بسم الله

* micro lec.1

→ mycobateria → Rod shape 0.3–3 μm
→ aerobic
→ no spors

قضيب | M

* MTC → cuse T.B → strong
* affect mainly immune comprimised
* MTB = koch bacillus
* stained by acid fast stain → zn stain → 95% CH_3-CH_3 / ≈ 5% HCl → Red color
* consumbtion → consume patient / weight loss
* white plague → pallor seen

بوبو بقرة

* M. Bovis → from cow → if it infect us → abdominal pain
→ when pastorization have done → not found.

* slow multiblication → 18 for dubling

* obligate intra cellular → non motile
→ " capsolated
→ " spor forming

* ZN.stain → Red stain → carbonfuchsin
→ heat → for good penetration

* cluteral MTB are most specific but take long time (4–6) wek

MTB: Rised, rough, clumber in colony

* inner membrane → PG peptidoglycans
→ AG arabinogalactance
→ mycolic acid MA

complex ← highly croos-liked

→ arabinan + LCMA → thik lipid → impermiability *
→ (70–90)%
→ 50% dray mass of the cell wall

* type 7 secrition
→ TDM → clumbing morphology

→ Hematogenus → can cause T.B menigites → the worst

* tranmition → airbone
→ unpasturized milk
→ Direct inoculation

* 10 / 3 \ 1 → alive
exposed, latent

→ Pathology:

main hallmark is granuloma formation

* M → no (end + exo) toxin
* in primary infection → Begin in Base of lung
* in 2° nd reactivation → " " apex " " → more O_2

* clinical manifistation
– Hemoptisis
– night sweats
– malaise \ fatign
– dyspnaa

→ so times acording to the infected organ

→ laborotory diagnosis

* ZN stain
→ for domonostration → yellow flurescence after staining with auramine

→ Treatment

* first phase (intinsive) → 2-month
→ isonazid (INH)
→ Rifampin (RIF)
→ pyranazamide (PZA)
→ ethambutol or streptomycin SM / EMB

* 2nd " (continuation) → RIF, INH

* these durgs have side effct → INH → cause hepatitis
→ RIF → Red urin

* Isoniazide preventive therapy for
a) LTBI
b) children whose parent have T.B
for 9 month

* the prevention
→ best way is dignos + isolate
→ BCG vaccination and treatment of LTBI
* BCG → the only vaccine → live attenuated
→ have diffrent degrees of efficacy (0–80)%

→ other. m → non NTM → environmental T.B → not contagus

* classified into → fast → < 7
→ slow > 7

* pigment production
– photochromogenus: need light to pigment
– scotochromogen: no pigment at all

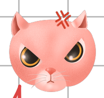
* M. lepRAE → cause leprosy → insidus infection
– acid fast Rod
– only in animal modle → no vitro not clutured.
* intra cellular

– she like to be coold site → closer to skin
and Schwan cells of sensory nerve → sensory loss
* the severity depend on cell mediated immunity

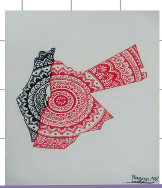
* leprosy types
– TL – the least sever → strong cell immunity
L.L ← lepromatous . L ⇒ most sever → weak cell immunity
– Borderline . L : intermediate

* clinical manifistation
* lesion on the body
  → skin: Painless, nodules
  → nerve: loss of sensory

→ Pathiant have the lion like face



* Dignosis → Biopsy are smered and staind with Z.N stain
* side note: serology not used on intracellular infection
→ Treatment
• Dapson → first line therapy
• RMP



*lec.2 introduction to mycology

* mycoses → disease by fungi → less than 300
  → allergis
  → toxins
  → infection

* most of thes infetion subclinical, insidius
→ most of infection are harmless

* fungi
  → eukaryotic → nucleus, 80s Ribosome
  → Don't have chlorophyll
  → Heterotrophic

* fungi
  → Yeast → unicellular → Reproduse by budding
  → molds → multi cellular
  → Dimorphic → mold in cold and yeast in heat → reversable

* Reproduse by spores
– sexular → telomorphic
– asexual → anomorphic

* cell wall → chitin, β-glucan, manan → antiphagocytic
* cell membrane: they have ergestrol in cell.m → P, A, A
* mainly they obligatory aerobes
* Pinicilin from fungi

→ so they are opportunistic
* immuno comprimised people more likly to be infected
* fungal infection → no Anti-B response

* Yeast infetion ex:
  → Candida ablican → endogenus
  → creptococucus.neoformus → exogenus → not normal flora → intial lung infection

* mycelium: mass of branching

* Dimorphic fungi
  → Blastomyces dermatitids
  → Sporothix
  → Histoplasma capsulatum

* fungal diseases → especially in immunocomprinised
– allergie: IgE, hypereosinophilia, Bronco constriaction → asthma
  → notablein Asperigllus fumigatus
– Toxin: main groups is aflatoxin → made in Aspergillus → flavus, parasiticus
humidity + tempriture = good condition
* nuts, peanut, corn → contamination
* aflatoxin in liver → epoxid → potant carcinogenic
  → induse mutation P53 → tumer suppresor gene
  most naturry occuring

** * fungal infection (most important)
increase use of antibiotics, immunosuppresor drugs → high chance to be infected.

– a
  → superficial → no immune response, no destruction
  → cutanus → minor symptoms + changes + immune response
    → ex: cutaneous candiasis → between fingers (due to frequent washing)
    → ex: dermatophytes + candida albicans
    immunity are present
– b. subcutanus
  → tromatic immplantation → epithelial barrier distroy
  → cornea, muscles + joints, hypodermis
  → no dissemination
– c) systimic (endimic) mycoses → North + South Americ
* By dimorphic fungi + can affect healthy people
* Par- histo- coccidi- B a&l
* infection starts in the lung

* Dignosis
– clinical investigation → no respond to antibiotic
– labotatory
  → PCR
  → microscopy
  → serology
  → culture → the best

* types of specimen: skin scalp, nail clipping,
* hair stubs are most specimen for dignosis of skin infection \ Rig worm also
* Subcutanus
  → most suitable are scarping and curst
  → fluid → pus
  → tissue → biopsies

* Systimic specimen must be from right site
* Stans and direct microscopic examination
* we use koh for partial disestion
* addition of calcofluor white (universal)
and use of fluoresence microscopy for bater detection ⇒ they bind to cell wall chitin part
* other
- India ink ⇒ for cryptococcus neformus
- PAS, CH3lenblue

we can use
- cycloheximide: ↓ funsi that are normal flora
- chloramphenicol ↓ for reduse bacteria

*antifunsar therapy
note: Bacterial durs have no effect on funsl infection
- the fungl durs
  - highly toxic
  - narrowth

Classified into six groups:

Type of antifungal	Mechanism of action	examples
Polyenes derivatives	The only Fungicidal; will KILL the fungi. They would bind the membrane ergosterol → disrupt the plasma membrane of fungi.	Amphotericin B → given IV in endemic mycoses or deep opportunistic mycosis. Systimic. Nystatin → topical
Azoles	Fungistatic; inhibit the growth without killing. They inhibit the synthesis of ergosterol	Ketoconazole (كيتو ك زول) Fluconazole Itraconazole Voriconazole Posaconazole
Griseofulvin (Extracted from fungi) penicillium	Fungistatic Given for superficial mycotic	Prescription is NOT common
5-flourocytosine (5-FC) F	Fungistatic , Inhibitor of DNA and RNA synthesis	Chemotherapeutic agent given to cancer
Allylamines	Fungistatic. Inhibit the enzyme for ergosterol synthesis; squalene epoxidase.	Terbinafine (Lamasil) Commonly prescribed
Echonocandins For dermatophytosis (superficial and cutaneous)	Fungistatic. Disrupt the cell wall by inhibiting β-glucans synthesis (1,3-β-glucan synthase).	Caspofungin

lec.3 - funsal infection -
*suberficial infection → affect staratum corneum also no immune respons, no invasion nor distruction
ex: Tinea vesicoler or pityriasis vesicoler
Cause malassezia complex
- m.furfu, m.globosa, m.srmpodialis
*Suberficia malassezia n infection
- they are
  - lipophilic yeast, rounded, normal flora not contasus
  - when they became pathogenic
short angular hyphal form ⇒ spaghetti + meatballs

most commonly → superficial
Pityriasis versicolor
- only affec stratum corneum mainly trunk also proxima lims surface
- Common in tropical site
- Pricipitated by sun

* Production of carboxylicacid → lesions de pegmentation
*clinically
- asymptomatic → non itch hypo, hyper pegements ⇒ Colorless
  - cucasian
  - Dark People

*Dignosis → spaghetti and meatballs
- ink stain

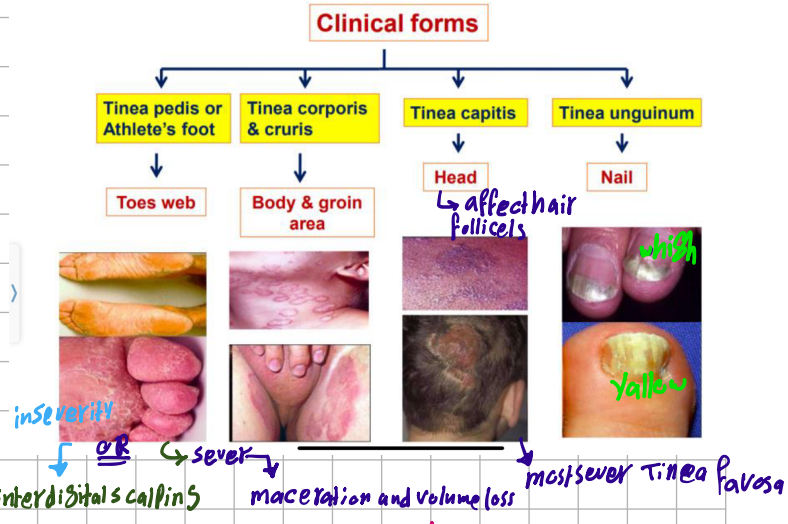
*treatment (سببه بيموت لحاله)
- azole
- azole containing things

*they have ↑ reoccurrane rate
→ Seborrheic dermatite
*mfferfor (تظهر بس لما انو السبب يطلع له)
*skin hyperproliferation + danduff
- Red lesion

→ Cutanus mycoses
*Ringwormor Tinea
• from Dermatophytes → molds only
*3 Senera
- epidermatophyton
- microsporum
- Trichphython

*mainly affect keretinized tissue
- skin
- hair
- nails

*Ring shaped, no desamination, Contagious
*Source of infection
- man to man Anthrophilic → chronic but mild, high recurnt
- Zoophilic
- Geophilic



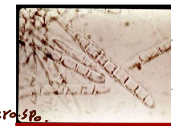
*tinea barbea ⇒ affect hair and beard
* Red, itchy, Ring like rashs, more inflamed
* Dignosis
- skin, nails are microscopically examinated after use in koh for partial digestion (10%)
- Branching hyphae can be detected (no yeast)

* spores can be → ectothrix → out side hair shaft
→ endothrix → within hair shaft → ex: T. capitis

* Dermatophytes

Epidermophyton floccosum: Bifurcated hyphae with multiple, smooth, club shaped macroconidia (2-4 cells) multi cellular

Microsporum: Thick wall spindle shape multicellular



Trichophyton: Large, smooth, thin wall, septate, pencil-shaped

* culture → Sabouraud's Dextrose agar (SDA) 4 weeks at room tembreture, and the colonies are examined after staining with lactophenole cotton blue stain

→ Subcutaneous infection

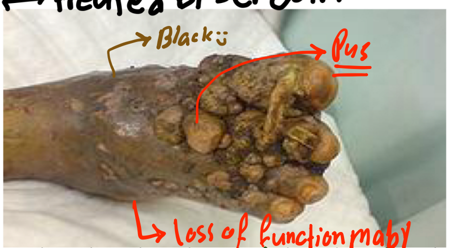
* sporotrichosis → Ross garden disease

* mycetoma →
- chronic granulomatus inflam...
- formation of hard noduls
- fungi from soil → farmers
- infection introdused by trauma

madura foot

can be from →
- Eumycetoma → fungi → the nodul is no pain
- Actinomycetoma → Bacteria → Painfull

* clinicly → treated by sergery



* Dignosis →
- macro.s → Black granules
- micro.s → septate hypae, with spors
- culture → SDA

* treatment →
- ketoconzole → oral (static)
- Intraconzole
- Amphotericin B → I.V → for sever case
- surgical

* opportunistic mycosis → in immunocompromised
ex: taking immuno suppressive lik corticosteriod.

* Candidiasis

- C. albicans → most importat one → oval gram + budding yeast which produse pseudo-hyphe
- Part of normal flora in mucos membrane
→ Predesposing factor →
- diabetes, AIDs
- Durg
- Debility
- Indwelling urine cather

Pathogenesis & Symptomatology

Most common

Skin invasion	Mouth infection	Vulvovaginitis	Nails infection	Systemic candidiasis
-They are red &weeping lesions. -Mainly affect worm moist areas. Such as axilla, intergluteal folds or infra mammary folds. -Mostly in obese & diabetics. -Pseudo diaper rash	C. albicans produces white patches in the mouth (oral thrush or moniliasis). Sometimes oral leukoplakia, esophagitis, gastritis	-With itching & thick vaginal discharge. -Common with diabetic woman & prolonged use of antibiotics, IUCD, Pregnancy. (IUCD → Interuterine Contraceptive device)	-Occurs with repeatedly immersing in water (dish washing). -Painful redness, swelling of nail folds, thickening & loss of nail (paronychia).	Occure in diabetics & Immuno-suppressed persons.

* Dignosis
- culture → morphology: oval budding gram + yeast cell; white creamy
- Germ-tube formation is a differential

Tratment

Oropharyngeal or oesophageal thrush	Skin lesions	Systemic candidiasis
Nystatin, Fluconazole ont	Nystatin ointment	Caspofungin IV, Ketoconazole (orally), Amphotericin B (IV)

* Cryptococcus neoformans →
- exogenus
- via inhalation
- opportunistic infection

→ yeast that inhibits soid around pigeon roosts
→ able to case meningitis → can case infection systimic

→ Dignosis
a) microscopic via india ink for capsule stain (50-80% in CSF) sensitivity
note: this fungi is the only encapsolated
B) cluture → Brid seed agar, Pcr, Routin blood agar

Aspergillosis → 600, 8 involved in human disase, mainly A. fumigatus
Flavas, Paraciticus
- mainly lung - spores → colinse earcanal
- opportunistic infection
- Bronchopulmonary allergy, OR invasive form → IgE
- surgery in sever form
- Amphotrisin - Binystan

Smear, Biopsy

Dignosis

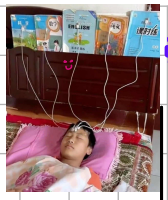
mucoromycetes → main hard defence → macrophage

* zygomycoses →
- saprophytic, found in: soil, H2O, food, organic debris
- the genera involved: Rhizopus, Absidia, mucor
- harmless air contaminant invade → Rinocerebral mucromycosis (nose, eyes, hard, Brain)
- mainly in diabets, malnutrition → sever results

Treatment
- control Diabetes
- surgery, amphotericin B
- very poor prognosis

* Pneumonocytes →
- can't be cultured ex vivo or isolated in lap, we can can see them in eosin methylin blue as honey comb or silver stain
- TMP_SMX → tretment

*lec.4 "Parasitetology"



- endoparasite → inside
- ectoparasite → outside

• Protozoa → unicellular

- types: movent classification

a) Sacrodina (Rhizopoda) → Pseudopodia
↳ ex: E. histolica

b) mastographora → flagella
↳ ex: Giardia lamblia

c) ciliates → ex: Balantidium coli
(active)

d) Sporza → Gliding mechisim → no organ for it
→ intracellular only

→ a, b, c → asexula
→ D → " + sexula

• Hiliminths → metazoa (multicellur)

- divided into:

Round worms
a) Nematoda → Intistinal nematods → ex: Ascaris
→ Tissue nematod → ex: wuncherein
→ Sefrate gender ♀, ♂

Tape worms
b) Cestoda → Segmented worms → important indignosis
↳ ex: Taenia saginata

c) Trematoda → flattend-shaped worms → ex:
no separte sex
Hermaphrodites exception → Schistosoma

• Arthropods → have exoskeletone + jointed

* Calss:
Insecta: Mosqutoes, lice (القمل), fleas (البراغيث)
Arachnida: Ticks, mites → T.M

* Temporary or Intmittent Parasite → stay short time for food, can live outside

* Coprozoic, spurious → Pass through the human intistinal canl with no symptoms + detected in stool

• Hosts

- Definitive H → harbours mature adult stage or where sexul Reproduction occur
- Reservoir H → Source of infection like dog in Leishmania donovani
- Intermediate H → have lavar stage → ex: snail
- Accidintal H → not normal infect us
ex: Toxo cara → dog infect

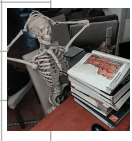
*Relations → ex: entomobacolia

a) commensal: organism get benefit and host not harmed
b) Parasitic → " " " " Harmed
c) mutulism → Both benfit

*transmition

- → Direct Skin Contact
- → Pentration → Vectors
- → ingestion → Bood + contaminiate syring
- → inhalation → organ trans plantation
- → conjnital
- → sexual contal
- → Autoinfection

* Habitat: home
* Carrier: invective + host
* Zoonosis



*Pathogensis

- mechanical → obstraction of normal Passage
- Traumatic effect → internal → via attachment → ulcer form
→ external → invasion of skin
- Toxin Production, tissue damage
ex: dysentry
- cellular distruction: Plasmodim in RBC, REC damage
- Immune response: hepatic Granuloma
- Allergy → IgE
→ hyper-eosinophil

*Dignosis → clinical → sign
→ Laporatory

- Stool → minly in intestinal infection
→ macroscopic → see an adult parasite
ex: T. segmnt, Ascaris, entero bis vermicularis
→ micro-s → for eggs, cyts
must be prepared

* Urin
ex: Schistsoma
Trichnomonas vaginalis → in vagina and can be detected in urethra

* Blood → this → for give info about morphology
→ thick → for detection
→ malaria, Leishmania, Tryanosome,

* Tissue biopsy → muscle biopsy
→ Rectal
* Sputum → Related to lung
* Aspirates → csf → in Trypanosoma rhodeniense
sleeping sickness
→ Duodenal → in G. lamblia, Gryptosporidium parvum