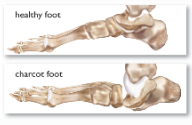
MSS | lec. 5 micro sum.
Done by: Ola Alahdab

Diabetic foot infections

any infection in a patient with DM that is bellow the malleolusكعب القدم.
- **diabetic ulcer** is the commonest.
- these infections range from: Paronychiaالتهاب حول الظفر/داحس - cellulitis - myositis - abscess formation - necrotizing fasciitis - septic arthritis - tendonitis - osteomyelitis

Risk factors:

• **Neural**: development of peripheral sensory, motor, and/or autonomic neuropathy, neuro-osteopathic deformity (e.g. Charcot joint),



• **Vascular**: vascular insufficiency
• **Immune**: hyperglycaemia leading to poor immune function and wound healing
• **Other factors:** poor vision, limited mobility, previous amputationsبتر, poor healthcare

Clinical features: (mild ➡️ severe ➡️ life-threatening)

➡️no signs
➡️ surrounding inflammation or cellulitis <2cm from edge of the wound
➡️ Local complications:

cellulitis > 2cm from edge of wound
lymphangitis, spread beneath the superficial fascia
deep tissue abscess
gas gangrene
involvement of muscle, tendon, or bone.
➡️systemic toxicity—fever, chills, tachycardia, hypotension, confusion, vomiting, leucocytosis, acidosis, hyperglycaemia, uraemia.

Diagnosis:

1-Clinically
2-Doppler ultrasound (to determine ratio of ankle Vs brachial pressure index (aBPIs)).
3-Imaging (MRI)
4-Deep tissue specimens (not superficial swabs).

Management: (Inpatient Vs outpatient)

• Inpatient Rx is based on correcting systemic instability

non infected ulcers: Do not give antibiotics.

initial infected ulcers:empiric therapy Oral for mild cases & IV brad spectrum for severe cases

sever infections in deeper tissues (necrotizing fasciitis, gas gangrene, extensive tissue loss, critical limb ischemia): Surgery

An obese 50-year-old man with no known medical history presented with a necrotizing infection of his right foot that had begun 10 days previously with lesions that he attributed to wearing new shoes. He was found to have diabetes (glycated hemoglobin level, 10.5%) with peripheral neuropathy; he was afebrile, without leukocytosis or radiographic evidence of bone involvement in his right foot. The patient had photographed the lesion twice daily, thinking it would heal spontaneously (Panel A). The preoperative photographs show erythema (day 1), blisters (day 3), a necrotizing abscess (day 6), and wound infection requiring surgery (day 10). The patient underwent operative débridement; tissue cultures grew *Enterobacter cloacae* and *Streptococcus agalactiae*. He was treated with antibiotic agents for 3 weeks. The infection resolved, with no recurrence or sequelae during 3 years of follow-up (Panel B); during this period, the infection-related swelling disappeared and the patient lost a considerable amount of weight. Diabetic foot infection may evolve rapidly, especially in patients with neuropathy.

Table 22.1 Aetiology of diabetic foot infections

Foot infection syndrome	Pathogens
Cellulitis	β-haemolytic streptococci (groups A, B, C, and G), *S. aureus*
Infected ulcer, antibiotic-naïve	Often monomicrobial: *S. aureus* or β-haemolytic streptococci (groups A, B, C, and G)
Infected ulcer, chronic, previous antibiotic therapy	Usually polymicrobial: *S. aureus*, β-haemolytic streptococci (groups A, B, C, and G), *Enterobacteriaceae*
Macerated ulcer	*P. aeruginosa* ± other organisms as above
Long-standing, non-healing wound, prolonged antibiotic therapy	Usually polymicrobial with antibiotic-resistant organisms: aerobic Gram-positive cocci (*S. aureus*, CoNS, enterococci), diphtheroids, *Enterobacteriaceae*, *Pseudomonas* spp., non-fermentative GNRs, fungi
'Fetid foot': extensive necrosis or gangrene	Mixed aerobic Gram-positive cocci (*S. aureus*, CoNS, enterococci), *Enterobacteriaceae*, non-fermentative GNRs, obligate anaerobes

'apular and nodular lesions

Fish-tank or swimming-pool granuloma	*Mycobacterium marinum*
Creeping eruption (cutaneous larva migrans)	*Ancylostoma braziliense*
Dracunculiasis	*Dracunculus medinensis*
Cercarial dermatitis	*Schistosoma mansoni*
Verruca vulgaris	Human papillomaviruses 1, 2, 4
Condylomata acuminata (anogenital warts)	Human papillomaviruses 6, 11, 16, 18
Onchocerciasis nodule	*Onchocerca volvulus*
Cutaneous myiasis	*Dermatobia hominis*
Verruca peruana	*Bartonella bacilliformis*
Cat-scratch disease	*Bartonella henselae*
Lepromatous leprosy	*Mycobacterium leprae*
Secondary syphilis (papulosquamous and nodular lesions, condylomata lata)	*Treponema pallidum*
Tertiary syphilis (nodular gummatous lesions)	*T. pallidum*

Papular and Nodular Lesions: Mycobacterium marinum

marine = بحري

Infections of the skin may present as cellulitis or as raised erythematous nodules.

• In aquarium cleaners, fishermen, seafood handlers.

• Organism growth requires lower temperatures than 37C (24-32) and thus is limited only to skin

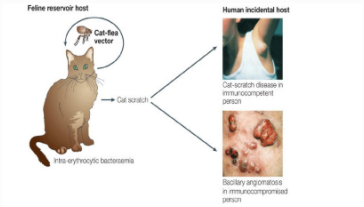
• **Diagnosis**: needle aspiration, acid-fast bacilli

• **Treatment**: rifampin+ethambutol for four months

Cat-scratch disease

Cat

Caused by: Bartonella henselae, G -ve bacillus, grows on Columbia agar supplemented with 5% sheep blood



• **Transmit** between cat and flea, then cats to humans by bite or scratch.

- **Symptoms** : myalgia ألم عضلي, arthlagia, malaise,

anorexia, low grade fever.

- **Signs**:

PP LOVE

- lesions (papule, pustule or large vesicles) developing at the primary site of inoculation of Bartonella henselae.

- painful regional ipsilateral بنفس الجانب Lymph adenopathy.

•**Diagnosis**: Serology (IgM, or IgG titers) or biopsy of lymph node

•**Treatment**: self limited.

there is increased risk of reaction if Abx given in first 48 hours

Schistosomiasis

Caused by Schistosoma; a parasitic blood fluke (trematodes, life cycle in two hosts).

• **Multiple erythematous papules** develop in schistosomiasis; each represents a cercarial invasion site (known as bilharzia).

• Acute phase (Katayama fever, whole body hypersensivity-fever, malaise..etc)

• **Diagnosis**: stool and urine microscopy (shows the presence of eggs)

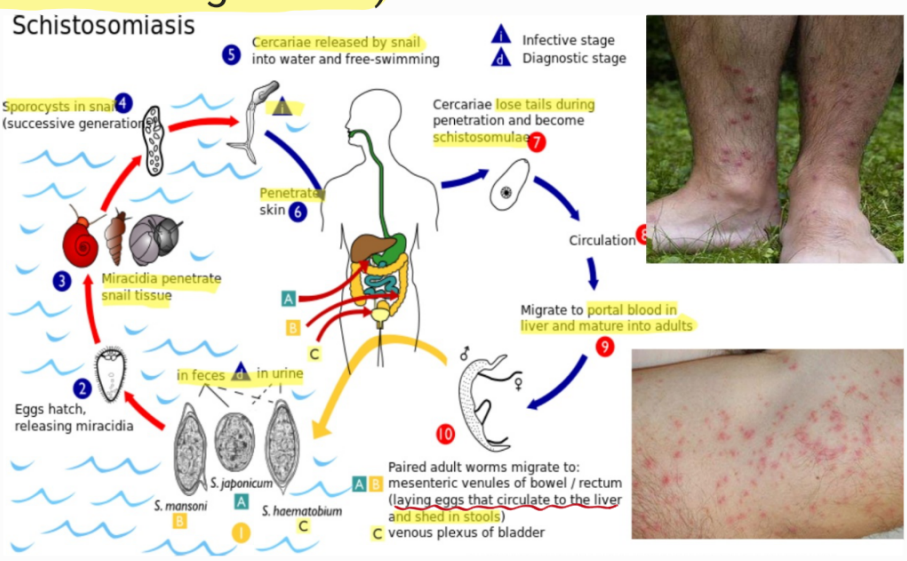
• **Treatment**: single dose Praziquantel (antiparasitic)

• **Skin signs:** especially in S.cercariae but other parasites as well (called swimmers' itch) due to allergic reaction at site of invasion by parasites Prognosis.

• More advanced stages with hepatic and urinary disease improve months or years treatment (even if fibrosis occurs).

• Renal, intestinal, & brain lesions pathology improves with treatment.

• Hepatosplenic schistosomiasis carries a relatively good prognosis because hepatic function is preserved until the end of the disease (unless variceal bleeding occurs).

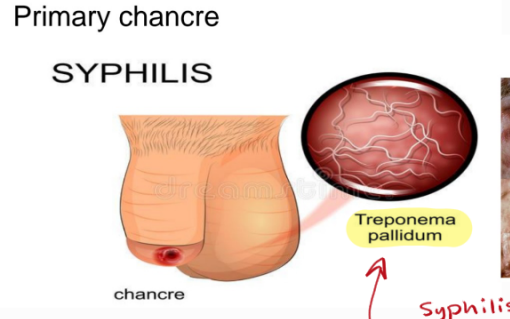


Leprosy (lepromatous leprosy)

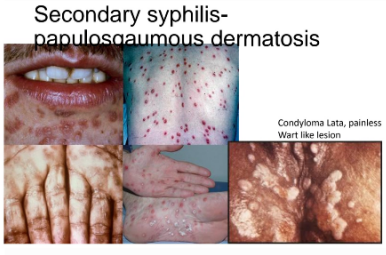
- Skin nodules & thickened subcutaneous tissue.
- **caused by** Mycobacterium leprae (acid fast bacillus).
• Chronic infection of the skin, causes granulomas at the body aims to contain the bacilli.
• Long incubation (years, 3-upto 20 years)
• **Transmitted by** nasal secretion.
• Has **two types** :
1. tuberculoid: patient has intact immunity, nerve changes predominate.
2. lepromoatous: defect in cell immunity, skin changes predominate.
• **Skin changes**: bilateral symmetrical **macules** and **papules** , progresses to **nodules** and even **plaques**. ma pa no pla
• hypopigmented in dark skinned people and seen on face, wrists, buttocks and kneed, but spares groin and axilla (folded skin) has higher temp which doesn't favor its growth.
• **Diagnosis**: acid fast stain, skin smears and biopsy (stain especially +ve in lepromatous leprosy).
• **Treatment**: Dapsone and Rifampin (anti mycobacterial drugs).

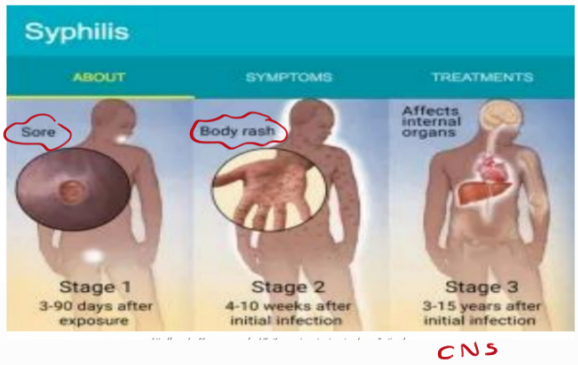
Syphilis

-Large **nodules** or gummas are features of tertiary syphilis (late untreated syphilis)
-Flat papulosquamous lesions are characteristic of secondary syphilis.



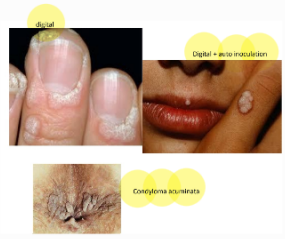
Syphilis is caused by:





CNS involvement

Human Papilloma virus:



- causes:

1. verruca vulgaris: singular warts بثور by HPV 1,2,4,7 in children and young adults
2. condylomata acuminata: multiple warts in the anogenital area (a major problem in HIV patients)

- Transmitted by skin contact.
- Usually on the hands and nail edges, can auto inoculate face
- Management : Dermatologists
- HPV is an STD, and can cause cervical cancer (16, 18, 31, 33..)

Sexually transmitted disease

Diagnosis of skin infections:

depends on three main clinical observation:

- Appearance of lesion (macule, papule, vesicle, etc)
- Location within the layers of soft tissues (stratum corneum, epidermis, dermis, etc)
- Location relative to the body (trunk, face, extremities, etc)

- information that helps fine tune the pathogenesis of the infective agent are :

- The temporal progression of the lesions (e.g. appear in crops, or appear acutely..etc)
- the patient's travel history (for exogenous or exotic sources of pathogens)
- animal exposure or bite history
- age of patient
- underlying disease status,
- lifestyle

Aiding diagnostic tests: for defining a localized abscess or detecting gas in tissue

1- Soft tissue radiography, CT, and MRI: determine the depth of infection (helps in assessing deeper infections).

- eg: serious ones like fasciitis are rapidly progressing

*Evidence of a systemic inflammatory response syndrome (to find a local infection that may be releasing toxins).

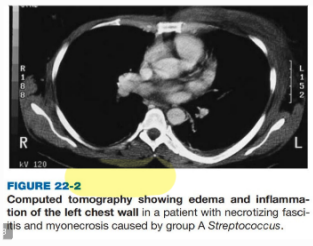


FIGURE 22-2
Computed tomography showing edema and inflammation of the left chest wall in a patient with necrotizing fasciitis and myonecrosis caused by group A *Streptococcus*.

2- lab diagnostics

- Aspiration (without saline better ➡️ less dilution) or punch biopsy with frozen section might help (if imaging is positive. -There is a large false negative rate ~ 80%-).

• Frozen sections are especially useful in distinguishing SSSS from TEN, also valuable in cases of necrotizing fasciitis (determining depth and level of involvement).
• Open surgical inspection (+debridement) is the optimal way to determine the extent and severity of infection, also to obtain specimen for culture and Gram stain. Although surgical approach maybe aggressive, it is important and maybe lifesaving in the course of **fulminant infections** where there is evidence of **systemic toxicity.**

Treatment, overview:

• Furuncles, carbuncles, and abscesses caused by MRSA and MSSA are commonly encountered, Rx here depends on the **size** of the lesion:
- Furuncles < 2.5 cm in diameter: treated with **moist heat.**
- Furuncles > 4.5 cm of erythema + induration: **surgical** drainage
- larger lesions + fever, chills, or leukocytosis: **drainage + antibiotics.**
- A study in children showed that surgical drainage of abscesses alone (mean diameter: 3.8 cm) was as effective as when combined with trimethoprim-sulfamethoxazole treatment.
- However, recurence was less in groups that added Abx with I&D.

TREATMENT OF COMMON INFECTIONS OF THE SKIN

DIAGNOSIS/CONDITION	PRIMARY TREATMENT	ALTERNATIVE TREATMENT	SEE ALSO CHAP(S).
Animal bite (prophylaxis or early infection)[a]	Amoxicillin/clavulanate, 875/22 mg PO bid	Doxycycline, 100 mg PO bid	35
Animal bite[a] (established infection)	Ampicillin/sulbactam, 1.5–3 g IV q6h	C Clindamycin, 600–900 mg IV q8h, *plus* C Ciprofloxacin, 400 mg IV q12h, *or* C Cefoxitin, 2 g IV q6h	35
Bacillary angiomatosis	Erythromycin, 500 mg PO qid	Doxycycline, 100 mg PO bid	65
Herpes simplex (primary genital) Virus	Acyclovir, 400 mg PO tid for 10 days	Famciclovir, 250 mg PO tid for 5–10 days, *or* Valacyclovir, 1000 mg PO bid for 10 days	84
Herpes zoster (immunocompetent host >50 years of age) Virus	Acyclovir, 800 mg PO 5 times daily for 7–10 days	Famciclovir, 500 mg PO tid for 7–10 days, *or* Valacyclovir, 1000 mg PO tid for 7 days	85
Cellulitis (staphylococcal or streptococcal[b,c]) Strong	Nafcillin or oxacillin, 2 g IV q4–6h	Cefazolin, 1–2 g q8h, *or* Ampicillin/sulbactam, 1.5–3 g IV q6h, *or* Erythromycin, 0.5–1 g IV q6h, *or* Clindamycin, 600–900 mg IV q8h	38, 39
MRSA skin infection[d] Strong	Vancomycin, 1 g IV q12h	Linezolid, 600 mg IV q12h	38
Necrotizing fasciitis (group A streptococcal[b])	Clindamycin, 600–900 mg IV q6–8h, *plus* Penicillin G, 4 million units IV q4h	Clindamycin, 600–900 mg IV q6–8h, *plus* Cephalosporin (first- or second-generation)	39
Necrotizing fasciitis (mixed aerobes and anaerobes) ACC	Ampicillin, 2 g IV q4h, *plus* Clindamycin, 600–900 mg IV q6–8h, *plus* Ciprofloxacin, 400 mg IV q6–8h	Vancomycin, 1 g IV q6h, *plus* Metronidazole, 500 mg IV q6h, *plus* Ciprofloxacin, 400 mg IV q6–8h	69
Gas gangrene	Clindamycin, 600–900 mg IV q6–8h, *plus* Penicillin G, 4 million units IV q4–6h	Clindamycin, 600–900 mg IV q6–8h, *plus* Cefoxitin, 2 g IV q6h	46

Myositis and myonecrosis	
Pyomyositis → Pus	*S. aureus*
Streptococcal necrotizing myositis	*S. pyogenes*
Gas gangrene	*Clostridium* spp.
Nonclostridial (crepitant) myositis	Mixed aerobic and anaerobic bacteria
Synergistic nonclostridial anaerobic myonecrosis	Mixed aerobic and anaerobic bacteria

Myositis and Myonecrosis

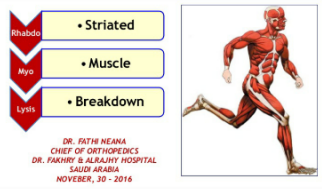
Muscle involvement (inflammation or infection) can occur with:

- viral infection (such as influenza, dengue, or coxsackievirus B infection)
- Parasitic invasion (trichinellosis, cysticercosis, or toxoplasmosis).
- Myalgia (muscle pain) can occur in most of these infections,

severe muscle pain is the hallmark of pleurodynia (caused by **coxsackievirus B** & called Bornholm disease أَلَمٌ عَضَلِيّ وَبَائِي), trichinellosis, and bacterial infection.

>Acute rhabdomyolysis انحلال الربيدات أو انحلال العضلات المخططة الهيكلية (breakdown of damaged muscle) predictably occurs with **clostridial** and **streptococcal myositis** (both these organism have enzymes that breakdown muscle).

Rhabdomyolysis is less so associated with influenza virus, echovirus, coxsackievirus, Epstein Barr virus, and Legionella infections



Necrotizing myositis

- S. pyogenes (GAS) may induce primary myositis (referred to as streptococcal **necrotizing** myositis) in association with severe systemic toxicity
- this is basically necrotizing fasciitis (Type2) that involves the muscle tissue.
- Myonecrosis occurs in about 50% of cases in typical necrotizing fasciitis without muscle involvement being the primary tissue infected! (meaning that **muscles are affected by necrosis when the surrounding fascia is involved, even without the infection being active in the muscle itself**!)

pyomyositis

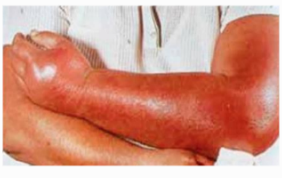
Pus

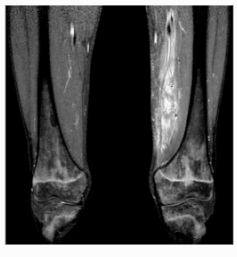
- Pyomyositis, or pus forming infection of muscles, is usually due to S. aureus (remember it is the typical pus former in skin)
- common in tropical areas, and generally has no known portal of entry (in contrast to necrotizing fasciitis).
- pyomyositis caused **by MRSA producing the PVL toxin** have been described among children in the United States.
- Muscle infection begins at the exact site of blunt trauma or muscle strain.

• a purulent -PUS forming- infection of skeletal muscle.
• Abscess formation is the usual consequence when these pyogenic bacteria reach the muscle tissue.
• usually arises from haematogenous spread (deeper infections (muscle and bone) are typically more related with haematogenous spread rather direct inoculation).
-usually remains localized and shock does not develop unless organisms produce:
• 1- **toxic shock syndrome toxin 1** (a super antigen, causes exaggerated immune response that is many fold the normal response: **shock**).
• 2- **enterotoxins** (exotoxins produced by S. aureus).
• If the patient lacks antibodies to the toxins above then they are prone to developing toxic shock when these toxins are produced.

Epidemiology

• Occurs in **M>F**, and more in tropical climate in two main age groups:
1. **children** (aged 2–5 years) 2. **adults** (aged 20–45 years).
- In temperate climes:
- pyomyositis typically affects **adults or the elderly** (not children).
- Patients usually have predisposing conditions such as HIV, infection, DM, malignancy, cirrhosis, renal insufficiency, organ transplantation (reduced cell immunity), immunosuppressive therapy.
- Other risk factors include trauma, IDU (injection drug use), and concurrent infections (toxocariasis –roundworms-, VZV).





Microbiology

• S. aureus 90% of tropical cases 75% of temperate cases.
• GAS account for 1–5% of cases all around.
• E. coli ST131 is an emerging cause in patients with haematological malignancy.
• Uncommon causes B, C, and G streptococci and S. pneumoniae, and S. anginosus.
• Rare causes include Enterobacteriaceae, Y. enterocolitica, N. gonorrhoeae, H. influenzae, A. hydrophila, anaerobes, B. mallei, B. pseudomallei, A. fumigatus, Candida spp., MTB(Mycobacterium tuberculosis), and MAC (Mycobacterium avium).

Clinical features

• 20% and 50% of cases patient have had recent blunt trauma or vigorous قوي exercise of the affected area →myolysis← (The muscle area is damaged and becomes susceptible for infections).
• Seen more in the lower extremity (thigh, calf, gluteal muscles), but not limited to that area and can affect any muscle group.
• Multifocal infection عدوى متعددة البؤر occurs in up to 20% of cases!
• Since it is usually from a hematologic cause, the patient must be assessed for complications of bacteraemia (endocarditis).

clinical stages:

• **Stage 1** (early invasive stage): crampy local muscle pain, swelling, and lowgrade fever. Induration(hardening)of the affected muscle + leucocytosis may be present.
• **Stage 2** (suppurative stage) at 10–21 days after onset of symptoms (most patients present at this stage): fever, very sharp muscle tenderness and swelling. An abscess may be clinically apparent, aspiration of which yields pus. There is marked leucocytosis.
• **Stage 3** (systemic stage) the affected muscle is fluctuantمتقلب. Patients may present with complications of S. aureus bacteraemia, e.g. septic shock, endocarditis, septic emboli, pneumonia, pericarditis, septic arthritis, brain abscess, and ARF (acute renal failure). Rhabdomyolysis may occur.

Diagnosis

• Early pyomyositis is difficult to distinguish from other Dx (thrombophlebitis, muscle haematoma, muscle rupture, fever of unknown origin osteomyelitis).
• Iliacus pyomyositis may mimic septic arthritis of the hip, and iliopsoas pyomyositis may mimic appendicitis.
• **Imaging**:
• MRI is gold standard technique (may show muscle enhancement and intramuscular abscesses).
• CT (may detect muscle swelling and well defined abscesses).
• Ultrasound can be helpful for Dx and Rx
• **Microbiology:**
- diagnostic aspirates before starting Abx to get a specific culture
- BCs are only positive in 10% of tropical cases and 35% of temperate cases
- Pyomyositis inner thigh in young patients with severe aplastic anemia (E.coli)

Management

• Antibiotics: **stage 1:** antibiotics alone.
stage 2/3: antibiotics + drainage.
• Empiric therapy:
• S. aureus and streptococci: (**flucloxacillin or vancomycin**, if MRSA is suspected or there is a risk of MRSA).
• Immunocompromised patients: broader Abx such as **piperacillin–tazobactam ± vancomycin.**
• Once culture is out: Tailored Abx for 3-4 weeks
• **Drainage**: percutaneous drainage (drainage and send drain sample for Micro).
This may be CT-guided or ultrasound-guided.