Micro summary
All final material except L4

🔻 هذا الملخص شامل تقريبا 95% من السلايدات + نسبة كبيرة من كلام الدكتور ما عدا ال diagnosis للامراض (باستخدام سكتشي)

صلّوا على سيدنا محمد و استعينوا بالله

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كبروا السلايدات الخط صغير 🤦

L1

① cholera

Vibrio Cholera - Colonel Cholera's Base Camp → Slightly curved

السنارب

It is actively motile by means of a polar flagellum called shooting star motility

1. Mustache - COMMA SHAPED Gram Negative Curved rod Enteric Tract Bacilli
2. BASE in BASE cAMP – Prefers to grow in alkaline media
3. Blue Ring - Oxidase Positive - Grows on TCBS agar → produces yellow colonies (sucrose fermented)
4. Lemon - Grows in alkaline environments, ACID LABILE - Dies with acid
5. Rice Patties - Causes Profuse watery diarrhea **"Rice Water"** stool and nausea and vomiting.
6. Outhouse dumping directly into the river - Cholera is transmitted fecal oral due to poor sanitation that gets into food and is not an invasive infection
7. River walls are mucosal wall and the water is the intestinal lumen - Found in the intestines and is found in the intestinal mucosae
8. Raft that is attached to the shore - Attaches to the mucosa by fimbriae that attach to ganglioside receptors in the intestinal wall.
9. Then releases **cholera toxin - Main Virulence Factor AB type toxin**
   a. BASE cAMP map - Upregulates production of Gαs cAMP by binding to and increasing activating adenylate cyclase.
   b. GS grenade - Then it will activate the GS pathway. Activates GS, upregulates cAMP, Produces watery diarrhea through an efflux if Cl and H2O
10. Treatment
    a. Drinking some water - Oral rehydration therapy with electrolytes → الدكتور ركز عليها كتير
11. Vibrio Vulnificus and paraliticus
    a. Oysters - Can contaminate seafood, especially oysters.
    b. Vibrio V. causes Acute Gastroenteritis
    c. Vibrio P. Causes fulminating septicemia leading to death. Marked edema and necrosis

choleragen. Derived from bacteriophage, called ctx which infects the Vibrio first.

The toxin-coregulated pilus (TCP) is essential for V. cholerae to survive and multiply in (colonize) the small intestine.

1. Ganglioside GM1 is a mucosal receptor for subunit B, which causes toxin internalization, which promotes entry of subunit A into the cell.
2. Activation of subunit A1 >> activtes Gs >> increases levels of (cAMP) and results in prolonged hypersecretion of water and electrolytes.

→ + antimicrobial → tetracycline and doxycyclin but in Pregnant → erythromycin

- V alginolyticus occasionally causes localized eye, ear, and wound (cellulitis) infections.

■ V cholerae serogroups O1 and O139 cause cholera in humans . Non-O139 strains cause mild, self-limiting cholera-like gastroenteritis

■ Most Vibrio species (like cholera) are halotolerant, and NaCl often stimulates their growth. Some vibrios (parahaemolyticus and vulnificus) are halophilic, requiring the presence (high concentration) of NaCl to grow (oceanic water).

■ Two biotypes of V. cholerae O1, classical (50% of infected mild, 50% asymptomatic) and El Tor (75% asymptomatic, 25% typical cholera symptoms), are distinguished Each biotype is further subdivided into three serotypes, termed Inaba, Ogawa, and Hikojima.

■ Prevention

1. safe water and food.

2. Patients should be isolated.

3. Three oral killed cholera vaccines:

• WC-rBS (Sweden) contains several biotypes and serotypes of V. cholerae O1 supplemented with recombinant cholera toxin B subunit.

• BivWC (India) contains several biotypes and serotypes of V. cholerae O1 and V. cholerae O139 without supplemental cholera toxin B subunit.

• Vaxchora, a live-attenuated vaccine, approved only in the USA by the FDA. It contains O1 serotype, typically Inaba strain.

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(2) Campylobacter Jejuni

▪ Motility (dart motility)

▪ C jejuni and C coli cause infections that are clinically indistinguishable (from names, we can infer C. Jejuni affect the jejunum and C. Coli the colon).

→ Clinical findings

▪ profuse diarrhea at first that may be grossly bloody afterward.

▪ Local suppurative complications of infection include cholecystitis, pancreatitis, and cystitis, Hepatitis, interstitial nephritis, and hemolytic-uremic syndrome, Guillain-Barré syndrome, a form of ascending paralytic disease , reactive arthritis and Reiter's syndrome.

Campylobacter Jejuni - Camping Guy and the bears – guy and bears = Guillen Barre (S, or "gull wing" shape)

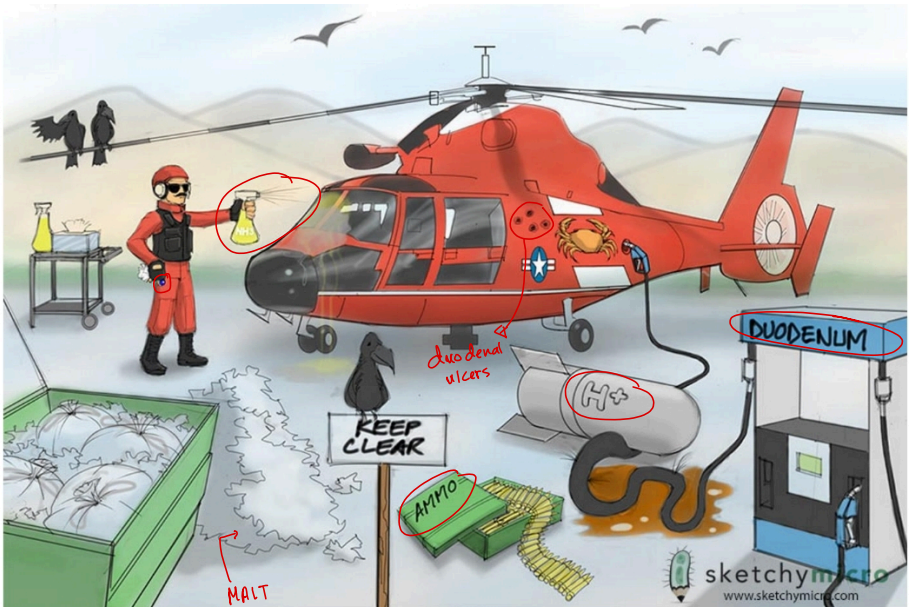
1. Mustache is curved or comma shaped - Gram Negative Spiral / Curved Rod Bacilli - Enteric
2. Campy medium or **Skirrow Agar** (Skirrow's (has 5 antibiotics, a few bacteria survive), Butzler's, Blaser's, Campy-BAP, and Preston media
3. Microaerophilic (high co2 and lower o2)
4. Camp Fire - Prefers warm environments around 42 deg Celsius, thermophile (Special Incubator)
5. Chicken being cooked - Main reservoir is intestinal tract of poultry and transmission is fecal oral / also contaminated water supplies or ingestion of raw milk
   - Domestic animals are used for food (including poultry, cattle, sheep, and swine) and many household pets (including birds, dogs, and cats).
   - The infection is acquired by the oral route from food, drink, or contact with infected animals or animal products, especially poultry.
6. Red Stools - Bloody Stools and diarrhea
7. Blue Ring - Oxidase Positive
8. Bear cub invading the cooler - Can get Bacteremia, INVASIVE
   a. Laughing and slapping his knee - Reactive arthritis, riders syndrome
9. Bears being tripped by the sausage links on his ankle - Can cause Guillen barre syndrome due to an autoimmune response damaging myelin of peripheral nerves leading to an ascending paralysis will start at the feet then ascend.
10. Pathogenesis
    a. Bacteria Colonize intestinal Mucosa and attach to epithelial cells then replicate intracellularly causing an acute PMN response, edema of the mucosa and ulcerations. Presenting with acute enteritis and diarrhea (Localized tissue invasion coupled with the toxic activity (classic enterotoxins and cytotoxins (cytolethal distending toxin, or CDT)) → inflammation
11. Treatment
    a. Supportive Care

- Fluid and electrolyte replacement.
- not all patients clearly benefit from specific antimicrobial therapy. Indications for therapy include high fever, bloody diarrhea, severe diarrhea, signs of dehydration, persistence for >1 week.
  >> Erythromycin.
- For systemic infections, treatment with gentamicin or imipenem or chloramphenicol should be started for C. Fetus infections.

read them

- Campylobacter jejuni and Coli is the prototype organism in the group and is a very common cause of diarrhea in humans.
- Campylobacter fetus has two subspecies, fetus and venerealis (transmitted by venereal, means sexual, route). C fetus subspecies fetus is an opportunistic pathogen that causes systemic infections (e.g. sepsis) in immunocompromised patients. It may occasionally cause diarrhea in affected healthy individuals.
- Other organisms that cause diarrheal disease include Campylobacter coli, Campylobacter upsaliensis from dogs, Campylobacter lari from seagulls, Campylobacter hyointestinalis, Campylobacter fetus, Arcobacter butzleri, Arcobacter cryaerophilus, Helicobacter cinaedi, and Helicobacter fennelliae (called Helicobacter but cause diarrheal/intestinal disease; H. Pylori in stomach and cause peptic ulcers). Just know that helicobacter has been separated and Arcobacter and Campylobacter kept together.

(3) H.pylori



- Urease , oxidase, and catalase positive
- microaerophilic (i.e., requires low levels of oxygen).
- if pain increases with eating, then it is a gastric ulcer, while if it decreases, it is duodenal.
- Most H. pylori-colonized persons do not develop clinical sequelae. That some persons develop overt disease whereas others do not is related to a combination of factors:

Helicobacter Pylori: The helicopter Pilot

1. Red Helicopter -Curved Gram Negative rod
2. Mustache that is comma shaped - Helical slender curved rod Shape found in pylorus of the stomach
3. Not a rare infection.
4. Helicopters are motile - Motile by way of flagella
5. **Ammonia bottle - Urease positive - MAJOR VIRULENCE FACTOR** - allows to reduce the acidity of the stomach and allows Helicobacter to survive there.
6. Can be tested with Urea breath test, radioactive urea that is broken down and exhaled as CO2 and NH3 by urease positive organisms.
7. Blue ring - Oxidase Positive – all curved rods are oxidase positive
8. Bullet holes in helicopter, Gas pump w/duodenum - Causes 95% of all duodenal ulcers ↳ and 60%-80% of gastric ulcers
9. Crab - Mechanism of Chronic infection causes increased acid infection. At risk of developing gastric adenocarcinoma
10. Tissues that are thrown in garbage - Patient can develop lymphoma of mucous associated lymphoid tissue. (MALT) lymphomas (also called MALToma: Non-Hodgkin B-cell lymphoma)
11. Treatment
    a. Gas Pump with duodenum and H+ Bomb - Proton Pump Inhibitor
    b. Amoxicillin - ammo
    c. Crow w/ Keep Clear - Macrolide - Clarithromycin
12. Transmission
    a. Fecal Oral or Oral

Humans are the only important reservoir of H. pylori

- it may be one initial precipitant of pernicious anemia and also may predispose some patients to iron deficiency .

Pathogenesis (path)

- H pylori is found deep in the mucous layer near the epithelial surface where physiologic pH is present (away from low pH) and multiply in gastric pits (glands).
- H pylori is quite motile, even in mucus, and is able to find its way to the epithelial surface. H pylori overlies gastric-type but not intestinal- type epithelial cells.
- H pylori also produces a protease that modifies the gastric mucus and further reduces the ability of acid to diffuse through the mucus.
- H pylori produces potent urease activity, which yields production of ammonia and further buffering of acid. Also, it has mucinase to navigate through the mucus.

- The mechanisms by which H pylori causes mucosal inflammation and damage are not well defined but probably involve both bacterial and host factors. The bacteria invade the epithelial cell surface to a limited degree. Toxins and lipopolysaccharide may damage the mucosal cells, and the ammonia produced by the urease activity may also directly damage the cells.
- Polymorphonuclear and mononuclear cell infiltrates are seen, causing acute and, in the long run, chronic gastritis leading to the complications we stated before within the epithelium and lamina propria. Vacuoles within cells are often pronounced. Destruction of the epithelium is common, and glandular atrophy may occur. H pylori thus is a major risk factor for gastric cancer.
- H. pylori colonization induces chronic superficial gastritis, a tissue response in the stomach that includes infiltration of the mucosa by both mononuclear and polymorphonuclear cells.

- An acid-suppressing agent given for 4-6 weeks enhances ulcer healing. Proton pump inhibitors (PPIs) directly inhibit H pylori.
- The preferred initial therapy is 7-10 days of a PPI plus amoxicillin and clarithromycin (Triple therapy) or a quadruple regimen of a PPI metronidazole, tetracycline, and bismuth subsalicylate for 10 days

L2

① Brucellae

The disease in humans, brucellosis (undulant fever, Malta fever Mediterranean fever, and Cyprus fever)

- It forms Granulomatous nodules that may develop into abscesses form in lymphatic tissue, liver, spleen, bone marrow, and other parts of the reticuloendothelial system.
- The granulomas form and consist of epithelioid and giant cells, with central necrosis and peripheral fibrosis.

Bruce Farms - Brucella

short coccobacillary, aerobic, nonmotile, and nonspore forming

1. Bruce farms - red to remember it is Gram neg
2. Farm animal is the reservoir - cows and pigs, goats, veterinarian, slaughterhouse worker, or rancher.
3. Milk Bucket on the ground - Indirect contact with milk or cheese products that unpasteurized / also it is transmitted by ingestion, inhalation, skin and Accidental injection of the live vaccine
4. Open Cage on Barn house - Facultative intracellular can live inside or outside of host cells
5. Symptoms - fever, chills, and anorexia initially.
6. Undulating hills - Undulant fever + Sweat (undulant fever : their degree rise afternoon and drops in the night)
7. Markings on the cow - Can travel through multiple endothelial organs leading to enlargement of spleen, liver and lymph nodes / Hepatitis with jaundice.
8. Fish Bones - Osteomyelitis - chronic infection → Deep pain and disturbances of motion
9. Wheel -Treatment - tetracycline, doxycycline (Combined treatment with a tetracycline (eg, doxycycline) and either streptomycin for 2-3 weeks or rifampin for 6 weeks is recommended)
10. Rifle - Along with rifampin for primary treatment blocks oxidative bursts
11. Cage in the background - Infect macrophages
12. Large amounts of catalase and superoxide dismutase to protect from respiratory burst
13. Urease and H2S positive
14. Require cO2 to grow (campylophic conditions (increase CO2 concentration))

- Brucella melitensis infects goats; Brucella suis, swine; Brucella abortus, cattle; and Brucella canis, dogs.
- chronic stage may develop, characterized by weakness, aches and pains, low-grade fever, nervousness

Diagnosis:

A. Specimens : Blood should be taken for culture, biopsy material for culture (lymph nodes, bone, and so on), and serum for serologic tests.

B. Culture : Brucella agar , specifically designed to culture Brucella species bacteria. The medium is highly enriched.

C. Serology: (IgM) antibody levels rise during the first week of acute illness, peak at 3 months, IgG and IgA antibody levels rise about 3 weeks after onset of acute disease, peak at 6-8 weeks, and remain high during chronic disease.

Agglutination test ① and ELISA assays ② (more sensitive and specific than the agglutination test)

- Prevention and control

1. active immunization of heifers with avirulent live strain 19, or combined testing, segregation, and immunization.
2. pasteurization of milk and milk products, and reduction of occupational hazards wherever possible

(There is no vaccine for humans)

② Leptospira

Leptospira comprised two species: the pathogenic L. interrogans and the free-living L. biflexa

- Kidney involvement in many animal species is chronic and results in the shedding of large numbers of leptospirae in the urine; this is probably the main source of environmental contamination resulting in infection of humans.
- Human urine also may contain spirochetes in the second and third weeks of disease.

Leptospirosis: The Surfers Oasis

They are actively motile, which is best seen using a dark-field microscope.

1. Question mark on the board - Spirochetes may be question marked shaped
2. Surfing in the water - Water sports,
3. Yellow tide - water contaminated with animal urine'
4. Surfer rubbing his eyes, rose colored sunglasses and dripping wet - Fever and conjunctival suffusion redness around the eyes without the puss
5. Hawaii - Tropical regions (tropics and subtropics because of the climate)
6. Water (Leptospirae can survive for weeks in water, particularly at alkaline pH)
7. Whale – Weil's disease (A small percentage of infections (~1%) lead to severe, potentially fatal complications.)
8. Inner tubes that look like RBC's – travels in blood stream → Hemorage
9. Rubber dingy shaped like a kidney - Renal dysfunction → nephritis
10. Yellow suit - jaundice from live damage → Hepatitis

→ Triad

- It is biphasic : it has 2 phases –> leptospirosis phase (circulated in blood circulation, then they establish themselves in parenchymal organs, it also named , they prefer liver and kidneys) and parenchymatous phases .

Treatment

- mild >> oral doxycycline, ampicillin, or amoxicillin.
- Severe >> IV penicillin. Why? Because leptospira has penicillin binding proteins

Prevention : (mainly by control of rats as they are the main reservoir).

- Avoidance of exposure to urine and tissues from infected animals through proper eyewear, footwear, and other protective equipment.
- Vaccines for agricultural and companion animals are generally available.

(There is no vaccine for humans)

③ Mycobacterium Tuberculosis (Mtb) Koch bacillus.

without sketchy 🙃

- Cases Tuberculosis , consumption(consume patients ,weight loss), white plaque (extreme pallor seen among patients).
- The family mycobacterium tuberculosis complex(MTC) can cause tuberculosis (TB) in humans and other livings.

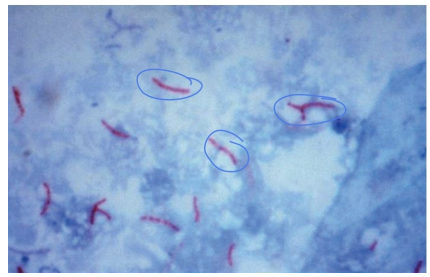
→ airborne infectious disease

Principal member (main cause of tuberculosis) يصيب الأبقار / والأشخاص اللي بشربوا حليب غير مُبَسْتَر

- It includes M. tuberculosis (Mtb), Mycobacterium africanum, Mycobacterium bovis, Mycobacterium microti, Mycobacterium caprae, Mycobacterium pinnipedii, Mycobacterium suricatte, Mycobacterium mungi, Mycobacterium dassie, Mycobacterium oryx and Mycobacterium canetti.

- Mtb is a slow growing ,obligate aerobe, facultative intra- cellular bacterium, non-spore forming, and non-motile acid fast bacilli.

- Acid fast bacillus (Ziehl-Neelsen technique). The first dye in it is Carbolfuchsin dye (it's red dye).
  → diagnosis



- Two TB-related conditions exist; latent TB infection (LTBI) and TB disease. People who have latent TB infections do not feel sick, do not have any symptoms, and can not spread TB to others.

→ 10:3:1 كل 10 اشخاص بنصابوا بال TB

لـ (3) منهم → Latent و (1) → Active و (6) → Clear the infection

- Tuberculosis TB
  - 80% Pulomonary (lung)
  - 20% extraPulmonory → 5%-10% → GIT

>> The abdominal TB occurs in four forms: tuberculous lymphadenopathy, peritoneal tuberculosis, gastrointestinal (GI) tuberculosis and visceral tuberculosis involving the solid organs

→ Pathogensis

- Tuberculosis of the abdomen may occur via reactivation of latent TB infection or by ingestion of tuberculous mycobacteria (as with ingestion of unpasteurized milk, or sputum or undercooked meat). In the setting of active pulmonary TB or miliary TB, abdominal involvement may develop via hematogenous spread via contiguous spread of TB from adjacent organs (such as retrograde spread from the fallopian tubes) or via spread through lymphatic channels
- The mucosal layer of the GI tract can be infected with the bacilli with formation of epithelioid tubercles in the lymphoid tissue of the submucosa. After 2-4 wk, caseous necrosis of the tubercles leads to ulceration of the overlying mucosa which can later spread into the deeper layers and into the adjacent lymph nodes and into peritoneum. Rarely, these bacilli can enter into the portal circulation or into hepatic artery to involve solid organs like liver, pancreas and spleen

Gastrointestinal TB clinical finding

- The clinical presentation tends to be non-specific, with abdominal pains and general complaints.
- Although any portion of the gastrointestinal tract may be affected, the terminal ileum and the cecum are the sites most commonly involved. Abdominal pain (at times similar to that associated with appendicitis) and swelling, obstruction, hematochezia, and a palpable mass in the abdomen are common findings at presentation. Fever, weight loss, anorexia, and night sweats are also common.

Treatment of TB usually involves a drug cocktail :
intensive initial 2-month phase (→ 4 drugs) followed by a slower 4- to 6-month continuation phase (→ 2 drugs → INH and RIF.)

- Isoniazid preventive therapy IPT is the recommended treatment for LTBI, but the regimen's main drawback is the duration of therapy

→ isoniazid (INH), rifampin (RIF), pyrazinamide (PZA), and either ethambutol (EMB) or streptomycin (SM).

- Prevention:

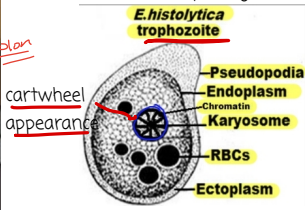
1. The best way to prevent TB is to diagnose and isolate infectious cases rapidly.
2. BCG (an attenuated vaccine derived from M. bovis) vaccination and treatment of persons with LTBI who are at high risk of developing active disease. If it has a low efficiency rate, 0-80%.

L3 ① Entamoeba histolytica:(Rhizopoda)

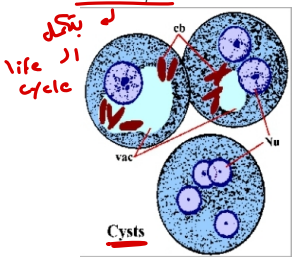
↳ large intesine / tropical and subtropical climate

↳ cecum, sigmoidorectal regions, colonic flexures and transverse ascending and descending colon. → fecal stasis and low Peristaltic movement

Active, Motile, Feeding and replicating form(which causes pathogenesis)



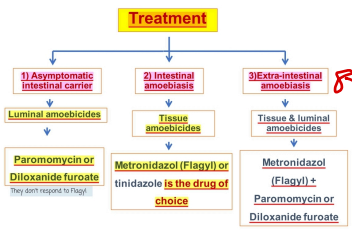
(a) Immature cyst (Uninucleate cyst and Binucleate cyst):
b) Mature cyst (Quadrinucleate cyst



Amoebiasis or amoebic dysentery ▪ D.H: Man ▪ R.H: Man, Dogs, pigs, rats and monkeys.

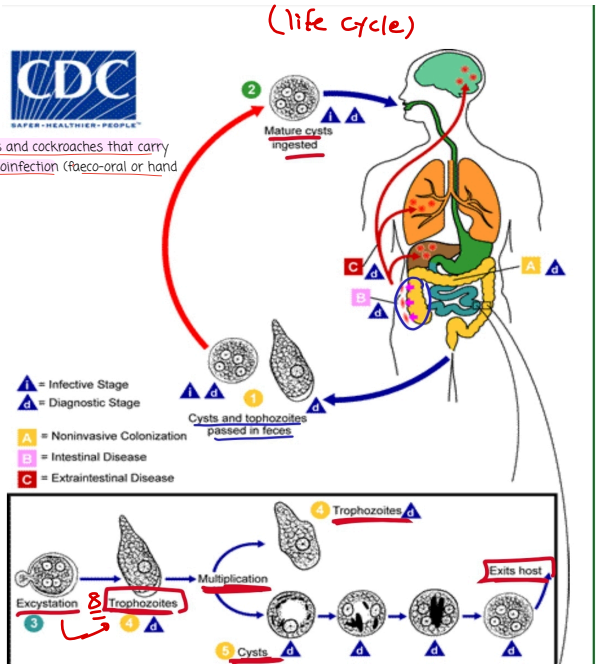
Entamoeba histolytica – Entering the Historical Dig

1. Red stool - Bloody Diarrhea
2. Do not enter historical site – Entamoeba Histolytica
3. Liver shape –
4. 2 main life cycle stages
   a. Bubbles in a puddle – Cyst form ingested from contaminated water ①
      i. Men drinking water holding hands ② gay men get disease from anal oral transmission (homosexual)
   b. Liver shape dig site and hole in right side of liver – Right lobe is most common involved site of amoebic liver abscess
      i. Map is a ct scan of liver and a man holding his right upper quadrant – RUQ pain w/ enlarged and tender lover
      ii. Anchovy paste truck - Abscess described as having "anchovy" paste consistency
5. Sewer pipe w/ out pouches like haustra to represent the colon - Causes intestinal amebiasis
6. Rust spots - Can cause ulcerations along the colon
7. Erlenmeyer flasks - Flask shaped ulcers
8. Red stools – bloody stool
9. Puddle w/ floating red cups - Stool O&P looking for cysts or trophozoites, stool will contain **trophozoites that contain endocytosed RBC's** (خاصية مميزة لها)
10. Flask in colon - An elisa antigen test or serology
11. Metra – Metronidazole
12. Drugs that work in lung of intestine
    a. Pair of mice near metro - Paramycin
    b. Sign labeled queen iodos tomb – Iodoquinol



العفروض حافظينهم حارمانه

(life cycle)



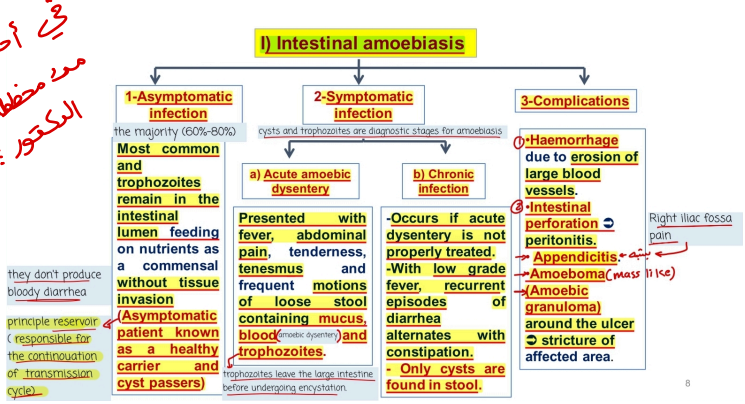
② Handling food by infected food handlers, ③ flies and cockroaches that carry the cysts from feces to exposed food, and ④ autoinfection (faeco-oral or hand to mouth infection).

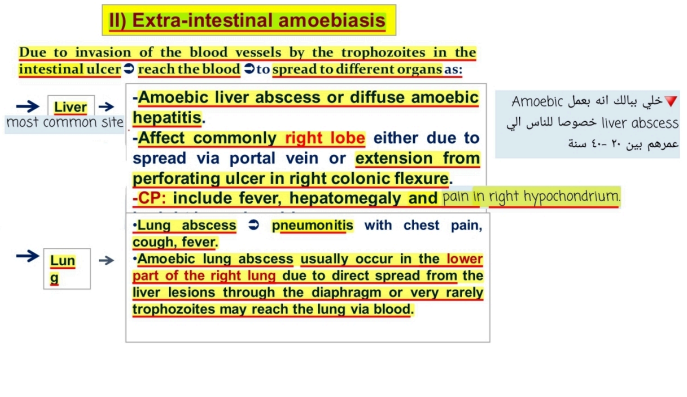
▼ The diagnostic stage is both the cyst & the trophozoite.
▼ The infective stage is the mature Quadrinucleate Cyst.

الدكتور حكى هم

- prevention:

- prevented by eradicating fecal contamination of food and water
- Amoebic cysts are not killed with low doses of chlorine or iodine
- Bringing water to a boil ensures the absence of amoeba and mechanical filtration.





خلي ببالك أنه ←

Acanthamoeba and Naegleria fowleri causes primary meningoencephalitis. But Entamoeba histolytica causes secondary amoebic encephalitis

② Giardia duodenalis (Giardia Lamblia and Giardia intestinalis)
↳ (Flagellates)

Giardiasis (Beaver fever)

Giardia Lambia – Giardia Jungle Ride

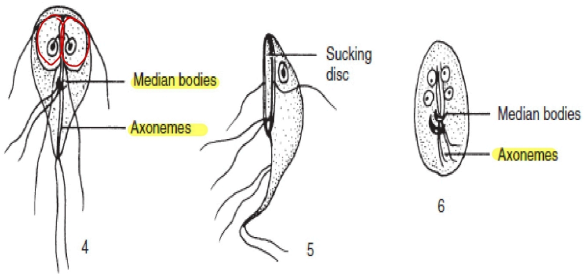
1. Poop in water and bubbles - Amongst travelers or campers drinking unfiltered or unpurified water that contains cysts of giardia from feces (fecal-oral route)
2. Backpacking backpacks – remind of backpackers and campers
3. Campers holding noses - Bloating, flatulence, and foul smelling diarrhea (Symptoms)
4. Yellow stool - Steatorrhea (fatty diarrhea) due to excessive mucus production that impairs absorption of intestine (A,D,E,K will be deficient) Malabsorption and weight loss
5. Shields on the boat - Trophozoite form that is flagellated
6. Shields in the water and OP guy pointing at the water - Attach but do not invade intestinal wall, so only cause diarrhea. If found in stool they are diagnostic
7. Elisa stool antigen to detect (Diagnosis)
8. Metra – Treat with metronidazole or tinidazole

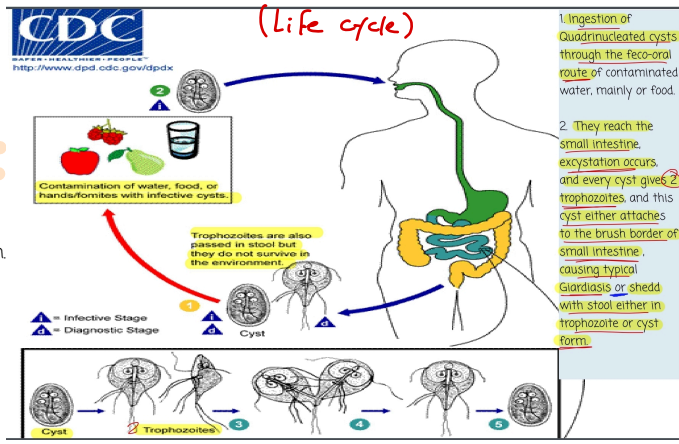
may cause chronic diarrhea, which can cause mental retardation in the pediatrics population.

☆ Another method for diagnosis is the "string test or the Entero-test," which is a gelatin capsule that's tied to a string and it is ingested by the patient, after several hours , we pull the string to pull out the capsule that carries the duodenal content .

▪ Trophozoites(زي شخص البس نظارة): Binucleated , it has parabasal bodies ventral disc, which help them in attachment on the brush border of epithelial cell of small intestine) and 4 pairs of flagella.

▪ Cyst (quadrinucleated cyst) : Oval in shape (elliptical) has the axonemes (source of the flagella)



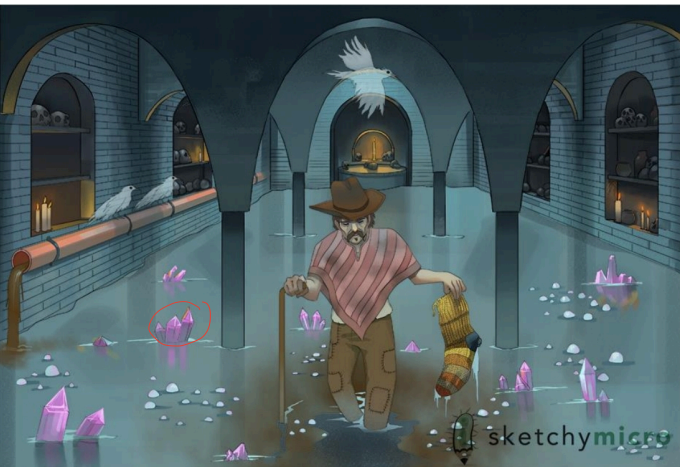


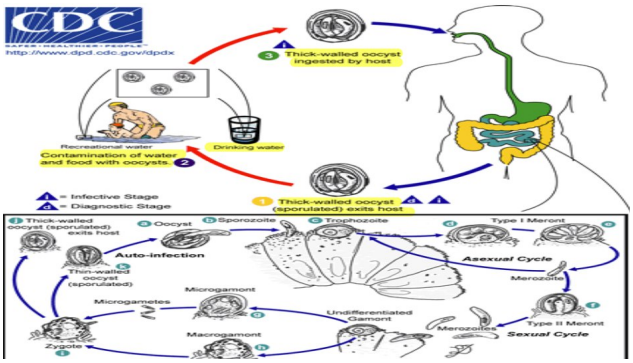
▼ The diagnostic stage is both trophozoite and cyst.

▼ The infective stage is only cysts.

③ Cryptosporidium spp (sporozoa)

C. parvum (mammals, including humans) and C. hominis (primarily humans).





Cryptosporidium – Tales from the Crypt

1. Brown water - Aids patients Cryptosporidium causes profound diarrhea
2. Immunocompromised cane – HIV Population
3. Red poncho and cowboy hat - Unicellular partially **acid fast** creating oocytes released fecally and absorbed orally
4. Bubbles – infectious cysts that get passed through watery stool
5. Gems scattered in water (amethyst) – acid fast and under staining looks like amethyst in water
6. Small Broken pipe in background - Cysts contain 4 motile sporozoites that will attach to small intestinal wall and cause damage.
7. Knitted sock – nitazoxanide treatment in immunocompetent
8. Water dripping out of sock – oocysts can be removed via filtration
9. Spirit crow – spiromycin txt (not FDA approved in US)

- Diagnosis: oocyst in stool using modified acid fast stain

- Treatment:Nitazoxanide is used for immunocompromised individuals e.g HIV patients

oocysts (due to sexual multiplication in their life cycle).
Thick wall oocysts >> الي بتطلع برا الجسم
Thin wall oocysts >> الي بتضل جوا الجسم
Thin wall oocysts continue the life cycle of cryptosporidiosis without needing to leave the host.

1. Ingestion contaminated water or food.

2. It reaches the small intestine in crypts on the epithelial cell. They alternate in their life cycle between sexual and asexual reproduction.

- Asexual reproduction called schizogony, and the daughter cells we call them merozoites(meront) , they go asexual reproduction and give us type 1 meront and they convert into thin wall oocyst which continues the life cycle without the need for leaving the host.

- Sexual reproduction of them will give type 2 meront which will convert to macro and microgametocytes, they will form the zygote and this zygote will form the thick-walled oocyst, that will go out with the stool and continue the transmission cycle outside the host.

▼ The diagnostic stage and infective stage are thick wall oocysts.

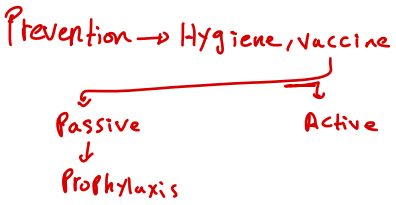
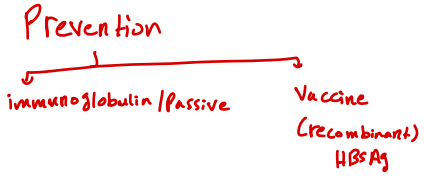
- All infections of Entamoeba histolytica ,Giardia Lamblia, and Cryposporidium are associated with poor sanitation and poor personal hygiene. all of them are transmitted by feco-oral route.

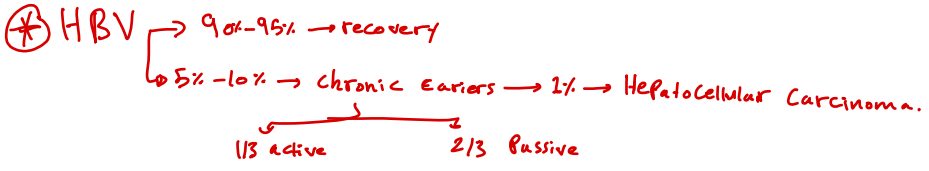
L5+L6

- Hepatitis: inflammation of liver; presence of inflammatory cells in organ tissue.
- The causes of hepatitis are varied and include viruses, bacteria, and protozoa, as well as drugs and toxins (eg, isoniazid, carbon , tetrachloride, and ethanol).
- Acute hepatitis: symptoms last less than 6 months.

❖ Viral hepatitis: All of them are not cytopathic

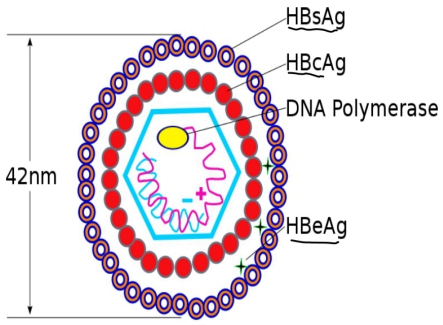
Virus	Type	Transmission	Epidemiology	pathogenesis
HAV (Infectious hepatitis) (Enterovirus)	Picornavirus +ssRNA	(fecal/oral route). associated with poor sanitation and overcrowding in countries	40-50 % of all acute hepatitis. mostly in children and young adult.	ingestion > Multiplies in oropharynx and intestinal epithelial cells > blood > Liver > Periportal necrosis + mononuclear infiltrates
HEV	Hepevirus +ssRNA	(fecal/oral route)	Waterborne epidemics of hepatitis. High mortality rate in pregnant women	----------
HBV (serum hepatitis)	Hepadnavirus Ds DNA	1. Parenteral via blood or plasma, needle stick injury. 2. Vertically: mother to baby 3. Body fluids 4. Sexually especially in homosexuals	-it's distributed worldwide. -400 million ppl with chronic infection of HBV (carriers of HBV) -1 Million deaths annually	Blood borne > liver cells > hepatocytes injury and necrosis (piecemeal necrosis: inflammation from the portal tract to periportal zone). -Largely cell mediated (Cytotoxic T cells).
HDV (Deltaviruses) It needs HBV to replicate (provide the envelop)	Defective –ssRNA virus	As HBV	----------	✓ Co- infection with HBV ✓ Super infection of HBV chronically infected patients (High risk of liver failure) , suffering from relapse of jaundice and have a high likely hood of developing of cirrhosis.
HCV (post-transfusion hepatitis)	Flavivirus, +ssRNA genome has 3 stuctural and 5 non-structural gene	blood and sexual contact (sexually is much less than HBV).	170 million cases globally	---------- --

	Clinical manifstaions	Diagnosis	Treatment
HAV	• Incubation period: 2-6 WEEKS • Most HAV infections are asymptomatic. • pre-icteric phase : fever, anorexia, nausea and vomiting. • icteric phase: jaundice , abdominal pain, hepatomegaly, splenomegaly, Dark urine and clay-colored stools and elevated transaminase levels.	1.Clinically - Liver enzyme: High AST and ALT, mild elevation of bilirubin. -leukopenia, lymphocytosis and elevated Erythrocytes Sedimentation Rate (ESR). 2.Serology: ➢IgM: Acute infection remains high for 3-6 months. ➢IgG: Past infection or vaccine.	-Usually, full recovery in 90% of patients in 3-6m. -Acute: Supportive: Do not give Paracetamol and Alcohol. -Fulminant hepatitis: Supportive, but may need liver transplantation. 
HEV	• Incubation period is 2-8 weeks with deeper jaundice when compared to HAV.	Anti-HEV AB and usually, bilirubin is higher than HAV.	treatment is Supportive
HBV	• Incubation period: 1-4 months (infectious dose). ✓Asymptomatic: 90% of children and 50% of adults (increased liver enzymes). ✓Symptomatic: • Preicteric phase: flu like symptoms nausea, anorexia, malaise	• Diagnosis of HBV: 1.Clinical picture. 2. Liver, kidney function tests, other tests to roll out other causes e.g: CMV, EBV infection. 3. Serology: • Anti core antibodies • DNA detection	1.Peg Interferon alpha for chronic hepatitis. 2. Lamivudine, Tenofovir, entecavir. 



	• Icteric phase: Jaundice, pale stool, dark- colored urine, increased liver enzymes and bilirubin.		
HDV	----------	serology: Anti-Delta IgM (in 3 weeks) or IgG (appear after and remain for years)	as HBV
HCV	• 6 - 8-week incubation period / most infections are sub-clinical. • Clinical infections are generally less severe than HBV, damage due to cell mediated immune response. • HVC has a higher incidence of chronic liver disease than HBV (70-80% of patients remain viremic for more than 1 year)	1. Anti HCV IgM: antigens tend to not be detectable in blood so serologic tests aim for Antibody. 2. RNA detection: quantitative assays might be used for diagnosis / estimating prognosis / monitoring therapy.	Antivirals: interferon alpha and ribavirin (aka taribavirin) are the drugs of choice of Hep C infection. Prevention → ① no vaccine ② Blood Screening

• The main components of the HBV include the following:
core - hepatitis B core antigen (HBcAg) and the pre- core hepatitis Be antigen (HBeAg), and the envelope of the virus contains the hepatitis B surface antigen (HBsAg).



For (HBV)

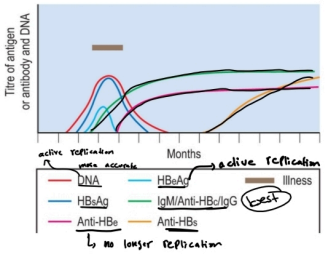


TABLE 41-4 Serologic Test Results in Four Stages of HBV Infection

Test	Acute Disease	Window Phase	Complete Recovery	Chronic Carrier State
HBsAg	Positive	Negative	Negative	Positive
HBsAb	Negative	Negative	Positive	Negative[1]
HBcAb	Positive[2]	Positive	Positive	Positive

	HBsAg HBeAg* HBV-DNA	HBcAb IgM	HBcAb IgG	HBeAb	HBsAb
Acute infection	+	+	–	–	–
Window period	–	+/–	+	+	–
Prior infection	–	–	+	+	+
Immunization	–	–	–	–	+
Chronic infection	+	–	+	+/–	–

Anti-HBcAg IgM —>acute hepatitis
Anti-HBcAg IgG —>past or chronic infection
HBsAg is general marker of infection —>if longer than 6 months —>chronic infection
HBsAb is used to document recovery or immunity to HBV infection
HBeAg indicate active replication and infectiveness of the virus
HBeAb indicates that there's no longer replication but still the pts will be +very for HBsAg which is made by the integrated HBV

viral DNA is more accurate than HBeAg especially in cases of escaped mutants (used in monitoring response to therapy)