

Musculoskeletal system lecture 5

Erysipelas

Erysipelas

S. pyogenes

- Erysipelas is due to *S. pyogenes* and is characterized by an abrupt onset of fiery-red swelling of the face or extremities.
- The distinctive features of erysipelas are well-defined indurated margins, particularly along the nasolabial fold; rapid progression; and intense pain (inflammation, acute).
- Flaccid bullae may develop during the second or third day of illness, but extension to deeper soft tissues is rare.
- Treatment : penicillin(flucloxacillin, clindamycin) is effective
- swelling may progress despite appropriate treatment, although fever, pain, and the intense red color diminish.
- Desquamation of the involved skin occurs 5–10 days into the illness.
- Infants and elderly adults are most commonly afflicted, and the severity of systemic toxicity varies.

Cutaneous abscesses

- • Collections of **pus** within the dermis and deeper skin structures.
- • Usually **polymicrobial** containing **skin/mucous membrane flora**; *S. aureus* is the sole pathogen in 25% of cases.
- • Clinical features—**painful, tender, fluctuant nodules**, usually with an **overlying pustule and surrounded by a rim of erythematous swelling**.
- • **Treatment is I&D** → Antibiotics are **rarely necessary** (except in extensive infection or systemic toxicity, or immunocompromised).

Notice:

raised lesion,
White head
Hair follicle might
Be port of entry



Diabetic foot infections

- Defined as any infection in a patient with DM that is below the malleolus.
- The commonest of these lesions is an infected diabetic ulcer
- However, as mentioned it is a broad spectrum of infections ranging from:

Paronychia - cellulitis - myositis

abscess formation - necrotizing fasciitis

septic arthritis - tendonitis - osteomyelitis.

Epidemiology

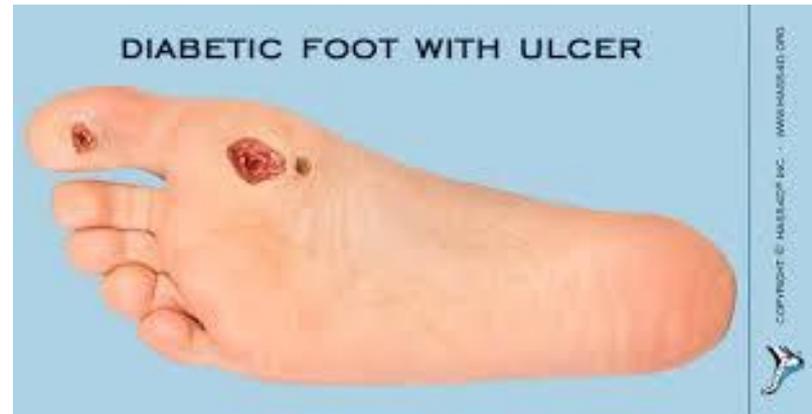
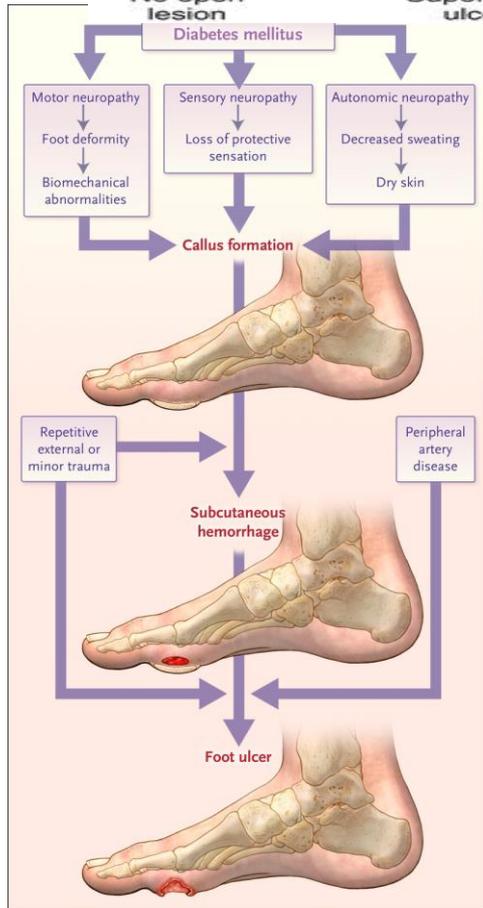
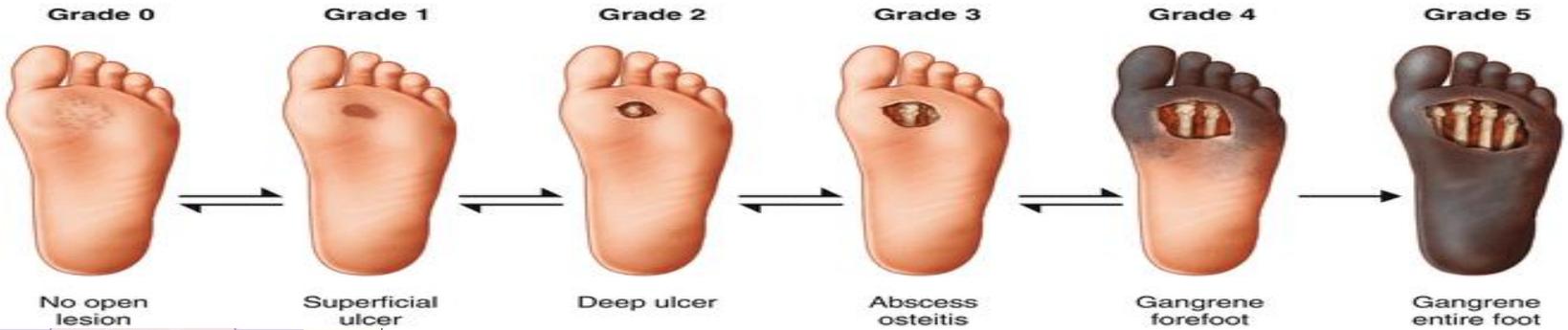
- Foot infections in DM are a **common complication**, they often carry a **high burden of morbidity** and are **notoriously difficult to manage** (there are DF clinics!)
- Risk factors: *recall table from last lec.*
- **Neural** → development of **peripheral sensory, motor, and/or autonomic neuropathy, 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 amputations, poor healthcare*

Etiology

Table 22.1 Aetiology of diabetic foot infections

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

Dysvascular foot breakdown - Natural history





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.

Clinical features

- Range from mild → severe → life-threatening
- As the following
 - foot ulcer with no signs of infection
 - foot ulcer with surrounding inflammation or cellulitis <2cm from the 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 or metabolic instability—fever, chills, tachycardia, hypotension, confusion, vomiting, leucocytosis, acidosis, hyperglycaemia, uraemia.

Diagnosis

- **1-Clinical features** : must assess **perfusion** (peripheral pulses, less perfusion = more anaerobes), as well as sensation (using a mono filament).
- 2-Doppler ultrasound to determine ratio of ankle Vs brachial pressure index (aBPIs).
- **3-Imaging (MRI)** may help determine the extent of infection (osteomyelitis, fasciitis).
- *4-Deep tissue specimens (not superficial swabs) need to be sent to microbiology lab for microscopy and culture before Abx treatment.*

Management

- • Inpatient Vs outpatient management.
- Inpatient Rx is based on correcting systemic instability
- I- Medical treatment:
 - Do not give Abx for non infected ulcers, initial infected ulcers → empiric therapy Oral for mild cases and IV broad spectrum for severe cases
- II- Surgery—severe infections in deeper tissues (necrotizing fasciitis, gas gangrene, extensive tissue loss, critical limb ischemia)
- Wound care plan following discharge

Papular and Nodular Lesions

Papular 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

- *Mycobacterium marinum*

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

- Occupational hazard, usually for aquarium cleaners, fishermen, seafood handlers.

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

- Dx: needle aspiration , see acid fast bacilli

- Rx: rifampin+ethambutol for four months



Cat-scratch disease

- *Bartonella henselae* : Gram negative bacillus, usually grows on Columbia agar supplemented with 5% sheep blood
- Transmission cycle between cat and flea, then cats transmit to humans by bite or scratch
- Symptoms : myalgia, arthralgia, malaise, anorexia, maybe low grade fever

- **Signs:**

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

Then in 85-90% of cases persistent painful regional **IPSILATERAL Lymph adenopathy**

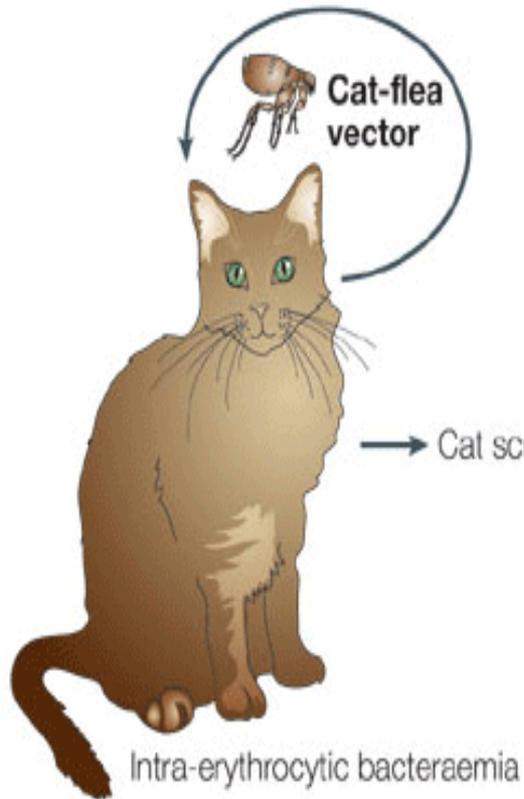
- **Dx:**

Serology (IgM, or IgG titers) or biopsy of lymph node

- **Rx:**

self limited in immunocompetent, resolves **within 8 weeks**, there is increased risk of **reaction if Abx given in first 48 hours**

Feline reservoir host



Human incidental host



Cat-scratch disease in immunocompetent person



Bacillary angiomatosis in immunocompromised person

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 hypersensitivity-fever malaise..etc)
- Dx
stool and urine microscopy (shows the presence of eggs)
- Rx:
single dose Praziquantel (antiparasitic)
- Skin signs:
esp in *S. cercariae* but other parasites as well (called swimmers' itch) due to allergic reaction at site of invasion by parasites

Schistosomiasis

▲ Infective stage
 ▲ Diagnostic stage

5 Cercariae released by snail into water and free-swimming

4 Sporocysts in snail (successive generation)

Cercariae lose tails during penetration and become schistosomulae

6 Penetrates skin

8 Circulation

9 Migrate to portal blood in liver and mature into adults

2 Eggs hatch, releasing miracidia

in feces ▲ in urine

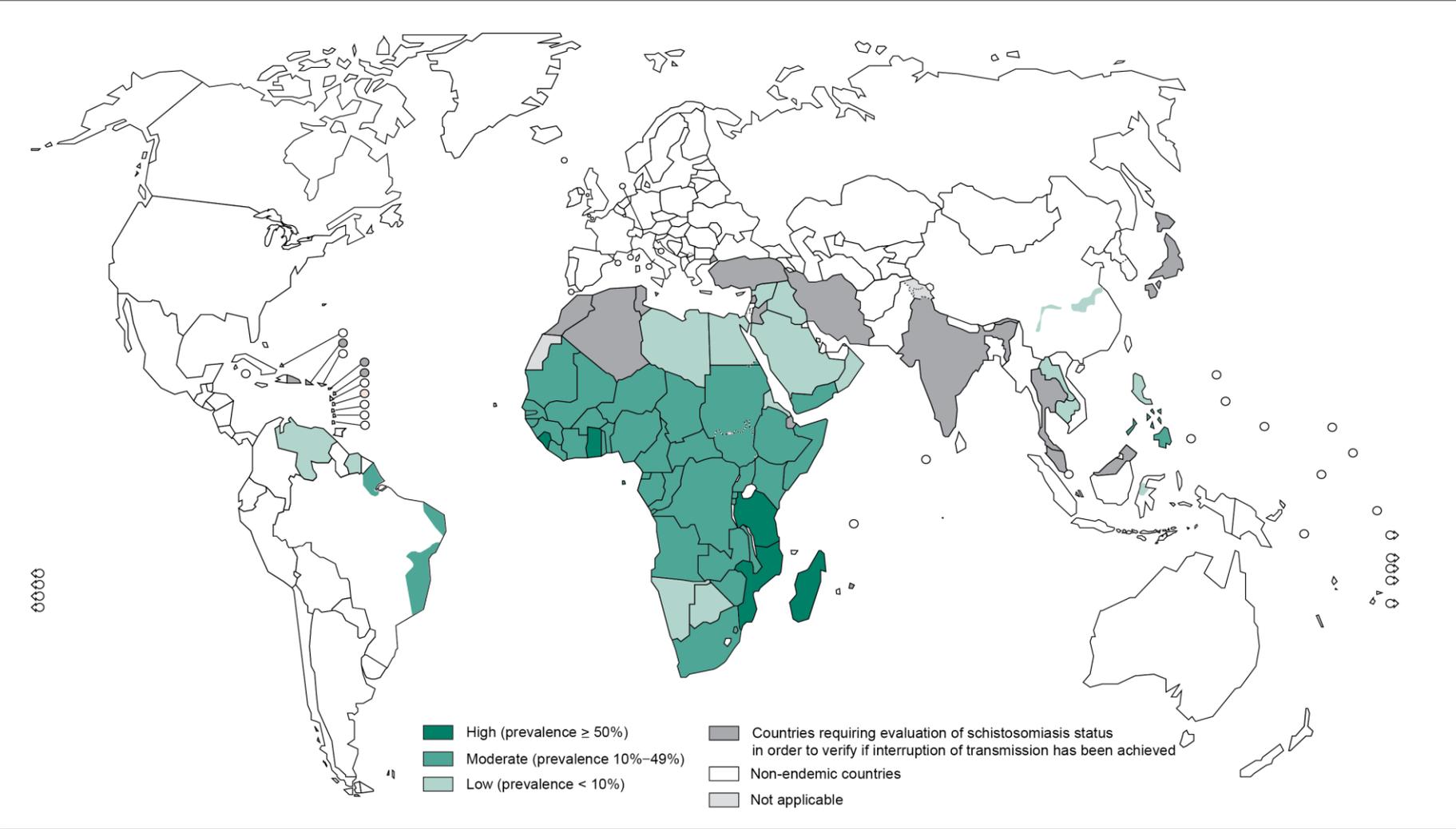
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Paired adult worms migrate to:
 A B mesenteric venules of bowel / rectum (laying eggs that circulate to the liver and shed in stools)
 C venous plexus of bladder

S. mansoni B
S. japonicum A
S. haematobium C



Distribution of schistosomiasis, worldwide, 2012



The boundaries and names shown and the designations used on this map do not imply the expression of any opinion whatsoever on the part of the World Health Organization concerning the legal status of any country, territory, city or area or of its authorities, or concerning the delimitation of its frontiers or boundaries. Dotted lines on maps represent approximate border lines for which there may not yet be full agreement. © WHO 2014. All rights reserved

Data Source: World Health Organization
 Map Production: Control of Neglected Tropical Diseases (NTD)
 World Health Organization



Prognosis

- Early disease usually improves with treatment.
- More advanced stages with hepatic and urinary disease improve following a long term therapy over months or years (even if fibrosis occurs)
- Renal and intestinal pathology also improves with treatment, as, usually, do brain lesions (depending on their location and size). ^[29]
- Hepatosplenic *schistosomiasis* carries a relatively good prognosis because hepatic function is preserved until the end of the disease (unless variceal bleeding occurs).

Leprosy

- Skin nodules as well as thickened subcutaneous tissue are prominent features of lepromatous leprosy.
- Leprosy is 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)
- Transmission mostly by nasal secretion of Untreated patients, however low risk from casual contact is present.
- Has two types : tuberculoid → patient has intact immunity, usually nerve changes predominate

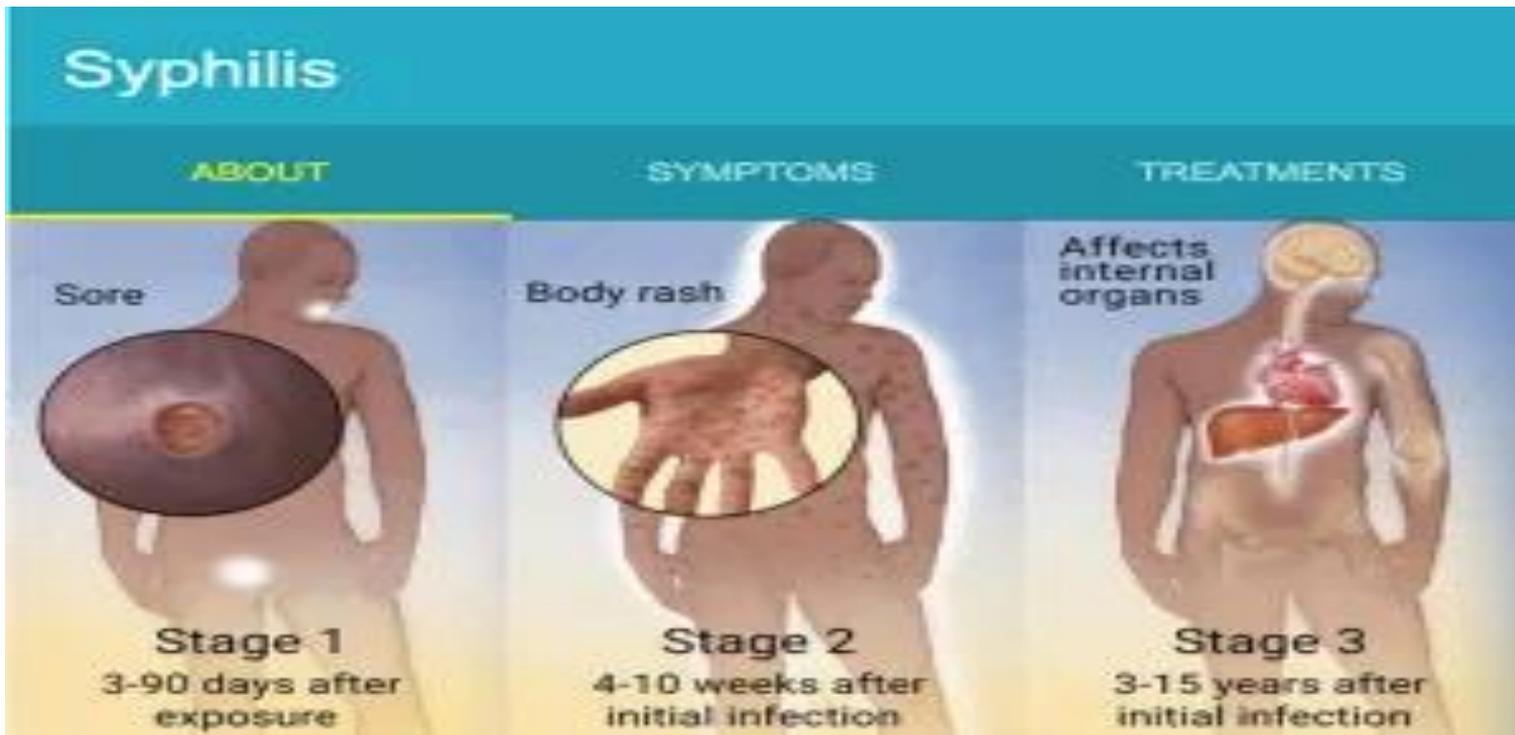
- Second type: is lepromatous where skin changes predominate (defect in cell immunity)
- Skin changes : bilateral symmetrical macules and papules , progresses to nodules and even plaques.
- Usually hypopigmented in dark skinned people and seen on face, wrists , buttocks and kneed, but spares groin and axilla (folded skin has higher temp which doesn't favor its growth)
- Dx: acid fast stain skin smears and biopsy (stain especially +ve in lepromatous leprosy).
- Rx: anti mycobacterial drugs (Dapsone and Rifampin), treatment at early stages is for 1-2 years, in leprosy treatment maybe lifelong.



Lepromatous Leprosy: Leonine Face

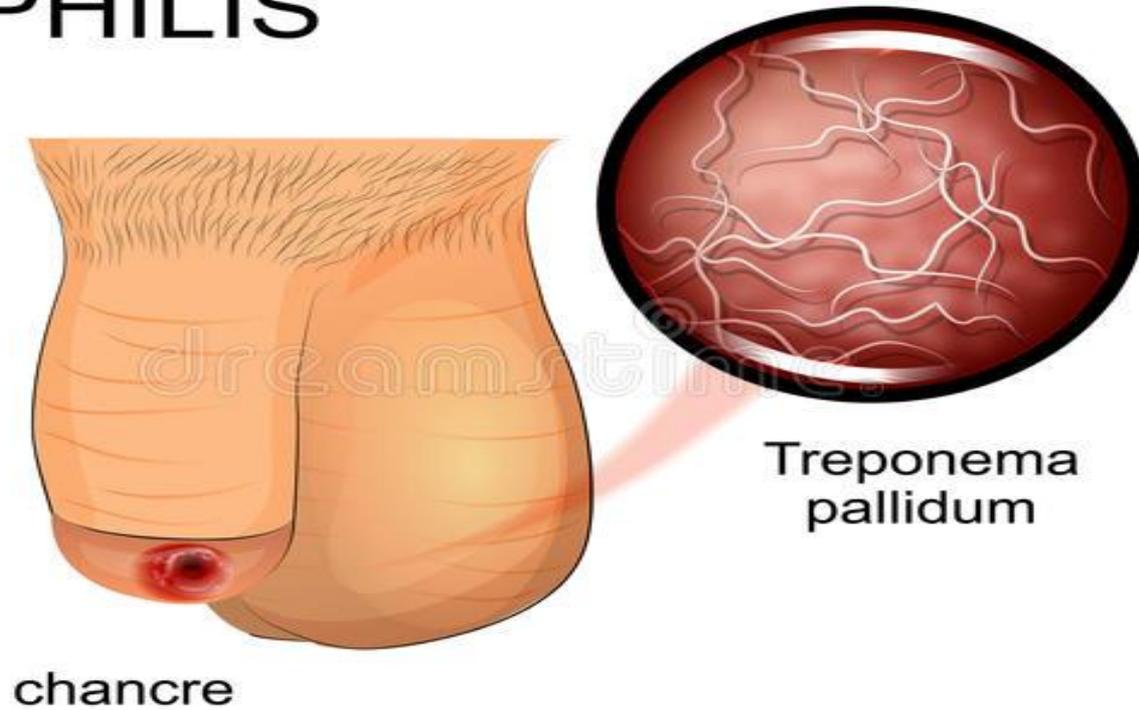
Syphilis

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



Primary chancre

SYPHILIS



Secondary syphilis- papulosquamous dermatosis



Condyloma Lata, painless
Wart like lesion

Human Papilloma virus

- Human papillomavirus may **cause singular warts** (verruca vulgaris)
Or **multiple warts in the anogenital area** (condylomata acuminata-
this is a major problem in HIV patients).
- Verruca vulgaris: **HPV 1,2,4, 7,** and common in children and young adults
- Transmitted by skin contact, sharing certain tools/cloth.
- 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..)**

digital



Digital + auto inoculation



Condyloma acuminata

Diagnosis of skin infections

- As discussed, so far diagnosis of skin infections depends on three main clinical observations :
- → 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)

- Aiding 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

- However, even the most experienced clinician will find it difficult to diagnose all infections of the skin and soft tissue by History and inspection alone.
- Thus aiding diagnostic tests can help:
- 1- **Soft tissue radiography**, CT (next figure), and MRI may be used to help determine **the depth of infection**
- The helps in assessing deeper infections-
- → You might recall that **serious ones like fasciitis are rapidly progressing**
- → Evidence of a **systemic inflammatory response syndrome** (to find a local infection that may be releasing toxins).
- → another value for these tests is for **defining a localized abscess** or **detecting gas in tissue** (air pockets) where anaerobes are present (not GAS infection, which may only show swelling as shown in next figure).



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, however, there is a **large false negative rate ~80%**.
- Frozen sections are especially useful in **distinguishing SSSS from TEN** (?) also quite 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 is the superior method to **obtain specimen for culture and Gram stain**.
- Although surgical approach maybe an aggressive approach, it is an important step 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 and antibiotic treatment.

A study in children showed that surgical drainage of abscesses (mean diameter, 3.8 cm) was as effective when used alone as when combined with trimethoprim-sulfamethoxazole treatment.

However, recurrence was less in groups that added Abx with I&D

TABLE 22-2**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	Clindamycin, 600–900 mg IV q8h, <i>plus</i> Ciprofloxacin, 400 mg IV q12h, <i>or</i> Cefoxitin, 2 g IV q6h	35
Bacillary angiomatosis	Erythromycin, 500 mg PO qid	Doxycycline, 100 mg PO bid	65
Herpes simplex (primary genital)	Acyclovir, 400 mg PO tid for 10 days	Famciclovir, 250 mg PO tid for 5–10 days, <i>or</i> Valacyclovir, 1000 mg PO bid for 10 days	84
Herpes zoster (immuno-competent host >50 years of age)	Acyclovir, 800 mg PO 5 times daily for 7–10 days	Famciclovir, 500 mg PO tid for 7–10 days, <i>or</i> Valacyclovir, 1000 mg PO tid for 7 days	85

Cellulitis (staphylococcal or streptococcal ^{b,c})	Nafcillin or oxacillin, 2 g IV q4–6h	Cefazolin, 1–2 g q8h, <i>or</i> Ampicillin/sulbactam, 1.5–3 g IV q6h, <i>or</i> Erythromycin, 0.5–1 g IV q6h, <i>or</i> Clindamycin, 600–900 mg IV q8h	38, 39
MRSA skin infection ^d	Vancomycin, 1 g IV q12h	Linezolid, 600 mg IV q12h	38
Necrotizing fasciitis (group A streptococcal ^b)	Clindamycin, 600–900 mg IV q6–8h, <i>plus</i> Penicillin G, 4 million units IV q4h	Clindamycin, 600–900 mg IV q6–8h, <i>plus</i> Cephalosporin (first- or second-generation)	39
Necrotizing fasciitis (mixed aerobes and anaerobes)	Ampicillin, 2 g IV q4h, <i>plus</i> Clindamycin, 600–900 mg IV q6–8h, <i>plus</i> Ciprofloxacin, 400 mg IV q6–8h	Vancomycin, 1 g IV q6h, <i>plus</i> Metronidazole, 500 mg IV q6h, <i>plus</i> Ciprofloxacin, 400 mg IV q6–8h	69
Gas gangrene	Clindamycin, 600–900 mg IV q6–8h, <i>plus</i> Penicillin G, 4 million units IV q4–6h	Clindamycin, 600–900 mg IV q6–8h, <i>plus</i> Cefoxitin, 2 g IV q6h	46

Myositis and Myonecrosis

Myositis and myonecrosis

Pyomyositis

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

- **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 (coxsackievirus B), trichinellosis, and bacterial infection.
 - > Acute rhabdomyolysis (breakdown of damaged muscle) predictably occurs with clostridial and streptococcal myositis, as 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 muscles are affected by necrosis when the surrounding fascia is involved, even without the infection being active in the muscle itself!)

pyomyositis

- Pyomyositis, or pus forming infection of the muscle tissue, is usually due to *S. aureus* (remember it is the typical pus former in skin)
- Pyomyositis is common in tropical areas, and generally *has no known portal of entry (in contrast to necrotizing fasciitis)*.
- Cases of 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

Cont.

- Pyomyositis infection usually remains localized, and shock does not develop unless organisms produce
- 1- *toxic shock syndrome toxin 1* (which acts as a super antigen, that 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.

Pyomyositis

- a purulent- PUS forming- infection of skeletal muscle.
- Abscess formation is the usual consequence when these pyogenic bacteria reach the muscle tissue.
- Pyomyositis usually arises from haematogenous spread (deeper infections, muscle and bone, are typically more related with haematogenous spread rather direct inoculation).

Epidemiology

- Occurs in M > F , and more in tropical climate in two main age groups:

→ children (aged 2–5 years)

→ adults (aged 20–45 years).

In temperate climates

→ 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, 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, and MAC.

Clinical features

- 20% and 50% of cases patient have had recent blunt trauma or vigorous 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 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 swellingAn abscess may be clinically apparent, aspiration of which yields pus. There is marked leucocytosis;

- • **Stage 3 (systemic stage)**—

- the affected muscle is fluctuant. Patients may present with complications of *S. aureus* bacteraemia, e.g. septic shock, endocarditis, septic emboli, pneumonia, pericarditis, septic arthritis, brain abscess, and ARF. 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- see next).
- 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!

the muscle will not yield purulent material. Because of unspecific symptoms, pyomyositis would not be usually the initial diagnosis



Pyomyositis inner thigh in young patients with severe aplastic anemia (F. coli)



Management

- **Antibiotics**— stage 1 antibiotics alone
 - HOWEVER, most patients present with stage 2/3 disease and require antibiotics+ drainage.
 - Empiric therapy for these stages:
Directed against *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 Dx and Rx (drainage and send drain sample for Micro)
- This may be CT-guided or ultrasound-guided.