- Osteomyelitis -

- infection involving bone -

Classified: [1] Mechanism of infection (hematogenous, nonhematogenous) [2] duration of illness (Acute / Chronic)

Acute → Several days to weeks

Chronic osteomyelitis Hallmarks → [1] Presence of dead bone (sequestrum) [2] involucrum [3] Bone loss

Nonhematogenous osteomyelitis ⇒ Results of contagious spread of infection to bone from adjacent soft tissue & joints OR via direct inoculation of infection to the bone.

Polymicrobial, monomicrobial

↳ [Staphylococcus aureas, coagulase⊖ staphylococci; Aerobic gram⊖ bacilli]

Younger Adults ⇒ nonhematogenous osteomyelitis occurs in the setting of Trauma or Surgery

Older Adults ⇒ occurs due to spread of infection to bone from adjacent soft tissues

Hematogenous osteomyelitis ⇒ Microorganisms that seed the bone in the setting of Bacteremia. [because of bacteria in blood = bacteremia]

Monomicrobial

↳ [S. aureus, Aerobic gram⊖ rods]

Most common form of osteomyelitis in infants & children

Adults: Vertebral osteomyelitis is the most common form of Hematogenous osteomyelitis. > 50 years

Tuberculous Osteomyelitis due to Reactivation of tuberculous bacilli in bone during Mycobacteremias.

Pathogenisis

it develops when there is large inoculation of organisms, Bone damage and other foreign material

ex// S. aureus Pathogenisis:
- Adherence to a number of components of bone matrix
- Survives intracellularly in cultured osteoblasts
- Undergoes phenotypic alteration to be more resistant to antimicrobials

{That's why we have high relapse even if given antimicrobials !}

Clinical Manifestation

- Acute osteomyelitis; gradual onset over several days
  - dull pain with or without movement
  - local Findings; tenderness, warmth, Erythema and Swelling
  - Systemic Symptoms; Fever & Rigors
- Chronic osteomyelitis; Warmth, Erythema and Swelling,
  - draing Sinus tract is pathognomonic (specific symptom for disease)
  - * Fever is absent
  - * Intermittent flares of Pain and Swelling

Cases: ~ Clinical Manifestation ~

In general => culture obtained from biopsy of the involved bone
أكيد الزرعة أفضل للتشخيص لكن ما بنلجأ الها دايما

1] If therapeutic debridement is needed =>
Radiographic findings + Positive blood Cultures

2] Recent Antibiotic Admenstration =>
Bone histopathology + the absence of Positive culture data
معو أكيد طلع معقم من قبل ليعطوه ال antibiotic

3] No positive culture data and a biopsy is not Feasable =>
Radiographic Findings + Elevated inflammatory Markers

~ Management ~

Radiographic imaging should prompt bone biopsy to confirm the diagnonis and to guide Antimicrobial therapy

The chosen regimen has high activity against common causes of osteomyelitis ex: staphylococci, streptococci, G- baccili

---

~ Septic Arthritis ~

infection in a joint

- Septic arthritis arises via; Hematogeneous seeding
- Bacteremia is more likely to localize in the joint in the case of Pre-existing arthritis.

- Monomicrobial S. Aureus -

Clinical Manifestation

- Single swollen and painful joint
- Most patients are Febrile

Knee is more than 50%.

Diagnosis

Synovial Fluid analysis and culture, Blood cultures & Radiograph.

Treatment

joint drainage and antibiotic therapy

- needle aspiration
- Arathroscopic drainage
- Arthrotomy

Synovial Fluid

demonstrates gram-positive cocci → Vancomycin

gram-negative bacilli → treatment should be guided by risk for Pseudomonas infection

1/3 of patients had a poor joint outcome (amputation, surgery, severe deterioration)

* Adverse prognostic Factors include: older age & pre-existing joint disease.

~Animal Bites~

Dog bites → 90% of animal bites , cats → 10%

Infections are Much more common after cat bites.

* Cat bites → occur on the Extremities and tend to penetrate deeply with higher risk of deep infection than dog bites

Management → we should Ensure that the patient is hemodynamically Stable

and assess for injuries and neurovascular assessment in the adjacent structures in distal areas to the wound

Bite → Superficial: cellulitis; with or without abscess

Fever, Swelling, Erythema, warmth & purulent drainage

→ Deep: Abscess, Septic Arthritis, osteomyelitis, necrotizing soft tissue infection

Bartonella → gram ⊖ / cocco bacillary

→ fastidious growth {which means it needs specific factors to grow requiring a prolonged incubation (2-6 weeks)}

→ facultative intracellular

→ Transmitted by vectors

B. henselae is responsible For cat-scratch disease

Symptoms; non-painful bump or blister at the site of injury & swollen lymph nodes.

Pathogen includes the oral flora of the biting animal & the human skin flora.

1. Pasteurella → 75% cat bites 50% dogs bites
2. Capnocytophaga canimorsus → bacteremia and fatal sepsis
3. B. henselae → cat scratches
4. Anaerobes
5. Mixed Aerobic and Anaerobic bacteria

- Clinical Scenarios -

Pressure ulcers localized areas of tissue necrosis when soft tissue in compressed between a bony prominence & External surface.

* Pressure ulcers cause pain, decrease the quality of life, Morbidity and prolonged hospital stays due to complicating infections

* Factors that contribute to infection of Pressure ulcers;
  1. Breaks in the integrity of the skin barrier.
  2. Pressure-induced changes.
  3. Contamination from contagious dirty areas. → most common is fecal contamination

* The ulcer is First colonized by normal skin Flora than Replaced by bacteria From the local environment & the urogenital or gastrointestinal tract

* Pressure ulcers are normally associated with bacteria count of $< 10^2$ colony forming units if cured. Higher concentrations may inhibit normal wound healing without inducing a host Response

Critical Colonization ↗

and the presence of biofilm → causes delayed healing.

Pressure Ulcers are Polymicrobial

* The Predominant Organisms were: ① Enterobacter ② staphylococci ③ Enterococcus Faecalis in SCI Patients;
  Spinal cord injury

* Organisms in ulcer-related bacteremia in SCI Patients; ① staphylococci ② streptococci ③ Proteus mirabilis

* The Extent of local infection associated with Pressure ulcers ranges;

From an infection limited to the superficial ulcer base
to one with surrounding cellulitis (spreading Erythema)
to more Extensive involvement of deeper structures (bone, Muscles & Fascia + erosion at sinus tracts)

* Sepsis + pressure ulcer-Related soft tissue infections → Bloodstream infection - sepsis & pneumothorax ect.

* Exudate alone Without additional signs or symptoms of Soft tissue or systemic involvement → Superficial infection → Diagnosis; local debridement and wound care.

* The most useful Specimen in Culture → Biopsy from the deepest tissue involved, obtained during debridement of an ulcer.

→ Imaging is not necessary ; However patients with 8

① Systemic Manifestations ② Positive blood cultures ③ indwelling Medical devices } - - - {

require Imaging like (MRI) for identifying the underlying necrotic tissu or abscess requiring debridement

* Patients with Osteomyelitis with pressure ulcers → - we have to obtain bone biopsy in order to confirm the diagnosis & guide the selection of Emperic Antibiotics.
- Histopathologic & Microbial Analyss

* Essential components of Management → ① Full debridement of necrotic tissue and bone.
② Emperic antibiotic Therapy

Burn wound infections and Sepsis

* infection at burn sites increase the Risk of Morbidity & Mortality.

* Factors increase the Risk of infection at burn sites:
1. TBSA > 20 Percent — TBSA : Total Body surface Ar
2. Delay in Excision
3. Extremes in Age
4. Impaired Immunity
5. Microbial Factors.

* The spectrum of Microorganisms causing infection in burn patients varies with time & Location.

→ The organism causing burn wound infection appear at varying stages post-burn injury.

* Early colonization of burn → gram ⊕ bacteria

* > 5 days → gram ⊖ bacteria

* If the gram ⊖ initiated, Yeast often appears.

* Early diagnosis depend on the Recognition of the infected burn wound site.

* Clinical Features → ① Rapid Change in the appearance of wound (ex partial thickness → full thickness injury)
② Loss of tissue or skin graft

* Acute (bacterial) infection manifests the development of discoloration, Pain Purulent Exudate, tenderness, swelling, drainage, Malodor From a burn

* Signs of burn wound infection caused by (Fungi) include the Separation of the Eschar due to Fat Liquefaction & rapid spread of Subcutaneous edema and ischemic necrosis.

* qualitative wound cultures → identifies the Presence of the Flora
* quantitative wound cultures → is Required to Confirm the diagnosis of burn wound infection (num of bacteria / gram of tissue) via Tissue Histopathology.

Identified By Histopathology

→ Noninvasive infection
- when there are typical clinical Features with no Systemic Signs
- NO microbial invasion into unburned tissue.
- $>10^5$ bacteria per gram of tissue obtained from the burn wound itself

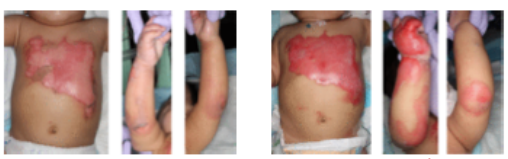
Invasive Infections;
- When there are clinical Features with systemic Signs.
- Microbial invasion into unburned tissue.
- $>10^5$ bacteria per gram of tissue in adjacent unburned tissue

* Initial Management → Stabilizing & Restoring Perfusion
* Emperic broad-spectrum antimicrobial therapy & Excision to all infected tissue to a healthy tissue bed

Burn Intensive Care Units (ICUs) → Highest Rate of Primary Bloodstream infection + sepsis + associated deaths

>> because of multidrug-resistant strains of bacteria found in hospitals

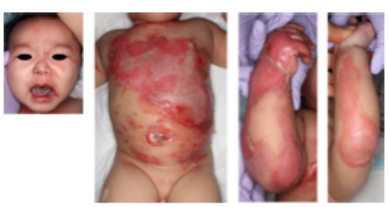
- An 8-month-old boy burned by hot water had partial thickness burns on 10% of his total body surface area. He was admitted to hospital and received fluid therapy and wound care treatment. During hospitalization, he developed a high fever and exfoliation of the skin, except for the burns. He then received antibiotic infusion treatment daily. Three days after initiating antibiotic therapy, he had epithelization of the raw surface, except for his burns. Skin exfoliation affected 36% of the total body surface area.



less color → more pink-reddish
a sign of healing

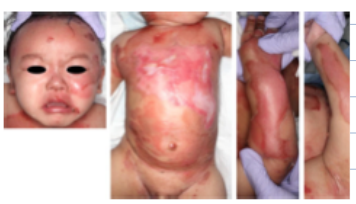
On Day 3, the patient's burn wounds show no infection.

By day 10: with antibiotic treatment the wound epithelized



By Day 5, the patient had sudden enlargement of the exfoliation.

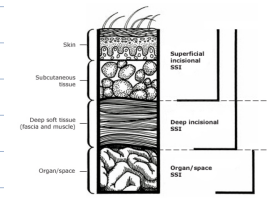
infection by S. aureus secreting an exfoliative toxin causing scalded skin syndrome



By Day 7, the exfoliated area, excluding the burn wound, has formed scabs.

~ Surgical Site infection ~

* Incisional SSI
  - only skin and subcutaneous tissue [Superficial]
  - Deeper Softer tissue of the incision [Deep]



* Surgeons can Reduce the Risk of SSI → ① Avoiding Elective surgery in active infection ② Prophylactic Antibiotics ③ Proper skin Preparation ⑤ Sterile conditions.

Note ⇒ Please go back to the Necrotizing Fasciitis case - Last 3 slides-

* Surgical wounds classification

[1] class I / clean :
- uninfected wound (no contamination)
- No penetration of mucosal surfaces
- ex of operation (surgery) : non penetrating (blunt) trauma

[2] class II / clean-contaminated :
- Uninfected, no contamination
- penetration of mucosal surfaces
- ex of operation : involving appendix, vagina, oropharynx ...

[3] class III / contaminated :
- Encountered contamination & inflammation because of breaks in sterile technique
- penetration of mucosal surfaces ex. GI tract
- ex of operation : open cardiac massage, gross spillage from GI tracts

[4] class IV / Dirty infection :
- Exiting infection, & contamination
- No sterile environment for the operation

تعطلوا حادت سيارة و انفتح بطن الكرسي

SSI
- Superficial SSI
  - Active drainage & Surgical wound Exploration
  - Imaging is not necessary
  - Antibiotics are adminshated only if there is Cellulitis.
- Deep SSI
  - Active drainage & Surgical wound Exploration
  - Imaging is necessary
  - Requires Antibiotic Adminstration

Skin and Subcutaneous Mycoses

1 Superficial Mycoses (versiwlor)

Tinea versicolor, Pityriasis versicolor

↓

Malassezia

M. Furfur M. Globose

- Lipophilic Yeast (Round in shape) and can be hyphae, while being pathogenic (inside) and nonpathogenic (outside)
- Normal Commensals of the skin. [can't be transmitted to others]
- Can cause skin infections and catheter-associated infections they become pathogenic due to ↓ gaining a virulence factor.
- It only affects the outermost layer of the skin, only cosmetic symptoms.
- Trunk And Proximal Limbs.
- Common in Tropics and precipited through Sun Exposure
- versicolor → Hyperpigmentation / → Hypopigmentation
  Carboxylic Acid Produced by the Yeast causes the depigmentation
- * Asymptomatic, non-itchy macules hypo or hyper pigmented
- * Can Coalesce to form scaly plaques
- * Diagnosis; 1 UV-light; pale greenish color under wood's Ultra Violet light
  2 Skin scraping the int and KOH Staining

2 Cutaneous Mycoses

A. Ring worm or Tinea

↓

[tinea+suffix] Dermatophytes [Fungi, Molds]

3 genera; 1) Microsporum
2) Trichophyton 3) Epidermophyton

→ These Fungi, they affect the keratinized tissue; skin, hair & nails

immunological Response [itchy] is present.

→ infection doesn't spread to deeper tissues

* Source of infections:
1) Man to Man; by direct contact [Anthrophilic]
2) From animals; [Zoophilic] — Acute low Resistance
3) From the soil [geophilic] — Acute low Resistance

Heat And Humidity Enhance the infection

* Clinical Forms;
1) Tinea pedis (Toes) 2) Tinea corporis (hairless skin Groin & Medial thighs)
3) Tinea Capitis (Scalp) 4) Tinea unguinum (Nails)

Tinea Pedis, Dermatophytes of the Soles
interdigital scaling Mild Form — Complete loss of tissue

T. mentagrophytes

⇒ Hyphae [Molds]

Diagnosis

Microscopic Examination
- Branching Hyphae are found among Epithelial cells of skin & nails.
- Hyphae or spores are found

Culture
Sabouraud's Dextrose Agar [SDA]
Staining with Lactophenol cotton

B. Cutaneous Candidiasis

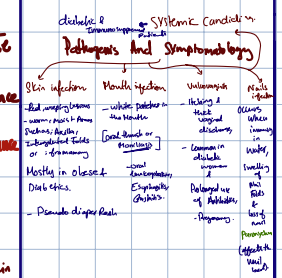
↓

Candida Albicans

the Most imp Species of Candida

Candida Albicans are oval gram-positive Yeast producing pseudohyphae when they become pathogenic

* It is a member of the Normal Flora that may go to sterile sites.
* it colonizes the Mucous Membranes of upper-Respiratory tract, GIT & Female genital tract
* Opportunistic Fungi
* Predisposing Factors to Candida Infection
1) Diseases as Aids & Diabetes Mellitus.
2) Drugs; Broad spectrum Antibiotics & Corticosteroids.
3) General debility; ICU
4) Indwelling Urinary or vascular Catheters



Diagnosis

Direct Microscopic Examination
Specimens from skin, Vaginal discharge or Exudates from mucous surfaces are Examined
C. Albicans are oval gram⊕ budding yeast cells with Pseudohyphae.

Culture
on Nutrient Agar, Corn Meal Agar & SDA, Creamy in color.
↓
Identified by:
1) Morphology; oval gram⊕ Budding Yeast
2) Differentiation tests:
a. Germ tube test
b. Chlamydospore; Formation on Corn Meal Agar
c. Biochemical Reactions
C. Albicans Ferment glucose & Maltose with Acid & Gas Formation

3 Subcutaneous Mycoses

A. Madura Foot
B. Rose handler disease

↓

Madurella Mycetomatis

Madura Foot →

Sporotrichosis → Sporothrix Schentii

↓

- Dimorphic Fungus; Yeast inside the body & Molds outside the body.
- The Fungi introduced the subcutaneous tissue through Trauma.
- Pathogenesis; traumatic implantation of Sporothrix schentii that normally resides in Roses; then they start Forming Small nodules [Chronic granulomatous Reaction]
- Rose Gardener's disease.
- Small nodule develops at the site of Trauma & week to 6 months after inoculation; then Series of Secondary nodules along the lymphatics that drain the site.

* Acanthosis Nigricans

↓

May be confused with tinea versicolor, it is in the Axilla, and the back of the neck.

↓

Sign of insulin Resistance & Related to T2DM

[illegible]

- They look under the Microscope as thick septate hyphae and Yeast clusters. [Spaghetti and Meatballs]

* Treatment:
  1. Some they disappear Spontaneously
  2. Topical Azoles for 2 weeks or Oral Azoles in severe Cases.

- Recurrance is common.

Seborrheic dermatitis

Skin hyperproliferation with hair dandruff

- lesions are Red and Covered by greasy Scales And itching is common

M. Furfur

- Treatment: Azoles

→ The Relationship between M. Furfur & Seborrheic dermatitis is Association. not Causation

in the hair.

Spores inside the hair Endothrix

Spores outside the hair Ectothrix

→ They Are Examined after digestion using 10% KOH

Treatment: Local Antifungal Creams; Miconazole oral; Terbinafine.

Common Dermatophytes

- Epidermophyton → Bifurcated Hyphae, Macroconidia 2-8 cells
- Microsporum → Spindle Shaped Multicellular
- Trichophyto → pencil-shaped

Treatment

- Oropharyngeal → Nystatin Fluco
- Skin lesions → Nystatin Ointment
- Systemic Candidiasis: Caspo... IV; Ampho... IV; Keto... only

Written by Jude Kirresh ♥

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