# MSS

## Musculoskeletal System

Doctor 2019 | Medicine | JU

5

# Microbiology


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السلام عليكم
الشيت فيها كثير مواضيع لهيك كل موضوع رئيسي فيها رح اكتبه بالاحمر

الشيت طويلة لكن معظمها صور لهيك شدوا حيلكم

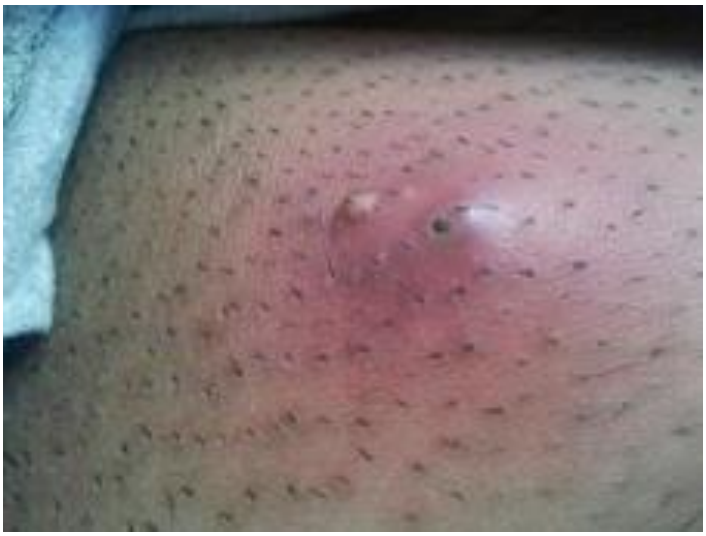
بالتوفيق يا رب

**(00:00-10:00)**

# 1-Cutaneous Abscesses

- **Collections of pus within the dermis and deeper skin structures.**

- **Clinical features**:
  painful (elicits pain without touching) ,tender (elicits pain when you touch it), fluctuant nodules (not fixed to the under-laying structure, because the outer board of the abscess won't fix to surround the structure.)
  For example, cancer cell and lymph nodes can be fixed), usually with an overlying pustule and surrounded by a rim of erythematous swelling.
- Usually polymicrobial containing skin/mucous membrane flora; S. aureus is the sole pathogen in 25% of cases.
- Treatment is I&D (incision and drainage).
- Antibiotics are rarely necessary except in: extensive infection, systemic toxicity, or the immunocompromised.



Notice:

• The lesion is raised.

• It has a white head.

• The hair follicle might be the port of entry.

# 2-Diabetic Foot Infections

**Is defined as any infection in a patient with DM that is below the malleolus**.

Almost everything we took in this module as microbiology, immunology, infectious causes will have representative in diabetic foot.

- **It is a broad spectrum of infections ranging from: paronychia, cellulitis, myositis, abscess formation, necrotizing fasciitis, septic arthritis, tendonitis, and osteomyelitis!**
- **The commonest of these lesions is an infected diabetic ulcer.**

## Epidemiology

- Foot infections in DM are a **common complication**, they often carry a high burden of morbidity for the patient and **notoriously difficult to manage** (there are specialized DF surgeons and clinics!)
- Risk factor:

1. **Vascular**: vascular insufficiency

Including coronary artery disease, kidney disease, healing disorders (diabetic foot), stroke. All of these due to accelerated atherosclerosis and vessels disease.

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

Reduce sensation specially in lower extremities, more likely to have unnoticed trauma, proprioception (don't feel your surrounding well)

3. **Immune**: hyperglycemia leading to poor immune function and wound healing.
4. **Other factors**: poor vision, limited mobility, previous amputations, and poor healthcare, these due to hyperglycemia which changes certain proteins' structure, for example, protein in the lens might change, overtime they will have vision pacification and change the lens.

## Etiology

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 |

**Notes:**

1. Cellulitis in a diabetic foot will be similar to cellulitis in any other area. Infection includes gram positive streps and staphs. **Group A is the most dominant one,** but the other are more seen in diabetes then staph. .
2. Infected ulcer antibiotic naïve has never been treated by antibiotics. This ulcer is better than one that has been exposed to antibiotics. If an infection happens in a naïve ulcer, most likely to be monomicrobial, and the same organisms we notice in cellulitis: staph or strep.
3. Infected non-naïve ulcer (chronic) exposed to antibiotics, so the organism makeup is changed, and the patient presented to hospital and exposed to enterobacteria, polymicrobial, more difficult to manage than naïve ulcer.

**(10:00-20:14)**

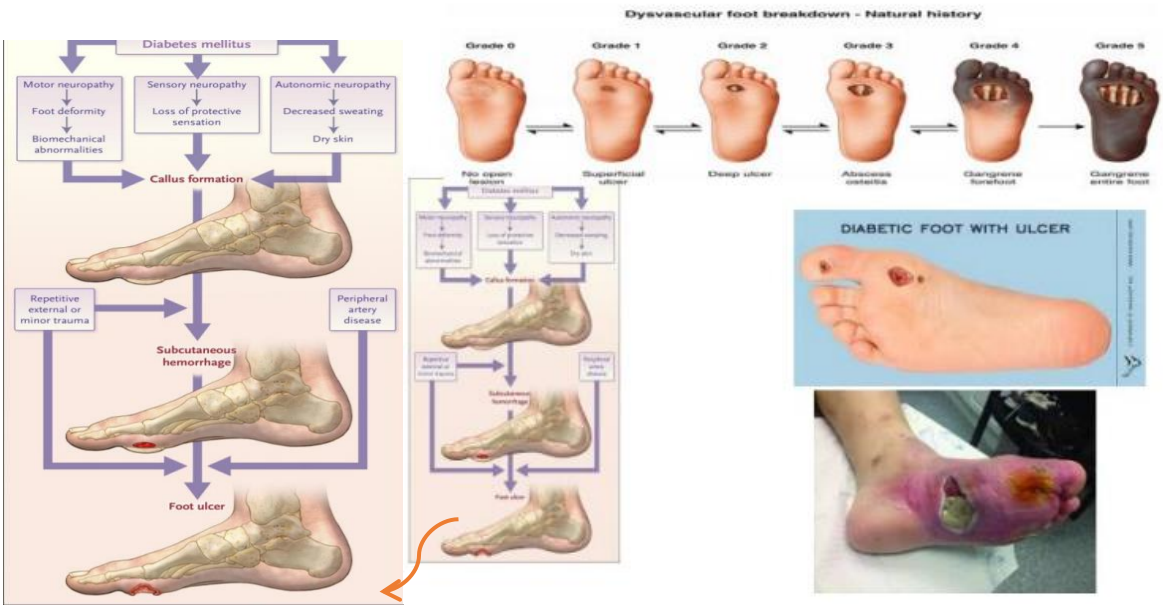
4. Macerated ulcer where the surrounding tissue is Laceration, here Pseudomonas is introduced, this more likely to happen in nosocomial.
5. Long-standing, non-healing wound, with prolonged antibiotic therapy, where there is sub-optimal level antibiotic that enters the wound, so the best way to treat it is giving optimal doses of antibiotic.

- Low doses (sub lethal): the remain bacteria that didn’t die will regroup and grow again, until they tolerate resistance because we aren't obligating them completely.

- High doses: toxicity

When the wound has reduced perfusion, even if giving the right doses, it isn't sending enough antibiotic and in case of diabetes its worse.

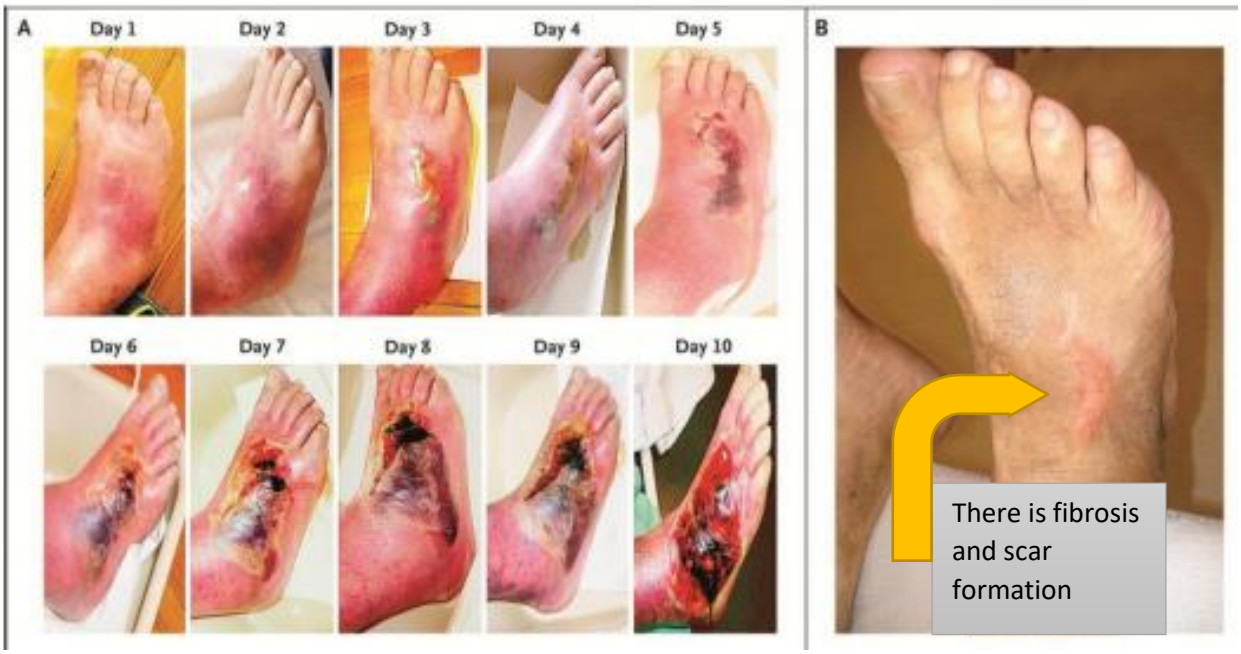
6. Fetid foot is necrosis or gangrene, it stopped the flow of blood which will cause irreversible cell injury then **gangrene**, here **obligate anaerobes** take place.




About this picture:

It starts with non-open lesion, then superficial ulcer which will open to become a deep ulcer, the underlaying tissue and fascia start to appear, then abscess osteitis and upgraded to the bone in grade 3. Forefoot has ganger and the entire foot at grade 5 becomes gangrenous.

In diabetic foot, the sites of pressure are more likely to have ulcer, because these areas required remodeling and where trauma is more likely to occur.




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.

-The figure A and B is for the same patient, he had diabetic foot. The difference between B and A-day1 that its darker and thinner so probably the patient loss weight and exposed to the sun (control his blood sugar).

When the patient wasn't controlling his blood sugar, the infection kept monitoring every day and addiction from necrosis. B is where his foot treated, it

was a naïve infection so after antimicrobial therapy and debridement the infection resolved.

What is the optimal treatment ? Blood sugar control .

you control the blood sugar, you control the infection.

## Clinical Features

• Range from mild -> severe -> life-threatening

• Progresses:

1. Foot ulcer with no signs of infection.
2. Foot ulcer with surrounding inflammation or cellulitis <2cm from edge of the wound.
3. Local complications – cellulitis >2cm from the edge of the wound. +lymphangitis, spread beneath the superficial fascia, deep tissue abscess, gas gangrene, and involvement of muscle, tendon, or bone.
4. Systemic toxicity or metabolic instability—fever, chills, tachycardia, hypotension, confusion, vomiting, leukocytosis, acidosis, hyperglycemia, and uremia.

**(20:14-31:18)**

## Diagnosis

1. **Clinical features**: must assess perfusion (peripheral pulses, less perfusion = more anaerobes), as well as sensation (using a mono filament).

The better you feel the pulses the better outcome you will have, while the filament is used acessing neuropathy and sensation.

2. **Doppler ultrasound** to determine ratio of ankle Vs brachial pressure index (aBPIs).

To see how well it perfuse

3. **Imaging (MRI)** may help determine the extent of infection (osteomyelitis, fasciitis). This considered as a seed to continue the infection in the feet, because osteomyelitis is much more chronic problem than soft tissue.

4. **Deep tissue specimens** (not superficial swabs) need to be sent to microbiology lab for microscopy and culture before Abx (antibiotic) treatment.

## Management

- Inpatient Vs outpatient management.

- Outpatient management is preferred to prevent exposure to pathogens in hospitals, like pseudomonas aeruginosa.
- Inpatient Rx is based on correcting systemic instability.

1- Medical treatment: Do not give Abx ( antibiotics) for non-infected ulcers (to ensure a future infected ulcer remains naïve). For infected ulcers, initial treatment is empiric therapy, oral for mild cases and IV broad spectrum for severe cases. Also work on fixing blood sugar levels.

2- II- Surgery (Debridement)—For cases of severe infections in deeper tissues (necrotizing fasciitis, gas gangrene, extensive tissue loss, and critical limb ischemia).

• Wound care plan following discharge.

* Debridement is both therapeutic and diagnostic, as samples of the removed tissues can be sent to the microbiology lab.

## Papular and Nodular Lesions

These lesions can be caused by viruses, bacteria, fungi and parasites.

**Papular and Nodular Lesions**

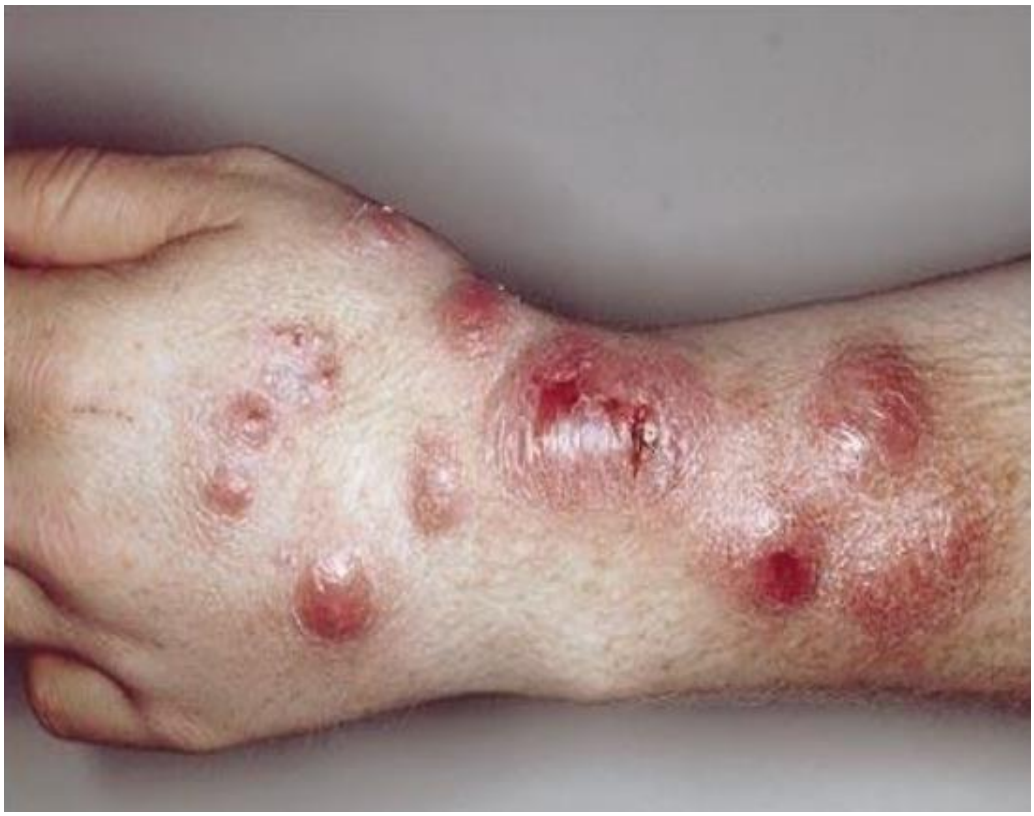
| Papular and nodular lesions | |
|---|---|
| Fish-tank or swimming-pool granuloma | *Mycobacterium marinum* |
| Creeping eruption (cutaneous larva migrans) | *Ancylostoma braziliense* |
| Dracunculiasis | *Dracunculus medinensis* |
| Cercarial dermatitis | *Schistosoma mansoni* |
| Verruca vulgaris | 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* |

## 1. 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.

The core temperature of the body is 37, but at the first layers of skin it is less than this.

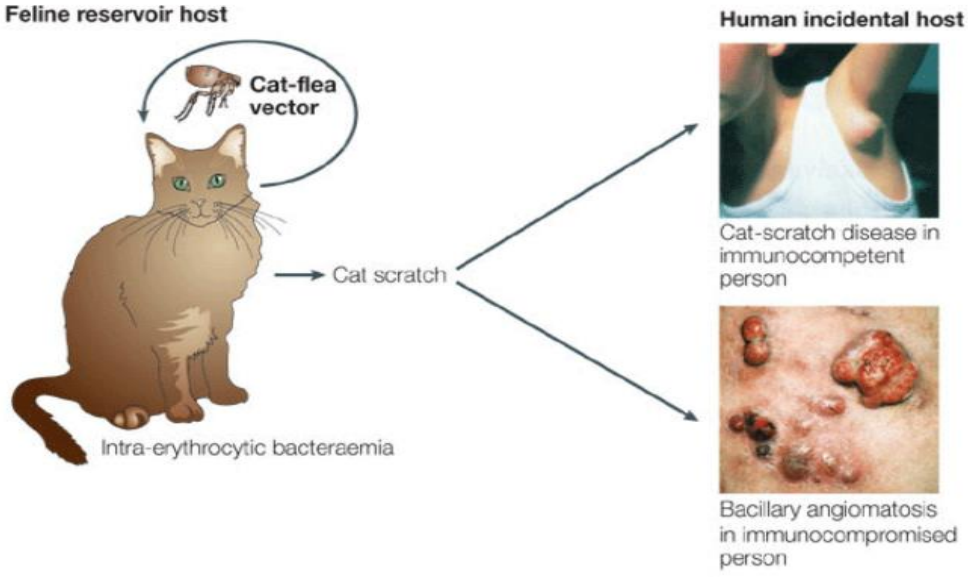
- Dx: **needle aspiration, see acid fact bacilli.**
- Rx: **rifampin + ethambutol for four months.**
- History is your helper in discovering this case.



## 2- 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. Home cat that is well clean and taken care of is less probably to has this, street cat has bigger chance to scratch and transmit this bug.
- Symptoms: **myalgia, arthlagia, 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, its painful only in scratch region.
- **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, but it's a problem in immunosuppressive.




- Immunocompetent person: IPSILATERAL Lymph adenopathy might be painful and can resolve by its own. Some guidelines indicate that we can give antimicrobial.
- Immunocompromises person: this will give bacillary angiomatosis which is an advanced disease and gives serious skin reaction and requires treatment.

## 3-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 hypertensives-fever malaise..etc.)**

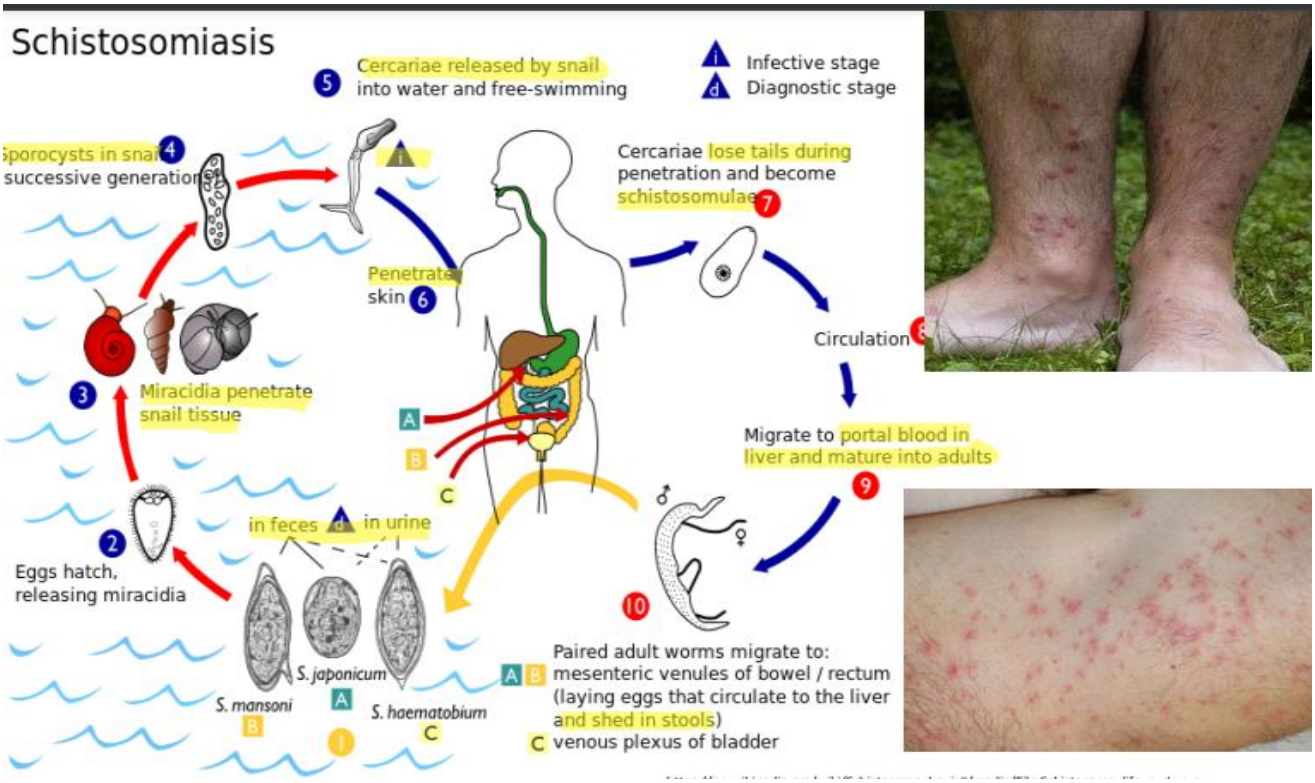
• **Dx** :stool and urine microscopy (shows the presence of eggs)

• **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.

Bilharzia has been a big problem in our country especially in Egypt. Children used to play in small body of water that doesn't have any tributary , it's a good environment for these organisms' growth.

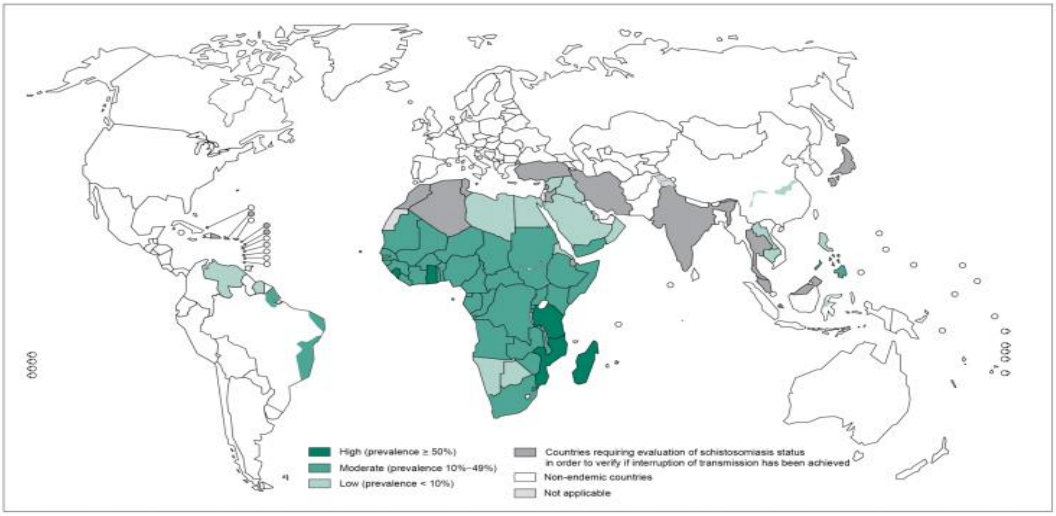
- If kept untreated the complication include liver failure!



## Life Cycle

Infected by Schistosoma--> the eggs of the parasite will grow in their gut--> They will migrate to the rectum and shed in the stool (and sometimes through the urine) into a water supply--> The eggs will then hatch and must pass through a snail host--> the parasites will infect a snail and mature there leave as mature parasites-->When someone else comes and stands in this infected water, the parasites will invade the skin, and nodules will form where the parasites penetrated the skin--> Bilharzia will occur and the parasite goes to the portal circulation and reaches the liver, spleen, and kidney.

Distribution of schistosomiasis, worldwide, 2012




* Endemic area: Egypt (and the rest of Africa).

**(31:18-46:33)**

# Prognosis

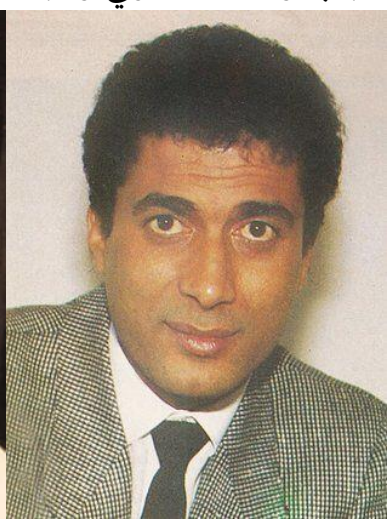
• Early disease usually improves with treatment.

• 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).

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

من اسباب وفاة احمد زكي وعبدالحليم حافظ هو التشخيص المتاخر للبلهارسيا.

## 4- 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 (more intimate prolonged contact increase risk of transmission).

• Has two types:

tuberculoid: patient has intact immunity, usually nerve changes predominate.

Lepromoatous: **where skin changes predominate (defect in cell immunity**), this time is worse and more common for those who have bad immunity.

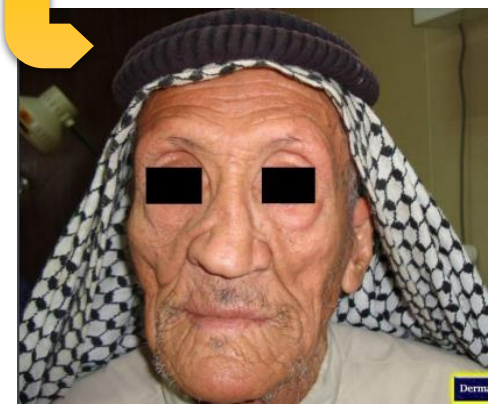
• **Skin changes:** bilateral symmetrical macules and papules, progresses to nodules and even plaques.

• 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 (is special for leprosy) and Rifampin, treatment at early stages is **for 1-2 years**, in leprosy treatment maybe **lifelong**.

When people get older they have symmetrical changes in the skin which might be unnoticed, but if you see a picture of the patient before 10 years it might indicate that this is not going to progress as this .

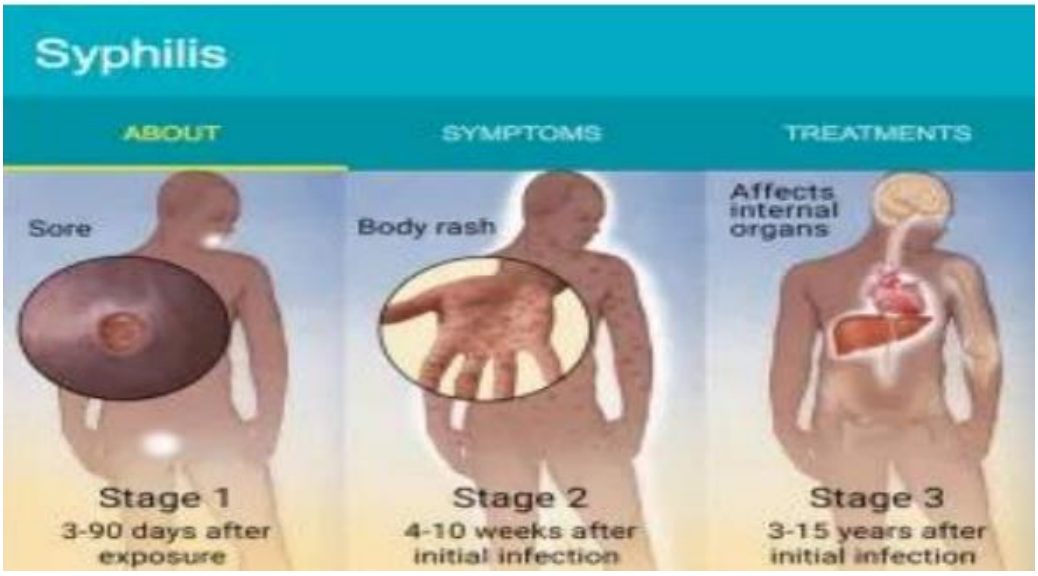


**This case is quiet sever.**

## 5-Syphilis

• **Large nodules or gummas are features of tertiary syphilis (late untreated syphilis)**

•whereas **flat papulosquamous lesions** are characteristic of secondary syphilis



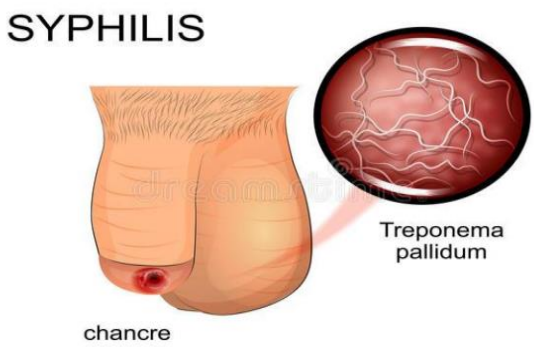

-stage 1: sore after (3-90) days after exposure.

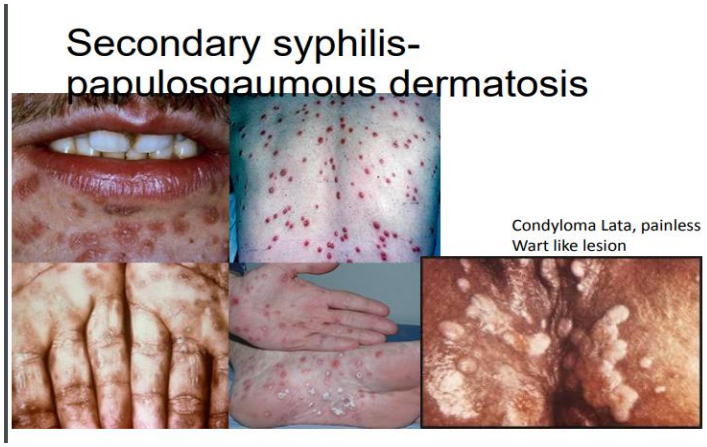
-stage2: body rash

-stage3: internal organs are affected.

condyloma Lata : wart like lesion near the anal

Primary chancre



## 6-Human Papilloma virus

• Human papillomavirus may **cause singular warts** (verruca vulgaris) Or **multiple warts in the anogenital area** (condylomata acuminatathis is a major problem in HIV patients).

• Verruca vulgaris: HPV 1,2,4,7, and common in children and young adults

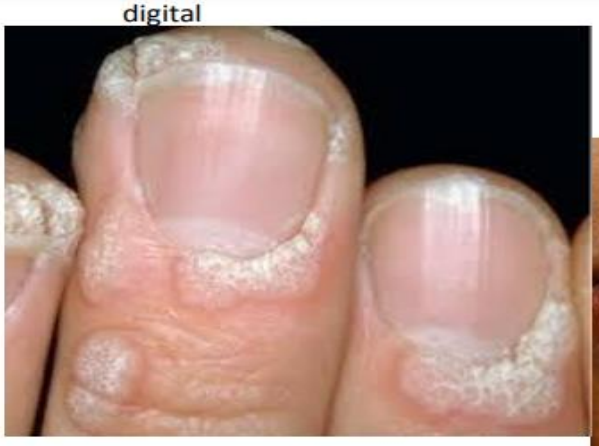
• HPV is an STD, and can cause cervical cancer (16, 18, 31, 33..)

• **Transmitted by skin contact**, sharing certain tools/cloth.

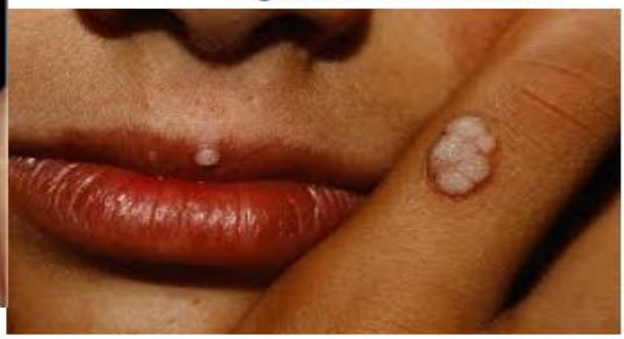
• Usually on **the hands and nail edges**, can **auto inoculate face**

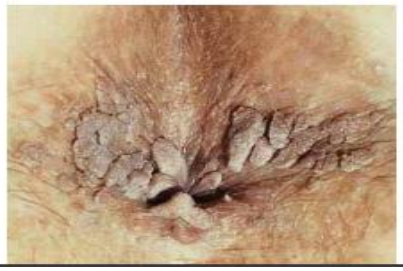
• Management : Dermatologists

digital



Digital + auto inoculation





Condyloma acuminata

## Diagnosis of skin infections

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

1. Appearance of lesion (macule, papule, vesicle…etc.)

2. Location within the layers of soft tissues (stratum corneum, epidermis, dermis..etc.)
3. 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 /gender/sexual contact

→ 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.

مثلا لو عملت مخطط لأكثر التشخيصات المحتملة لهذا المريض و وضعت ٣ احتمالات ممكنة

وكان الاحتمال الاول بالنسبة الك ٧٠٪ هو سبب الحالة، وثاني احتمال ٢٠٪ و الثالث ١٠٪

فتروح تعالج المريض حسب تشخيصك، وتعطيه علاج على اساس اول احتمال

و تطلع مخطئ والتشخيص الاصح يكون صاحب الاحتمالية ١٠٪ ( الله لا ينكس حدا 😂😂😂😂)

. لهيك لازم تجمع تفاصيل اكثر

• **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).

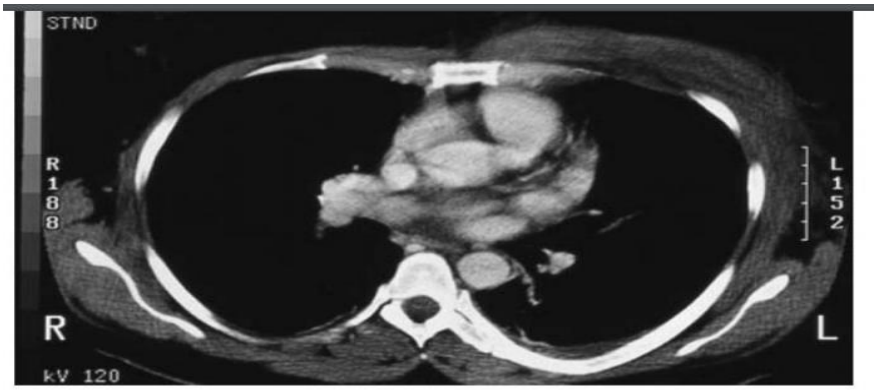



**FIGURE 22-2**
**Computed tomography showing edema and inflammation of the left chest wall** in a patient with necrotizing fasciitis and myonecrosis caused by group A *Streptococcus*.

It is good to practice on these pictures because a lot of exams in the future will ask use about CT scans.

-you can see spin, aorta, pulmonary artery, lungs, chest wall and a swollen in one side deeper tissue, necroses and inflammation in left chest wall.

## 2- lab diagnostics

- Aspiration (without saline better → less dilution) and reduce the captured number) 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**, this will give

me the sensitivity profile to determine the best antimicrobial therapy.

- 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

•treatment 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. So, drainage alone was enough.

However, recurrence was less in groups that added Abx with I&D, so antimicrobial usage isn't treating, it only reducing the Currence.

**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, *plus* Ciprofloxacin, 400 mg IV q12h, *or* 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, *or* 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, *or* Valacyclovir, 1000 mg PO tid for 7 days | 85 |

**سُبحَان الذي إذا ذَكرتَهُ ذكَرَك**

**سُبحان الله، الحمد لله، لا إله إلا الله، الله أكبر لا حول ولا قوة إلا بالله**