vesicles enlarge →flaccid bullae with clear yellow fluid.	-clinical manifestations -gram stain + culture of pus or exudate.	- can be initiated in apparent cases. -reduces spread, resolves discomfort, improves appearance. -topical MUOIRCIN'' pseudomonic acids inhibiting isoleucyl-Trna=> inhibition of protein synthesis'', RETAPAMULIN. -severe cases => oral.	- contagious superficial bacterial infection most frequently in children ages two to five years, older children, adults. -**PRIMARY IMPETIGO**: (direct bacterial invasion of previously normal skin), **SECONDARY IMPETIGO**: (infection at sites of minor skin trauma, abrasions, insect bites, eczema. - **Nonbullous impetigo** is the most common form of impetigo. - **Bullous impetigo** in young children. - **Ecthyma:** ulcerative impetigo. lesions extend through the epidermis and deep into the dermis.
Folliculitis	S. aureus is the most common cause, P. aeruginosa (unchlorinated hot tubs), Rarely Candida and certain Dermatophytes.	- Bacteria and purulent material accumulate in hair follicles in the epidermal layer of the skin. -pinpoint erythema around hair follicles, small amount of purulent material in selected areas or throughout the skin.	-clinical -culture of purulent material	-resolves by its own, TX not needed. -selected cases→ warm compresses, topical antibiotics.	- inflammation of the superficial or deep portion of the hair follicle.

skin abscess / carbuncles / furuncles	- indigenous to skin involved (predisposing factors such as trauma). - abscesses on the trunk, extremities, axillae, head and neck→ most common organisms are Staphylococcus aureus (with [MRSA] being most common in US) and streptococci. - abscesses in the perineal region contain organisms found in the stool, commonly anaerobes or a combination of aerobes and anaerobes.	-cutaneous abscesses with purulent material, round feels firm and squishy due to the thick membrane around it and the liquid pus inside, usually painful (tender), overlying skin is often redm warm→ taking 2 weeks to fully form. - painful, fluctuant, erythematous nodule, with or without surrounding cellulitis.	-clinical. -culture to identify MRSA.	- incision and drainage. - Antibiotics 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.	- Conditions resembling simple cutaneous abscesses include hidradenitis suppurativa'' chronic inflammation of hair follicle and associated structures. cause is unknown, occurs near hair follicles where there are sweat glands, usually around the groin, bottom, breasts and armpits'' and ruptured epidermal cysts.
Cellulitis	- beta-hemolytic streptococci most commonly group Streptococcus (Streptococcus pyogenes), S. aureus (MRSA) is a notable but less common cause.	- edema, erythema (spreading in weeks), warmth, Petechiae and hemorrhage in erythematous skin, and superficial bullae, fever, systemic manifestations, discoloration in dark ppl and erythema in white ones. -purulent or non. - more indolent course with localized symptoms over a few days.	-clinical. - Laboratory testing is not required for patients with uncomplicated infection.	-Non purulent cellulitis: managed with empiric therapy with CEFAZOLIN for intravenous therapy and CEPHALEXIN for oral therapy.	-bacterial entry by breaches in skin, involvement of lower extremities unilateral. -deeper dermis and subcutaneous fat. -less demarcated. - most frequently middle-aged and older adults. -high incidence in non-tropical regions with seasonal predilection in warmer months. -Deepening of erythema may be observed following initiation of antimicrobial therapy. -symptomatic improvement within 24 to 48 hours of beginning antimicrobial therapy, more severe cases may take up to 72 hours.
Erysipelas (Cellulitis)	- beta-hemolytic streptococci.	- edema, erythema, warmth, Petechiae and hemorrhage in erythematous skin, and superficial bullae, fever, systemic manifestations, discoloration in dark ppl and erythema in white ones. -non purulent. - acute onset of symptoms with systemic manifestations, fever, chills, severe malaise, headache, raised, advancing border of erythema with central clearing. "butterfly" involvement of the face.	-clinical. - Laboratory testing is not required for patients with uncomplicated infection.		-bacterial entry by breaches in skin, involvement of lower extremities unilateral. -well demarcated. -upper dermis and superficial lymphatics -young children and older adults.

*Predisposing factors associated with risk of **CELLULITIS** and/or **SKIN ABSCESS**:

●Skin barrier disruption due to trauma (such as abrasion, penetrating wound, pressure ulcer, venous leg ulcer, insect bite, injection drug use)

●Skin inflammation (such as eczema, radiation therapy, psoriasis)

●Edema due to impaired lymphatic drainage or due to venous insufficiency

●Obesity

●Immunosuppression (such as 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 in **CELLULITIS** and **ERYSIPELAS** and blood cultures (prior to addition of antibiotic therapy) are warranted:

●Severe local infection (eg, extensive cellulitis)

●Systemic signs of infection (eg, 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)

Necrotizing fasciitis	- polymicrobial (typeI) : anaerobes (most commonly Bacteroides, Clostridium, or Peptostreptococcus) + Enterobacteriaceae (E. coli, Enterobacter, Klebsiella, Proteus) and one or more facultative anaerobic streptococci (Other than group A Streptococcus [GAS]). - Monomicrobial (typeII): GAS, staph.aureus.	-overlying tissue can appear unaffected. - Fournier gangrene: begins abruptly with severe pain and may spread rapidly to the anterior abdominal wall and gluteal muscles. Involvement in men may include the scrotum and penis. - Rapid progression to extensive destruction, systemic toxicity, limb loss, erythema, edema, severe pain, fever, crepitus, skin bullae, necrosis, ecchymosis, warmth hemodynamic instability.	- difficult to diagnose without direct visualization of fascia. -surgical exploration. - intraoperative culture, gram stain. - radiographic imaging for gas in soft tissue.	-early and aggressive surgical exploration and debridement of necrotic tissue, broad-spectrum empiric antibiotic therapy (carbapenem broad spectrum, vancomycin for MRSA, Clindamycin for the antitoxin activity) and hemodynamic support.	-infection of the deep soft tissues that results in progressive destruction of the muscle fascia and overlying subcutaneous fat. -Polymicrobial → near the perineal region (**Fournier gangrene**)→ breach in the integrity of the gastrointestinal or urethral mucosa, Men are more commonly affected than women, mostly in diabetics or immunocompromised patients. - monomicrobal (M protein→ STS syndrome) transmission: hematogenous of GAS from the throat (asymptomatic or symptomatic pharyngitis) to a site of blunt trauma or muscle strain, after skin abrasion. -can occur in healthy individuals with higher risk in: 1. Penetrating trauma (epithelial layer) 2.immunosupression (diabetics, HIV, neutropenia). -acute (hours), subacute (days). -associated with considerable mortality, even with optimal therapy.
clostridial Myonecrosis (clostridial gas gangrene)	- Traumatic gas gangrene C. perfringens, spontaneous gangrene more aerotolerant C. septicum.	-rapidly progressive invasion and destruction of healthy muscle. -pain, systemic toxicity, gas in soft tissue, crepitus (most sensitive and specific finding on clinical examination), rapid spread, swelling, bubbles filled with gas, black areas in xray.	radiographic test, blood culture.	-surgical debridement, antibiotic therapy, supportive measures, tetanus booster shot, HBO. - penicillin, clindamycin, tetracycline, chloramphenicol, metronidazole.	- similar to necrotizing fasciitis, the difference that it always forms gas. -Early recognition and aggressive treatment are essential. -life-threatening muscle infection that develops either contiguously from an area of trauma or hematogenously from the gastrointestinal tract with muscle seeding. -pathogens form endospores + alpha and theta toxins (septic shock). - Traumatic wounds with vascular compromise (deep penetrating injuries such as knife wounds, gunshot wounds, and crush injuries), IDU.

Pyomyositis	Staphylococcus aureus	- fever, pain with cramping localized to a single muscle group. -most often in the lower extremity, less common erythema, swelling. - Stage 1: crampy local muscle pain, swelling, low-grade fever. -Stage 2: 10 to 21 days after the initial onset of symptoms, fever, muscle tenderness, and edema. (Systemic signs, not just localized pain) -Stage 3: systemic toxicity. (all destroyed muscle).	-Radiographic imaging with magnetic resonance imaging is the most useful tool for diagnosis. Bacteriologic diagnosis cultures of drainage specimens and/or blood.	-stage 1: can be treated with antibiotics alone. -most patients present with stage 2 or 3 disease → require both antibiotics and drainage for definitive management.	-purulent infection of skeletal muscle that arises from hematogenous spread, usually with abscess formation (within the muscle). - infection of the tropics but can happen in temperate. -Risk factors: immunodeficiency (HIV), trauma, IDU, concurrent infection, and malnutrition. - Complications of S. aureus bacteremia such as septic shock, endocarditis, septic emboli, pneumonia, pericarditis, septic arthritis, brain abscess, and acute renal failure.

Diabetic foot infections	- Superficial diabetic foot infections → aerobic gram-positive cocci (Staphylococcus aureus, Streptococcus agalactiae, Streptococcus pyogenes, and coagulase-negative staphylococci). - Ulcers that are deep→ the above organisms in addition to enterococci, Enterobacteriaceae, Pseudomonas aeruginosa (common, resistant to many antibiotics), and anaerobes, anaerobic streptococci, Bacteroides species, and Clostridium species.	-extensive local inflammation, necrosis, malodorous drainage, or gangrene with signs of systemic toxicity. -localized superficial skin involvement at the site of a preexisting lesion or as infection of the skin or deeper skin structures that has spread beyond the site of local trauma→ extend to joints, bones, and systemic circulation. -cardinal manifestations of inflammation (erythema, warmth, swelling, and tenderness) and/or the presence of pus in an ulcer or sinus tract.	1) determining the extent and severity of infection, 2) identifying underlying factors, 3) assessing the microbial etiology. -Clinical examination, neurologic evaluation, extent of sensory loss, vascular evaluation. - Obtaining samples for culture, aspirate from an abscess or curettage from the ulcer base.	- Wound management, Surgery for severe and moderate infections, antimicrobial therapy based on severity and involvement.	-mostly start as necrotizing fasciitis. - Risk factors: neuropathy (sensory, motor, autonomic), peripheral vascular diseases, poor glycemic control, immune compromise, hyperglycemia, impaired neutrophil function, neuropathic or ischemic ulcers, traumatic wounds, skin cracks or fissures, or other defects in the skin of the foot or nail beds (paronychia). - the deeper the wound the more microbes contributing. -most infections are polymicrobial. - Osteomyelitis can occur.

Osteomyelitis Acute / Chronic	-**Non hematogenous osteomyelitis:** poly/mono microbial, Staphylococcus aureus (MRSA), coagulase-negative staphylococci (epidermidis), and aerobic gram-negative bacilli (p.aeruginosa). -**Hematogenous osteomyelitis**: monomicrobial, S. aureus, Aerobic gram-negative rods. -Elderly in endemic area→ reactivation of TB. -Diabetic foot (deep infection) → clostridium or p. aeruginosa.	**Acute osteomyelitis**: gradual onset of symptoms over several days, pain at the involved site, with or without movement, Local findings (tenderness, warmth, erythema, and swelling) and systemic symptoms (fever, rigors). **Chronic osteomyelitis:** pain, erythema, or swelling, sometimes in association with a draining sinus tract, fever is usually absent, intermittent flares of pain and swelling. The presence of a sinus tract is pathognomonic of chronic osteomyelitis.	-clinical + radiograph + positive blood culture→ not need for bone biopsy unless there is debridement. -histopathology→ negative blood culture with recent AB therapy. -clinical + radiograph + persistently elevated inflammatory Markers→ negative blood + impossible biopsy.	- if blood culture fails we give treatment against the most common pathogen of vertebral osteomyelitis, including staphylococci, streptococci, and gram-negative bacilli like vancomycin.	-infection involving bone and bone marrow. -**Acute osteomyelitis:** evolves over several days to weeks and can progress to a chronic infection. -**chronic osteomyelitis:** presence of dead bone (sequestrum), involucrum (reactive bony encasement of the sequestrum), local bone loss, and, if there is extension through cortical bone, sinus tracts. – **Non hematogenous osteomyelitis:** contiguous spread of infection to bone from adjacent soft tissues and joints or via direct inoculation of infection into the bone (trauma or surgery), younger adults (trauma/surgery), older adults (adjacent tissue), Risk factors: poorly healing soft tissue wounds, presence of orthopedic hardware, diabetes, peripheral vascular disease, and peripheral neuropathy. **-Hematogenous osteomyelitis:** microorganisms that seed the bone in the setting of bacteremia, infants and children. -attach to bone (matrix components)→ infection→ inflammation→ cut Blood supply→ death of bone→ formation of involucrum. -**vertebral osteomyelitis:** adults (>50 years), most common form of hematogenous osteomyelitis. -**Tuberculous osteomyelitis:** reactivation of tuberculous bacilli lodged in bone during the mycobacteremia. -large inoculation of organisms, presence of bone damage, and/or presence of hardware or other foreign material. -antibiotic failure: abscess, sequestrum, biofilm, intracellular (osteoblasts, clasts, macrophages). -small colony variants: AB resistance, low metabolic activity. -Patients with osteomyelitis involving the hip, vertebrae, or pelvis tend to manifest few signs or symptoms other than pain. –Complications: Sinus tract formation, Contiguous soft tissue infection, Abscess, Septic arthritis, Systemic infection, Bony deformity and Fracture, Malignancy.

acute bacterial arthritis / Septic arthritis	-by bacteria (destructive acute arthritis) or other microorganisms. – usually monomicrobial, S. aureus (MRSA) is the most common cause of septic arthritis in adults. Other gram-positives, streptococci are also important potential. -staph.aureus, strept. in splenic dysfunction, N.gonorrhoeae in sexually active pts, mycobacteria, spirochetes (borellia burgdorferi).	-acutely with a single swollen and painful joint (monoarticular arthritis), Joint pain, swelling, warmth with redness and erythema above it which is the site of trauma, restricted movement, febrile (afebrile adults),	-suspected in patients with acute onset of at least one swollen, painful joint, with or without relevant risk factors. --synovial fluid analysis and culture. -blood culture, radiographs ultrasound, imaging studies. -collection of blood sample and synovial fluid should always be prior to AB administration.	-joint drainage (in adults needle aspiration, arthroscopic drainage, or arthrotomy) and antibiotic therapy. -positive culture to G+→ vancomycin (MRSA). -positive culture to G-→ pseudomonas.	-infection in joints, hematogenous seeding (Bacteremia)→more likely to localize in a joint with pre-existing arthritis (RA, osteoarthritis, gout, pseudogout, Charcot arthropathy), particularly if associated with synovitis, direct inoculation of bacteria into the joint, or contiguous spread from an adjacent soft tissue or bone infection. -The knee is involved in more than 50 percent of cases; wrists, ankles, and hips. -adverse prognostic factors: older age and pre-existing joint disease.
Animal Bites	-oral flora of biting animal (Mixed aerobes, anaerobes 60 percent of cases), skin flora (40 percent of cases). -staphylococci streptococci. -Pasteurella: 50% dogs bite, 75% of cats bite. -Capnocytophaga canimorsus: cause bacteremia, fatal sepsis after animal bites, in patients with asplenia, alcoholism, hepatic disease. -B. henselae: bite of infected cat, cat scratches, flea exposure, contact with cat saliva via broken skin or mucosal surfaces. The incubation period is 7 to 14 days **(cat scratch disease).** -Anaerobes: Bacteroides, fusobacteria, Porphyromonas species, Prevotella species.	-superficial: fever, erythema, swelling, warmth, purulent drainage, and/or lymphangitis. associated superficial abscess present as tender, erythematous, fluctuant nodule.	physical examination should ensure that the patient is hemodynamically stable + asses injuries to adjacent structures + evaluated carefully for foreign material, neurovascular assessment should be performed in areas distal to the wound.		-dog bites→ rabies. -Infections are much more common after cat bites (up to 50 percent of wounds) than dog bites. -Cat bites usually occur on the extremities and tend to penetrate deeply, with higher risk of deep infection (abscess, septic arthritis, osteomyelitis, tenosynovitis, bacteremia, or necrotizing soft tissue infection) than dog bites. -superficial (cellulitis, with or without abscess) or deep (Abscess, septic arthritis, osteomyelitis, tenosynovitis, or necrotizing soft tissue infection).