Infectious diseases

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Vaccination

- Hepatitis B vaccine:
    - Protects against hepatitis B virus only (no cross protection for HAV)
    - Given as three doses at 0-1-6 months
    - Test the titer after 1-2 months of the last done. If it is > 10, the patient is immune. If it is not (5-10% of people), repeat the series again. If after the repeating the series, the titer is not raised, the patients are labeled as non-responders.
    - The vaccine gives protection for at least 20 years.
    - If a person's titers were measured years after vaccination, and they were low, a booster dose is given. If the titers do not rise after the booster dose, the original series is repeated. If titers remain low after the series, the patient is labeled as a non-responder.
- BCG vaccine:
    - Protects against tuberculosis (TB infection)
    - Given for children to prevent childhood tuberculosis, meningitis, and miliary disease.
    - Given once in a lifetime.
- Seasonal influenza vaccine:
    - Two types licensed by the CDC:
        - TIV: trivalent inactivated vaccine (IM)
        - LAIV: live attenuated influenza vaccine (nasal spray)
    - Contains three strains of influenza viruses:
        - Influenza A: H1N1 and H3N2
        - Influenza B
    - Given in November, but can be given up to April. Given as a single dose
    - 40-50% efficacy because of:
        - Multiple influenza virus strains
        - Patient may have common cold not influenza
        - Wrong injection into buttocks. The vaccine goes to fat no to blood stream
    - Contraindications:
        - Patients with febrile illness
        - Patients known to have egg allergy
        - Guillian-Barre syndrome
    - TIV should not be administered to infants less than 6 months old
    - People with egg allergy can receive the vaccine under medical observation with fractionation of the dose
    - The vaccine is recommended during pregnancy as it improves the health of the fetus and decreases birth complications.
- TDaP vaccine:
    - Tetanus, diphtheria, acellular pertussis

    - Given at age 11-12 years
    - Must be repeated every 10 years (Td vaccine, because immunity is lost)
    - Tetanus toxoid only protects against tetanus
- MMR vaccine:
    - Measles, mumps, and rubella
    - 10-20% will lose immunity after one dose
    - Complications in elderly are more common than in children
    - Examine the titer; if low, repeat the dose.
    - It is a live-attenuated virus, so it is not given for pregnant women during the first trimester. It has been linked to development of congenital anomalies.
    - If it was given to a pregnant woman accidentally, there is no need to induce abortion. The risk of having an abnormal child is extremely low. Many cases were reported to have normal children despite being given the vaccine
- VZV vaccine:
    - For chicken pox
    - Given for adults who did not develop the disease during childhood
    - Given as 2 doses
- Pneumococcal vaccine:
    - For patients with splenectomy and chronic diseases
- Hib vaccine:
    - Haemophilus influenza type B vaccine
    - Used to protect against meningitis caused by this bacterium. It used to be the most common cause of meningitis in children before the introduction of this vaccine
- Meningeococcal vaccine: given for people living in crowded areas (people going for Hajj)
- Hepatitis A vaccine:
    - People traveling to endemic areas
    - Patients with chronic liver disease, because these patients might develop fulminant hepatitis
- There is no vaccine for hepatitis C or HIV
- Vaccines given to health-care workers:
    - Influenza vaccine
    - Pneumococcal vaccine
    - Meningeococcal vaccine
    - Tetanus and diphtheria vaccine
- Hepatitis B vaccine can be given to the general population; however, there are no campaigns promoting hepatitis B vaccination (the general population is not at risk)
- All IM vaccines are given in the deltoid muscle not buttocks. Vaccines given in the buttocks stay in fat and don’t reach the circulation

Brucellosis

- B. melitensis is the most prevalent type. It causes severe, acute disease with disability and complications
- B. suis causes prolonged, suppurative lesions with destruction.
- Treatment is the same for all causative species
- In animals, it presents as contagious abortion.
- Brucella lives intracellularly. This accounts for its chronicity, resistance to many antibiotics, and escape of immune surveillance.
- It has a low mortality rate (<5%); most of these cases were attributed to the development of endocarditis (the most serious complication of Brucellosis)
- It is not fatal, but it is a depleting disease. If the patient was left untreated, they would not die but they would live a miserable life.
- Few cases have been reported with spontaneous remission
- It is more common in males, because males work as slaughterhouse workers. Slaughterhouse workers are patients with the highest risk.
- Strain 19 vaccine is the vaccine available for animals.
- Transmission:
    - Brucella cannot be transmitted between humans
    - It is unusual for brucella to be transmitted vertically (from mother to fetus); there is one case report only
    - It is unknown whether brucellosis can cause abortion in humans.
- Modes of transmission:
    - Ingestion of non-pasteurized dairy products; most common route.
    - Ingestion of infected tissue (rare)
    - Slaughterhouse workers exposed to animal meat through abraded skin (similar to anthrax)
    - Veterinarians due to accidental needle stick injury while vaccinating animals.
    - Aerosolization through respiratory mucosa and conjunctiva. Patients infected through this route develop pneumonia and conjunctivitis. Microbiology lab workers are infected through this route.
    - Exposure of intact skin to non-pasteurized dairy products is a risk factor
    - Spores can excreted in animal feces, urine, milk, and placental secretions
- Clinical manifestations:
    - Fever is the most common symptom and sign
    - Unlike other infections, fever can associated with relative bradychardia (called pulse-temperature deficit)
    - Generalized symptoms: anorexia, fatigue, abdominal pain, constipation, diarrhea, and vomiting

    - Dry cough with a negative chest X-ray is an important finding in patients with brucellosis. A dry cough with chest infiltration on X-ray indicates pneumonia caused by brucella
    - The first description of brucellosis was according to its fever's pattern. The fever is undulant (comes and goes with symptom-free intervals).
- Illness presentation:
    - Subclinical brucellosis:
        - Asymptomatic
        - Detected after serological screening of patients with high risk of exposure
        - Patients with anti-brucella antibody can be:
            - Patients with a disease
            - Patients who have been infected before, but are now disease-free
    - Acute brucellosis:
        - Can be mild, self-limited: B. abortus, or fulminant with severe complications: B. meditensis.
        - 90% of patients experience malaise, chills, sweats, fatigue, and weakness
        - It is one of the causes of prolonged fever or fever of an unknown origin.
    - Chronic brucellosis:
        - The diagnosis is made after the presence of symptoms for a period of more than 1 year
        - Low grade fever is a bad sign
        - No serological evidence of active disease (titer is negative), so it is difficult to diagnose
        - Those patients might have persistent disease caused by inadequate treatment of the initial episode.
        - Those patients ca have focal disease in the bone marrow, liver, or spleen
    - Localized disease:
        - Brucella can localize to any organ
        - It may come alone or with acute or chronic untreated brucellosis.
        - Common sites:
            - Osteoarticular:
                - Most common
                - Sacro-iliac joint in younger people and spondylitis in elderly
            - Epidydimoorchitis
            - Hepatosplenic abscess
            - Endocarditis: most common cause of death, but the least common complication
            - CNS: meningitis and encephalitis

    - Relapsing infection (<10%):
        - It is difficult to distinguish from reinfection in high risk groups
        - Associated with:
            - Inappropriate or insufficient antimicrobial therapy
            - Positive blood culture on initial presentation
            - Acute onset of disease
- Diagnosis of brucellosis:
    - CBC:
        - WBC can be high, normal, or low. Most patients have fever or malaise with a normal/low WBC count
        - Lymphocytosis
        - Pancytopenia: anemia, thrombocytopenia, and leukopenia
    - Liver function test: liver enzymes can be high
    - Blood or bone marrow culture:
        - Blood culture is positive in 10-30% of cases (up to 80% with B. melitensis)
        - With B. melitensis, bone marrow culture is of a higher yield than blood culture
    - Serology:
        - Standard tube agglutination (STA):
            - Looking for antibodies against antigents
            - It consists of sheep RBC's carrying brucella antigen. The patient's serum is added on top of it; if it agglutinates, it is a positive test.
            - Titer is measured on dilution bases of: 1/40, 1/80, 1/160 (above which is significant), 1/320, 1/640, 1/1280
        - ELISA:
            - To look for antibodies produced against brucella in the a patient's serum
            - Reaction happens in wells; a change in color indicates a positive reaction.
        - PCR: to look for brucella's genome. Still not in clinical practice
    - A negative blood culture with a positive titer on immune assay is diagnostic of brucellosis.
    - A positive titer + symptoms are diagnostic
    - Positive blood culture with no or minimal symptoms: the patient has the disease as an infection
    - Of the patient has no symptoms of brucellosis, but the antibody titer is 320: subclinical disease or previous exposure.

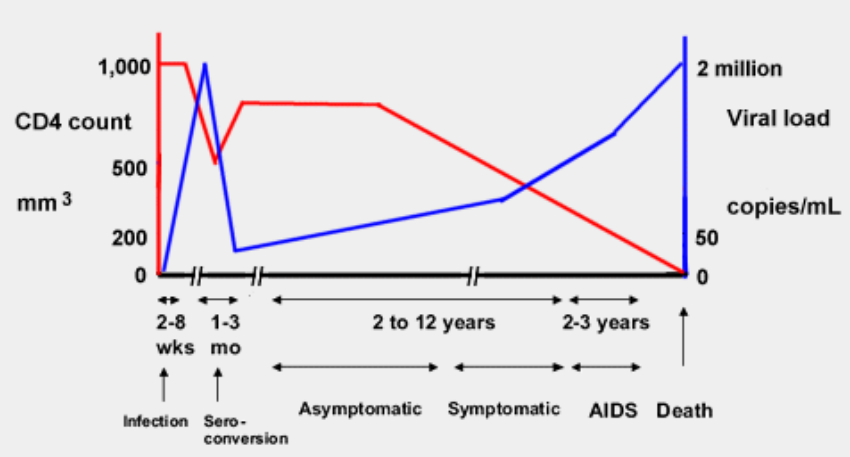
    - In endemic areas 10% of the population has a titer of 40 with no symptoms. This indicates previous exposure; however, if the titer is higher than 160, this necessitates treatment.
- Treatment:
    - Treatment must continued for a period of 6 weeks
    - Treatment must have a combination of medications.
    - Drugs used must have intracellular activity to reach the microorganism
    - Treatment options:
        - Doxycyclin (PO) + rifampin (PO) → 6 weeks
        - Doxycyclin for 6 weeks and streptomycin for 2-3 weeks
    - The most commonly used regimen is regimen number 1, because it is less toxic. Regimen 2 is more effective; however, it is more toxic.
    - Streptomycin is ototoxic
    - Doxycyclin inhibits bone growth and causes discoloration of bone (including teeth). Doxycyclin is not prescribed for patients less than 8 years of age. It is teratogenic, as well.
    - Treatment for children: trimethoprim-sulgamethoxazole (TMP-SMX) + rifampin for 6weeks
    - Treatment during pregnancy: rifampin is given until delivery, then a normal treatment course is initiated.
    - Ciprofloxacin based regimens have shown equal efficacy to doxycyclin based regimens.
- Prevention:
    - Test animals using ELISA. If they are infected, kill them. If they are not infected, vaccinate.
    - Avoidance of unpasteurized milk and milk products

Human immune-deficiency virus (HIV)

- In 1981, young healthy individuals started to present with diseases known to occur to patients that were immune compromised. Those diseases included pneymocystic pneumonia, invasive CMV, esophageal candidiasis, prolonged fever, Kaposi's sarcoma skin rash. All of these cases were homosexual males. Through investigations, they were found to lymphopenic with absent CD4 T-cells.
- Scientists called the disease AIDS: acquired immune deficiency syndrome. The virus was discovered and name the human immune-deficiency virus (HIV)
- In 2009, 33 million people were infected. 20 million people died due to complications of infection.
- AIDS is one of the serious outbreaks in the history of mankind
- The highest impact of disease is in Sub-Saharan Africa.
- In general, AIDS affects poor countries more.
- Since 2006, the number of deaths due to AIDS has been decreasing; however, the number of people living with HIV has been increasing. This means that there is a higher pool of infection.
- The incidence of infection has plateaued recently; however, it remains high.
- In a community, we can decrease the number of HIV infected individuals through:
    - Treatment of infected individuals (good)
    - Prevention of infection (better)
    - Comprehension of treatment and prevention (best)
- 5 million patients are infected annually:
    - 16,000 per day, mostly children
    - 50% of infected patients are females
    - 50% of infected patients are between 15-24 years old
    - 95% of infected patients live in developing countries
- Females of childbearing age ar at higher risk of infection, and their children will be infected.
- In Africa:
    - 80% of people below the age of 45 are infected
    - 80% of prostitutes are infected
    - Life expectancy is less than 40 years of age.
- High infection rates in Africa are due to:
    - Multiple sexual partners
    - Prostitution
    - STD's
    - Mother to child transmission
- In South Africa, a 15 year old boy has a 90% chance of dying because of AIDS
- HIV in Jordan:
    - 300 Jordanians have HIV

    - Outbreak is mostly in males
    - First reported case was in 1983
    - At first, all cases were reported cases of travel transmission; however, in the last years there has been reported cases with local transmission
    - High risk group is 25-35 years
    - Children are infected due to vertical transmission
    - There are patients infected after the age of 45
- Modes of transmission:
    - Most common mode of transmission worldwide is sexual intercourse (heterosexual); about 75%
    - HIV can enter as a free virus or within cells
    - Modes of spread:
        - Sexual (homosexual, heterosexual, oral)
        - Parenteral: blood or blood products, injection drug users, and occupational injury)
        - Vertical: (during pregnancy, birth, or breastfeeding)
    - Of those infected vertically, 80% are infected close to the time of delivery (due to mixing of maternal and fetal blood) and 20% are infected in utero.
    - All body fluids can have HIV
    - Exposure to infected fluids leads to a risk of infection which is dependent on:
        - Integrity of exposed site
        - Type and volume of body fluid
        - Viral load
    - The least common mode of HIV transmission is saliva due to the presence of factors that inactivate the virus. Moreover, the virus in the saliva is non-infectious.
    - Risk of infection after exposure:
        - Blood: 95%
        - Mother-child: 24%
        - Needle-stick: 0.3% (HBV; 30%, HCV; 3%)
        - Sexual intercourse: 0.1%
- The universal precaution is to treat any patient as if they are infected with HIV:
    - Hand washing
    - Wearing gloves if to be exposed to patient's fluids
    - Wearing a gown if to be exposed to patient's fluids
    - Hand washing after taking gloves off.
- HIV structure:
    - HIV is a ssRNA retrovirus from the lentivirus family
    - Binding via CD4 and chemokine receptor CCR-5
    - Target cells for HIV are CD-4 T-cells

    - Patients who have CCR-5 receptor deficiency are naturally protected from HIV infection.
- Types of HIV:
    - HIV-1: more common
    - HIV-2: rare, patients have lower viral loads, slower CD4 decline, lower rate of vertical transmission, and 12-fold lower progression to AIDS.
    - SIV (simian immune-deficiency virus): causes the same disease caused by HIV; however, it is found in monkeys. It is thought that HIV is a modification of SIV.
- HIV is composed of 2 molecules of ssRNA associated with:
    - Reverse transcriptase
    - Proteases: include integrase, P32, and P10
    - Core protein P24
    - Matrix protein P17
    - Cell wall (gp 160): made of gp 120 (envelope glycoprotein that binds CD4) and gp41 (transmembrane protein helps in fusion)
- HIV life cycle:
    - Attachment to CD4 receptors
    - Binding to co-receptor CCR-5 or CXCR-4
    - Fusion
    - Reverse transcription ssRNA → ssDNA; in the nucleus ssDNA is transformed in dsDNA
    - Integration (using viral integrase)
    - Either remains latent (not expressing genes) or active (transcribes viral genes)
- The presence of latent viral genome makes treatment impossible
- Transmission inside the body:
    - Selective infection of mucosal dendritic cells by HIV; the dendritic cells act as antigen presenting cells.
    - Fusion of dendritic cells and CD4 cells in lymph nodes.
    - Production of antibodies against HIV antigens (specific immune response). Antibodies produced are diagnostic; however, they are not protective.
- The viral load of the patient within the first year is within a viral steady state. It has a prognostic value
- Course of HIV infection:



- After infection (0 viral load), the viral load stats to increase to very high levels (10^6 copies/mL), then the decrease is very rapid.
- CD4 cells are normal at first, then they decrease sharply. Then, they increase again, but never reach normal levels.
- Clinical latency: no clinical symptoms, but the virus is active and replicating. It attacks CD4 cells, but the immune system is stronger than the virus. A patient in this phase has diseases indicated immune deficiency; however, they are not AIDS defining. Symptoms include weakness, weight loss, lymphadenopathy (generalized and persistent), fever, herpes zoster, and other things.
- CD4 cell count and viral load are used as prognostic factors:
    - Worst: high viral load, low CD4 count; progression to AIDS in 3 years
    - Best: low viral load, high CD4 count; progression to AIDS in 10 years.
- Acute HIV infection:
    - Mononucleosis like picture: fever, lymphadenopathy, skin rash on the upper trunk, nausea, and vomiting.
    - Occurs in 70% of patients infected with HIV
    - Corresponds to high viremia (10^8 copies/mL)
    - Corresponds to the first part of window period (2-6 weeks following the infection)
    - During this period, no antibodies are produced. Cannot be detected using standrad HIV detection techniques (ELISA and western blot)
    - The patient is highly infective. 1/3 of HIV patients have been infected during this period.
    - Diagnosed via PCR; high loads of viral genome
    - Clinical features of primary infection: fever with rash, pharyngitis with cervical lymph nodes, myalgia, arthalgia, headache, and mucosal ulceration
    - Differential diagnosis: acute EBV, CMV, streptococcal pharyngitis, toxoplasmosis, secondary syphilis
- HIV pathogenesis:
    - Destruction of CD4 cells
    - Evasion of immune response
    - Exhaustion of immunity
    - Lymph node pathology: during early stages of the diseases, lymph nodes are enlarged and inflamed. However, during later stages, they become small and calcified.
- Diagnosis:
    - ELISA (sensitive): looking for antibodies against HIV antigens. Used for screening in blood banks.
    - Western blot (specific): to confirm positive results from ELISA
    - PCR:
        - To detect viral load or acute infection.

        - Must be done after confirmation of infection to monitor response to treatment.
        - In treatment, we aim to reach undetectable viral
    - P24 antigen detection:
        - Not found in Jordan
        - P24 antigen is one of the viral capsid proteins. It can be used in place of PCR for detection of an acute infection.
    - 4th generation test for HIV:
        - Screen the serum for the presence of P24 antigen and antibody against HIV
        - Highly sensitive for acute HIV infection
        - Can detect HIV infection very early
- Why do we perform two tests for diagnosis (ELISA and Western blot)?
    - This is a serious diagnosis that needs to be confirmed
    - Some people have antibodies that are similar to those against HIV antigens (patients with connective tissue diseases like lupus and pregnant women)
- ELISA is not effective in the absence of the antibody; false negatives:
    - Window period (3 weeks- 3 months)
    - Immune compromised: hypogammaglobenemia, renal failure, and transplant patients.
- CDC classification of HIV (1993)

PGL: persistent generalized lymphadenopathy

CDC Classification System for HIV-Infected Adults and Adolescents

CD4 Cell Count Categories	Clinical Categories		
	A Asymptomatic, Acute HIV, or PGL	**B** Symptomatic Conditions, not A or C	**C** AIDS-Indicator Conditions
(1) ≥500 cells/μL	A1	B1	C1
(2) 200-499 cells/μL	A2	B2	C2
(3) <200 cells/μL	A3	B3	C3

- AIDS indicator conditions (STOP CATCHING IT D):
    - Salmonella (sepsis, diarrhea)
    - Thrombocytopenia
    - Oncologic disease (Kaposi's lymphoma)
    - Pneumocystic carini
    - CMV, cryptococcosis, cadidiasis, cryptosporidium (fungal infection of CNS)
    - Avium intracellulare mycobacterium (MAI)
    - Toxoplasmosis

    - Herpes simplex/ Zoster
    - Iatrogenic
    - Node enlargement, lymphadenopathy
    - Guillian-Barre syndrome, neuropathy
    - Isospora
    - Tuberculosis
    - Dementia
- B-category diseases:
    - Oral thrush
    - Bulbovaginitis
    - Prolonged fever or diarrhea (>1 month)
    - Oral hairy leucoplakia
    - VZV, ITP, PID
    - Peripheral neuropathy
    - Bacillary angiomatosis (doesn't occur in Jordan due to the absence of the causative agent)
- All AIDS indicator illnesses occur at CD4 <200 except for TB and Kaposi's sarcoma
- MAI occurs at CD < 50; it is a sign of impending death
- Anti retroviral agents:
    - ZDV (zidovudine) was the first agent. It was a chemotherapy agent; it belongs to the nucleoside reverse transcriptase inhibitor class.
    - Most effective treatment is HAART (highly active anti retroviral therapy):
        - Treat patients with a combination of 3 drugs: NRTI, PI, and NNRTI
    - Aim of treatment:
        - Suppress viral load to undetectable levels
        - Increase CD4 cell count to > 200
        - Improve quality and quantity of life without unacceptable drug-related side effects.
        - Reduce transmission
    - Strai-built: a new therapy found in 2012. It contains three drugs in one pill. Taken once daily
    - We treat patients who show interest in lifestyle modification (not drug users, not homosexuals,…) and those who have emotional support from the family to prevent recurrence of infection.
    - Treatment indications:
        - Symptomatic
        - CD <350 cell/mL
        - Pregnancy
        - Elderly
        - HIV nephropathy

        - Hepatitis B or C infection
    - Disadvantages of HAART:
        - Toxic
        - High cost
- In conclusion, HIV is a serious epidemic. Despite the use of combination therapy, resistance has been developing.

Sepsis

- Prevalence:
    - 300-500 million incidence per day in the USA.
    - Mortality rate ranges from 15-60%
    - Determining factors for mortality:
        - Age
        - Organism
        - Appropriateness of the empiric anti-infective therapy.
- Etiology:
    - Infectious:
        - Bacterial (most common)
        - Viral ex. Dengue fever
        - Fungal ex. Candidemia
    - Non-infectious: pancreatitis
- Definitions:
    - Infection: invasion of a normally sterile host tissue by microorganisms
    - Bacteremia: viable bacteria in the blood
    - SIRS (systemic inflammatory response syndrome): presence of 2 or more:
        - Respiratory rate >20
        - Heart rate >90
        - WBC count >12 $x10^3$ or < 4 x 10^3
        - Bandemia >10%
        - Temperature > 38 or <36
        - $PaCO_2$ < 32 mmHg or ventilated with pH >7.45
    - Sepsis: SIRS + documented infection
        - Not all sepsis syndromes are caused by bacteremia
        - In sepsis less than 40% have a positive culture because culture is not always accurate and SIRS may be caused by toxins of organisms in blood not the organism itself.
        - Bacteremia is not always present as the presence of organisms is cyclic in blood.
    - Severe sepsis: sepsis + organ dysfunction. One organ is enough to meet the definition
    - Septic shock: hypotension due to sepsis and unresponsiveness to initial fluid expansion. Hypotension is defined as systolic blood pressure <90 or a fall >40 from the baseline.
    - MODS: multiple organ dysfunction syndrome. Development of impaired organ function in critically ill patients with SIRS.
    - Spectrum: infection → SIRS → sepsis → severe sepsis → septic shock → MODS
- Pathogenesis:
- Pathogenesis:

- Activation of immune system + coagulation pathways with production of antibodies _ activated T cells. The immune system overshoots its response
- Activation is triggered by microbial triggers; lipopolysaccharides, peptidoglycans, lipoproteins, or super antigens.
- Cell wall factors:
  - In gram negative bacteria:
    - LPS (endotoxins): consists of O-side chain and lipid A portion
    - Inflammation and coagulation starts by interaction of LPS and mononuclear WBC
    - Lipid A is conserved in all gram negative bacteria i.e. sepsis due to Klebsiella is similar to sepsis due to E. coli
    - LPS can be found in blood of septic patients, so it can be used for research purposes
    - Presence of endotoxemia is correlated with a more severe form of sepsis
    - LPS infusion results in sepsis
  - Gram positive bacteria:
    - Peptidoglycans consist of lipoteichoic acid
    - In Jordan, gram positive is the most common cause of sepsis
  - Secreted factors:
    - Some strain of Staph aureus secrete toxic shock syndrome toxin 1 (SSTS1)
    - Some strains of group A beta hemolytic steptococcus secrete streptococcal pyogenic exotoxin A (SPEA). It causes necrotizing fascitis associated with hypotension. It is a superantigen that can bypass the macrophages to activate T-cells.
  - Cytokines involves in sepsis:
    - TNF-alpha
    - IL-1, 6, 8
    - Complement
    - Coagulation pathways
    - ROS
    - Gamma interpheron
  - Scientists have tried to stop the production of those cytokines by producing antibodies against them to stop the development of SIRS; however, this has failed.

- Most commonly damaged organs are the kidney, heart, and the lung
- Clinical case: a 66 year old male presented for thoracic aneurysm repair. 3 days later, he developed fever and confusion. No cough, no dysurea, no abdominal pain. Leaking surgical drain with serious fluid. Put on vancomycin for prophylaxis. On physical

examination he had a temperature of 39, heart rate 143, blood pressure 110/70. The patient is intubated, looking toxic and lethargic with warm extremities. Normal chest X-ray. WBC 1400 (wass 22,000 the day before). Differential PMN 24%, bands 37%. Hematocrit 41, creatinine 1.0, HCO_3 26. Blood + drain culture: E.coli. normal abdominal CT.

  - This patient has severe sepsis (SIRS + 2 organs failure).; warm extremities due to early vasodilation. Later on, it will change to vasoconstriction. He has a bad prognostic factor (WBC <4000). The patient has bacteremia. Treat with IV fluids and antibiotics.

- Hypothermia and septic neutropenia are indicators of a more severe infection
- A wide pulse pressure may be an early pointer to systemic sepsis
- Fever:
  - The first and most common manifestation
  - The higher the temperature, the more likely it is to be bacteremia
  - Fever is a good prognostic factor. The immune system and complement system work more effectively at higher temperatures
  - Hypothermia can occur with bacteremia; it is a bad prognostic factor.
- Hemodynamic changes:
  - Tachycardia: common
  - Bradychardia: unusual happens in cases of Brucella, typhoid, and lyme infection
  - Hypotension:
    - Most important in determining the outcome
    - Failure to correct hypotension will end in organ damage
    - Pre-shock (reversible): warm skin, oliguria, mental status changes
    - Shock (irreversible): cool skin, acute renal failure, hepatic injury
- Acid base disturbances:
  - Lactic acid is elevated due to tissue ischemia
  - Acidosis + cytokines: tachypnea which will lead to respiratory alkalosis
  - First sign of impending shock corresponds to the pre-shock stage
  - Metabolic acidosis develops just before or with hypotension; it signals the beginning of a fatal downward course.
- Respiratory changes:
  - Tachypnea develops due to acidocis, cytokines, and fever
  - ARDS can develop:
    - LPS activates PMN's, which become trapped in the small vessels of the lung causing an inflammation
    - Diagnosed by chest X-ray and severe hypoxemia
    - Differential diagnosis: pneumonia

- Diagnosis of sepsis:

    - Very difficult as you need to identify the actual infection source; however, most common sites include lungs, bloodstream, abdomen, wounds, and UTI
    - Clinical and labs:
        - The aforementioned SIRS criteria with chills, lethargy, and hemorrhagic skin lesions.
        - Any healthy person can develop two of the SIRS criteria in a physiological response; however, SIRS is a pathological state.
    - Work up:
        - Blood culture X 2
        - Urine culture
        - Sputum culture if abnormal chest X-ray
        - CBC with differential
        - DIC work up
        - ABG, chemistry
        - Other tests according to the condition.
- Treatment:
    - Antibiotics:
        - Empirical: before culture results; antibiotic that cover knowncausative organisms. At JUH, amkiacin works better than gentamycin.
        - For MRSA, use vancomycin
        - Covering the patient for every microorganism is impossible from the first time.
        - After 24-48 hours, the regimen should be adjusted according to the culture's results
        - Use the narrowest possible spectrum (to prevent resistance)
    - Volume expansion: with normal saline to correct hypotension. Every 1 hour delay (for up to 6 hours) increases mortality by 8%
    - Infection site management:
        - Surgical opinion for abscess or fascitis
        - Remove central line
        - Send to ICU to monitor vital signs.
    - Administration of vasopressors
    - There is no useful pharmacological treatment for SIRS; however, some drugs with potential benefits include NSAIDs, antibodies against LPS, antibodies against TNF alpha, IL-1 antagonists, and platelet activating factor antagonist
    - Steroids: their use is still debatable; however, they are given for pateitns who develop adrenal insufficiency (refractory hypotension is a sign of adrenal insufficiency). Given in low doses for 7 days; it can improve survival in patients who develop adrenal insufficiency

- Drotrecogin alpha (Activated protein C): not used anymore because of its limited value

Tuberculosis

- It is the most common cause of infectious disease mortality worldwide
- 1/3 of the world's population has latent TB
- The disease is increasing in the world; the most prevalent infectious diseases are TB, HIV, and malaria.
- Microbiology:
    - Caused by MTB (mycobacterium tuberculosis), which is a part of a complex of organisms including M. bovis (reservoir in cattle) and M. africanum (reservoir in humans)
    - MTB is a slow growing organism: 4-8 weeks for visible growth on solid media (Lowenstein-Jensen agar)
    - MTB is an acid fast bacillus, which stains with Ziehl-Neelsen stain
- Pathophysiology:
    - Humans are the only reservoir for MTB. It is transmitted via an airborne droplet nucleus.
    - M. bovis: arises by drinking non-sterilized milk from infected cows.
    - Once inhaled, droplet nuclei are deposited within the terminal airspaces of the lung (alveoli)
    - Initiate recruitment of macrophages and lymphocytes
    - Macrophages transform into epithelia and Langerhan's cells (multinucleated giant cells) that aggregate with lymphocytes to form tuberculosis granuloma. Numerous granulomas form a pimary lesion called Ghon's focus. Ghon's focus is a caseous nodule characteristically situated in the periphery of the lung.
    - Spread of the organism to hilar lymph nodes is followed by a similar pathological reaction. A combination of primary lesion and regional lymph nodes forms the primary complex of Ranke.
    - End result:
        - May be killed by the immune system (spontaneous remission)
        - May multiply and cause primary TB
        - May become dormant and remain asymptomatic (latent). This occurs if the reparative process encases the primary complex in a fibrous capsule limiting its spread. The end results of this process, if no complications occur, is calcification.
        - May proliferate after a latency period (reactivation disease)
    - Lymphatic or hematogenous spread may occur before immunity is established. This causes a blood-borne spread:
        - Few bacilli: seed in lungs, skeletal muscles, genitourinary tract, or the kidneys (infection often happens after months or years)
        - Massive spread: milliary TB and meningitis

- Timetable of TB:
    - 3-8 weeks: primary complex, positive skin test
    - 3-6 months: meningeal, miliary, pleural disease
    - Up to 3 years: GIT, bone, joint, lymph node disease
    - Around 8 years: rental tract disease
    - 3 years onward: post primary disease due to reactivation or reinfection
- Factors increasing the risk of TB:
    - Patient related:
        - Age: children > young < elderly
        - Travel to or from high prevalence areas
        - Smoking
        - Previous TB infection
    - Associated diseases:
        - Immune-suppression
        - Malignancy (especially lymphoma and leukemia)
        - Type 1 DM
        - Chronic renal failure
- Epidemiology:
    - Jordan: 7-10/100,000; TB infection is increasing in our region
    - USA: 4.4/100,000; 60% of which are foreigners
    - Mortality was 50% for untreated patients before antibiotics.
    - Mortality after antibiotics were introduced dropped to 4%, but drug resistant TB has a 50% mortality
- Clinical features:
    - Pulmonary TB:
        - Symptoms are not specific; fever, night sweats, anorexia, weight loss, chronic dry cough with hemoptysis, and chest pain.
        - Primary pulmonary TB:
            - Infection of a previously uninfected individual
            - Features:
                - Infection: 4-8 weeks; influenza-like illness, skin test conversion, primary complex
                - Disease: lymphadenopathy (often unilateral hilar, paratracheal, mediastinal), collapse or consolidation (especially right middle lobe), obstructive emphysema, pleural effusion, miliary, meningitis, and pericarditis
                - Hypersensitity: erythema nodosum, dactylitis, phlyctenular conjunctivitis
        - Miliary TB:
            - Following a massive blood-borne dissemination of MTB

        - On X-ray: millet seeds, which are fine 1-2 mm lesions distributed throughout lung fields.
        - Differential diagnosis: miliary TB, sarcoidosis, malignancy, pneumoconiosis, infection (histoplasmosis)
    - Post-primary pulmonary TB:
        - Either exogenous (new infection) or endogenous (reactivation of a dormant primary lesion) infection in a person who has been sensitized by earlier exposure.
        - It is most frequently pulmonary and characteristically occurs in the apex of upper lobes because oxygen tension favors survival of aerobic organisms and due to low lymphatic drainage
- Extra-pulmonary TB:
    - TB meningitis
        - It represents the most important CNS TB
        - Clinical features:
            - Headache that is either intermittent or persistent for 2-3 weeks
            - Subtle mental status changes, which may progress to coma over a period of days to weeks.
            - Fever may be low grade or absent
        - Mortality rate of 30% still persists despite the administration of an appropriate treatment. Many survivors are left with neurological sequelea
    - Skeletal TB:
        - Spine (Pott's disease)
            - Most common site for bony TB
            - Presents with chronic back pain and stiffness
            - Typically involves lower thoracic and lumbar spine. Lowe extremity paralysis occurs in 50% of patients
            - Infection starts as disctitis, then it spreads along the spinal ligaments to involve the adjacent anterior vertebral bodies causing angulation of the vertebrae with subsequent kyphosis
            - Differential diagnosis: malignancy; which tends to involve the vertebral body sparing the discs.
        - Joints:
            - Usually involves one joint (Hip or knee > ankle > elbow > wrist > shoulder)
        - Pancet's arthropathy: immunoligically mediated polyarthritis that is usually resolved within 2 months of starting the treatment

- Gastrointestinal TB:
    - Any site in the GI may become infected:
        - Non-healing ulcers of the mouth or anus
        - Difficulty swallowing
        - Abdominal pain mimicking PUD
        - Diarrhea
        - Hematochezia
    - Extraintestinal TB: in the liver or gallbladder. The least to infected is the pancreas because it has enzymes that resist infection
    - In jordan, peritoneal TB is the second most common form TB. It presents as exudative ascites. It is diagnosed by perineal lymph node biopsy
    - Ileocecal disease accounts for half of abdominal TB cases; 30% of patients present as a case of acute abdomen
    - Differential diagnosis: Crohn's disease
- TB lymphadenitis:
    - Cervical and mediastinal > axillary and inguinal (scrofula)
    - The nodes are usually painless and are initially mobile. But, they become matted together with time
    - Diagnosed by a lymph node biopsy
- Genitourinary TB: it is a rare entity because causes are diagnosed and early
- Cutaneous TB: very rare
- Pericardial disease:
    - In two forms:
        - Pericardial effusion
        - Constrictive pericarditis
    - Presentation: insidious with shortness of breath and abdominal swelling
    - Pericardial effusion: increased pericardial dullness, globular large heart on chest X-ray, blood stained effusion, and no edema
    - Constrictive pericarditis: raised JVP, early S3, atrial fibrillation, pulsus paradoxus, 25% present with calcification

- Differential diagnosis of lung cavitation:
    - Pulmonary TB
    - Lung abscess/pneumonia
    - Lung cancer
    - Pulmonary infarction
    - Wegner's granumalotosis
    - Progressive massive fibrosis

- Diagnosis:
  - Specimen required: sputum not saliva
  - Pulmonary:
    - Sputum:
      - 3 early morning samples or every 12 hours
      - Induced with nebulized hypertonic saline (for children)
      - Bronchoscopy with washing or BAL
      - Gastric washing: mainly for children
  - Extrapulmonary:
    - Fluid examination: low yield
    - Blood: in highly immune-compromised patients
    - Tissue biopsy: bone marrow, liver
  - Diagnostic tests:
    - Stain: Ziehl-Neelsen
    - Culture: solid medium (Lowenstein-Jensen agar)
    - PCR: to differentiate it from atypical mycobacteria
  - Obtain HIV in all patients with TB: treatment of such patients is complicated and mortality rate can reach up to 50% with treatment
  - Chest X-ray:
    - May show a patchy nodular infiltrate
    - Upper lobe involved in secondary type
    - Cavity indicates advanced disease. Smear positive patients with cavitation and laryngeal TB are the most infectious patients
  - Disseminated TB: means more than organ is involved. Not necessarily including the lung.
  - Miliary TB: lung involvement + other organs
  - PPD:
    - Purified protein derivative, a tuberculin skin test (mantoux test)
    - Indicated the development of delayed type cell mediated hypersensitivity (type IV) reaction for the PPD of TB
    - Needs at least 2-3 days; positive if:
      - High risk groups (5-9 mm):
        - take steroids
        - have HIV
        - have received an organ transplant
        - have a weakened immune system
        - have been in close contact with someone who has active TB
        - have changes on a chest X-ray that appear to be the result of a previous TB infection

        - Normal patients (>10 mm):
            - have had a negative PPD skin test in the past two years
            - have diabetes, kidney failure, or other conditions that increase their TB risk
            - are healthcare workers
            - are intravenous drug users
            - are immigrants who have come from a country that's had a high TB rate in the past five years
            - are under age 4
            - are infants, children, or adolescents who have been exposed to high-risk adults
            - live in certain group settings, such as prisons, nursing homes, and homeless shelters
    - vaccination: BCG, once only
    - infection control in hospital:
        - patient is put on respiratory isolation: negative pressure room, N95 mask
        - influenza and meningitis patients are put on droplet isolation: private room and the doctor puts on a surgical mask.
    - TB has no specific incubation period: most dangerous time after infection is the first two years
    - Health care workers must test for TB annually
    - Diagnostic role of PPD in TB: unknown, but in miliary TB 20% of the patients have a negative test. Al the cells are consumed at the infection site
    - Smear negative culture negative TB: PPD positive, diagnosed by response to therapy
    - Treatment:
        - Initaite empirical treatment of TB because culture takes a long time
        - Start on a 4-drug regimen:
            - INH (isoniazid) + rifampin + pyrazimmide + ethambutol or streptomycin for two months
            - Continue INH and rifampin for 4 months
        - if patients stop treatment, drug resistance develops.

Influenza

- **Pandemic**: a global outbreak of an infectious disease usually caused by a new virus. It causes serious illness.
- **Epidemic**: seasonal outbreaks; mostly in winter and autumn
- Influenza subtypes: A, B, and C (sporadic cases)
- **Structure:**
    - RNA virus 8 segments of RNA
    - Two types of spikes
        - Hemogluttinins (H): attachment to cell receptors
        - Neuroaminidase (N): cuts the bonds between the virus and the cell receptor completing the virus' life cycle.
- **Nomenclature of influenza virus:**
    - Subtype (A, B, C)
    - Place of discovery
    - Number of the virus
    - Year of isolation
    - H and N subtypes: we have 15 H subtypes, and 9 N-subtypes
- The problem of influenza virus is its large reservoir in nature. Subtype A is found in all species, B and C are restricted to human beings.
- Influenza is impossible to eradicate due to its large natural reservoir
- **Antigentic shift and drift:**
    - **Drift:**
        - Point mutation in the viral RNA; small change.
        - This leads to immunogenic escape, and it is responsible for the seasonal outbreaks.
        - Usually controlled by herd immunity.
        - Cross protection can be achieved with the use of other strains.
    - **Shift:**
        - Large change in the viral RNA; causes pandemics.
        - The change is caused by genetic reassortment.
        - The newly produced virus is usually a mixture of a human and an animal virus. Reassortment happens when the animal virus infects a human host in the presence of a human influenza virus. The two viral strains exchange RNA segment leading to the production of the new strain.
        - The virus is able to cross the species barrier.
        - Most communities have no immunity against the new virus causing a massive outbreak.
        - In the following seasons, the number of people affected decreases and it keeps decreasing until a new virus emerges.

        - This cycle usually takes 30 years; however, this number is decreasing due to globalization
        - Emergence of a new influenza virus is considered one of the top 10 threats to human life.
- **Famous influenza pandemics:**
    - Spanish pandemic: 1918, H1N1, 40 million deaths
    - Asian pandemic: H2N2, Asian pandemic
    - Hong Kong Pandemic 1968
    - Russian pandemic: H1N1, 1977
    - Swine flu: H1N1/09, 2009
- **Avian influenza (H5N1):**
    - Reservoir in birds
    - Birds have asymptomatic disease
    - Poultry had mild symptoms and were the main route of transmission to humans
    - Flight of birds was the main cause of spread
    - Avian influenza is still circulating; still can be transmitted, but the incidence is low.
- Sometimes, certain viral strains can reemerge
- Influenza used to be a fatal due severe inflammation and pneumonia. Mortality rate reached up to 65%. Mostly affected younger populations; elderly were relatively protected due to their lifelong exposure of different influenza strains
- **H1N1/09:**
    - Human-human transmission is possible
    - Flu-like symptoms including: runny nose, myalgia, headache, and can be accompanied with diarrhea
    - Low mortality rate 1-2%
    - It is no longer a pandemic; it has become an epidemic
    - Still susceptible to oseltamivir; very few resistant cases were reported.
- Common cold and influenza might cross symptoms. But, generally speaking, common cold is a much milder disease.
- Diagnosis is usually made on clinical basis; PCR and swabs are only used for research purposes
- Influenza patients are not treated with antibiotics. Treatment with antibiotics increases the risk for a superimposed bacterial infection. The mainstay of treatment, if needed, are neuroaminidase inhibitors. There are two types; oseltamivir and zanamivir. Oseltamivir (tamiflu) is an oral agent. Zanamivir is an inhaler, and is not used due to its complicated administration. These drugs prevent the disease by preventing influenza virus release from the cell. The recommendation is to give the agent within 24 hours of the start of the disease. If the patient has severe symptoms, it is okay to give the medication even if late. These drugs do not have a significant effect on the natural course of the disease; they

decrease the duration of symptoms by 1-2 days. However, in the last influenza outbreaks, these drugs had a huge role in stopping the progression of the disease.

- **Side effects of anti-influenza:**
    - GI toxicity (mainly)
    - Bronchospasm with zanamivir
    - Contraindicated in renal failure
    - Contraindicated in pregnancy
- Vaccines: the vaccine we use every year has the H1N1 strain of the 2009 pandemic
- **Influenza care:**
    - Sinal cavity care:
        - Do not sneeze in the direction of others
        - Cover the cavities when sneezing
        - Use tissues and dispose them right away
        - Wash hands
        - Take a sick leave to prevent spread of infection
    - Droplet isolation
        - Surgical mask
        - Private room
        - Duration of infectiousness is 7 days or until symptoms disappear, whichever comes first. In children, viral shedding continues for 2-3 weeks even with the absence of symptoms.
- **MERS/SARS**
    - Both are caused Coronavirus
    - Belongs to the coronaviridae family
    - Flu-like symptoms; indistinguishable from influenza
    - Mortality rate in MERS: 20-30%
    - transmitted from human to human in a poorly understood fashion
    - Reservoir: mainly in bats and camels. It is hypothesized that the cause of the MERS outbreak was camel to human transmission. Although many patients who were reported to have MERS had minimal contact with camels, it is believed that these patients contracted the virus from an asymptomatically affected individual
    - Diagnosis can be made through PCR (nasal swab)
    - Treatment: supportive care, antibiotics, hospitalization…

Infection control

- Infection control is defined as the prevention and control of infections in hospitals, communities, or healthcare centers. The goal of infection control is to prevent infection transmission from patient to patient, from patient to healthcare worker, and from healthcare worker to patient.
- Healthcare related infections epidemiology:
    - 1.7 million cases yearly in the US
    - 100,000 yearly deaths due to infections
    - It is one of the top 10 causes of death in the US
    - 1.4 million cases deaths globally at any given time.
    - Developed world: 5-10% of patients acquire infections in the hospital
    - Developing world: 20% of patients acquire infections in the hospital
- **Measures to control infection:**
    - **Hand hygiene:** (Soap and water, alcohol, and skin care)
        - Most effective measure is prevention of transmission of resistant microorganisms.
        - Doctors are the least compliant to hand hygiene protocols
        - Make sure to rub the following areas: between the fingers, palm of the hand, nail beds, thumb
        - Continue rubbing alcohol until it is dry
        - Put enough amount of hand rub so your hand is fully covered and you can continue rubbing until your hand is dry within 20 seconds.
        - Let your hands dry by rubbing only. Don't rub them on other surfaces.
        - The alcohol used in hospitals is not like regular rubbing alcohol. It has more moisturizing substances.
        - Excessive use of rubbing alcohol will cause stickiness. When this happens, wash with
        - Use of rubbing gel will cause dry skin. To prevent this complication, ue lotion. Dry skin is associated with an increased risk of infection transmission due to formation of micro-abrasions. Dry skin increases the risk of dermatitis.
        - What are the differences between regular soap and alcohol?
            - Alcohol has a better cidal effect; however, there are some organisms that are transmitted with spores, and these spores are very resistant to alcohol. Water and soap are important for prevention of transmission of these organisms
            - Clostridium difficile and ameba are resistant to alcohol
            - Soap and water work by mechanical removal of the organism

- Alcohol is gentler on the skin. Regular hand wash will remove the natural oils from the skin causing dryness. On the other hand, alcohol is a volatile substance, which preserves the natural oils of the skin.
- In case of visible dirt on the skin e.g. urine, stool, or blood, use soap instead of alcohol because the organism load is too high for alcohol to handle.
- After going to the toilet → soap and water
- After eating → soap and water
- After examining a patient → alcohol rub
- After removal of gloves→ soap and water
- Regular hand washing and alcohol are a bad combination → increased risk for skin dryness

- Long nails are not allowed in the hospital as many bacteria can gather under the nail.
- You are allowed to wear one small ring in your hand.
- After you remove the gloves, you need to wash your hands as viruses might be transmitted through the glove.
- Damaged skin is defined as any problem that involves skin (ranging froma rash to a wound). The presence of damaged skin is a risk factor for the transmission of many organisms. Normal skin has immunity against the transmission of viruses and other microorganisms.
- Exposure is defined as contact of 15 minutes of duration in the presence of intact skin. With damaged skin, risks of infection increase even with minimal contact

- **Important vaccinations:**
  - Influenza vaccine:
    - Taken yearly during autumn
    - For the sake of protection of both, healthcare workers and patients.
  - Hepatitis B:
    - The most important vaccine
    - Hepatitis B virus is widely spread in healthcare facilities
    - Three doses are given; check titers after third dose
    - Most commonly transmitted through needle prick injuries. Risk of transmission is 30% (3% with hepatitis C, 0.3% with HIV)
    - Vaccination does not spread the virus
    - The vaccine is taken only once
    - Management of a needle prick injury:
      - Wash hands with soap and water only
      - Report injury

        - If you do not know your titer levels, take the vaccine (protective 70%)
        - If you were not vaccinated, take the vaccine and an immunoglobulin (protective up to 90%)
    - Measles, mumps, rubella (especially females)
        - Rubella is associated with a high number of congenital anomalies
        - Vaccination should be given before pregnancy; vaccination during pregnancy, in case of an infection, is too late
    - Tetanus diphtheria:
        - Sharp injuries
        - Diphtheria is transmitted through droplets
        - Not common in Jordan
    - Varicella zoster:
        - If you have not been infected with chicken pox, you should be immunized against the disease.
    - HIV and hepatitis C don't have vaccines!
- **<u>Isolation measures:</u>**
    - Contact isolation
        - Transmission through contact (MRSA, VRE)
        - Use gloves when in contact
        - If you are exposed to bodily secretions, wear a gown and a mask
    - Respiratory isolation:
        - Transmitted through air (TB, measles, chickenpox)
        - Negative pressure room.
        - Wear a mask
        - Pneumonia, meningitis and influenza (droplets). Make sure to keep a distance from the patients.
    - Protective isolation:
        - Isolation to protect a patient from infection
        - Done when the patient has a high risk for acquiring a dangerous infection (infection might be life-threatening)
- Dealing with needles:
    - Do not break needles
    - Do not use needles more than once
    - Dispose needles in the specialized container
    - If you see a needle on the ground, remove it. But, use a tool to separate your skin from the needle.
    - In case of needle prick injury baseline HIV and hepatitis C titers should be measured. Check for the presence of any infections, and give appropriate

vaccination. If the person was injured with a needle exposed to HIV containing blood, post exposure prophylactic drugs are given (can prevent AIDS by 80%)…
    - Hepatitis C → no formal or solid recommendation after injury…
- **Environment of care:**
    - Within 24-48 hours of admission, the room will be full of the patient's flora.
    - This process is inevitable
    - This is why you should be careful and wash your hands every time you go into a patient's room.
- **Major hospital infections:**
    - Bloodstream infection:
        - Most commonly caused by central lines. Central line contamination can be through an external or an internal route. Infectioninthe first 10 days of line insertion is due to an internal contamination.
    - Ventilator associated pneumonia (major risk factor is intubation)
    - Surgical site infections
- In order to provide optimal healthcare, surveillance should be carried periodically. Surveillance is done to check for the prevalence of certain bacterial strains and their resistance.

Diarrhea

- Normal bowel movement is variable, but it can range from 3 times/day to 3 times/week. Diarrhea is defined as a change in the usual frequency of defecation accompanied by a change in the consistency.
- Classification:
    - Acute diarrhea: diarrhea lasting less than 2 weeks (14 days)
    - Chronic diarrhea: more than one month
    - Persistent diarrhea: diarrhea lasting more than 2 weeks but less than 4 weeks.
- Epidemiology:
    - Most common cause of death in children
    - Second most common cause of death worldwide
    - Second most common complaint in the ER
    - 2 million deaths yearly, mostly kids
    - High morbidity in the infants and elderly
    - Diarrhea in children is more common in:
        - China
        - Pakistan
        - Far East
    - It is more common in certain populations:
        - Pregnant
        - Daycare settings
        - Developing countries
        - Low hygienic environment
- Most cases of acute diarrhea are self-limited, and do not need medications (except in very rare cases)
- Frequent diarrheas during the year affect the normal growth process. Those who get diarrhea more than 45 times per year will never be normal
- Diarrhea costs 23 billion dollars yearly.
- Pathophysiology:
    - Inflammatory: inflammation decreases absorption of fluid from the GIT. We ingest 9 liters of water daily. Our GIT will absorb 90% of this amount; if we cannot do this properly, we will have diarrhea.
    - Secretory diarrhea: small intestinal cells secrete more fluids than normal (like cholera). Usually due to presence of toxins
- Transmission: all causative agents of diarrhea are transmitted feco-orally
- Risk factor for Gastroenteritis:
    - Improper disposal of feces
    - Lack of hand washing after defecation
    - Contact with feces before food preparation
    - Bad hygiene around food preparation area

    - Improper refrigeration of food
    - Food exposure to flies
    - Consumption of contaminated water
    - Personal hygiene
    - Stomach acidity: patients on PPI's are at a higher risk for infection (decreased acidity)
    - Immunodeficiency
- Etiology: there are many organisms (bacteria, viruses, parasites, or combined)
    - The most common cause of diarrhea in children is rotavirus
    - The most common cause of diarrhea in adults is norovirus
    - In up to 30% of cases, we cannot detect the etiology of the GE
    - The etiology depends on:
        - Season
        - Age
        - Immunity
        - Developed or developing country
        - Prevailing climate
    - Bacterial causes:
        - E.coli: toxogenic, invasive, O157H7 (discovered in 1982)
        - Shigella
        - Salmonella
        - Campylobacter jejuni (discovered in 1977)
        - Cholera
        - Listeria
        - Staph aureus
        - Clostridium: difficile, perfringens
        - Bacillus cereus
        - Neisseria gonorrhea
    - Parasitic causes:
        - Giardia
        - Ameba
        - Cryptosporidium (discovere in 1976)
        - Microsporidia
        - Ascaris
        - Cyclospora (discovered in 1986)
        - Isospora
    - Viral causes:
        - Rotavirus
        - Norovirus
        - Calicivirus (cup-shaped)

        - Adenovirus
        - Corona virus
        - HSV
        - Astrovirus (star-shaped)
        - CMV (in immunocompromised)
- Globalization has contributed to the spread of diarrhea. A famous story about this is the incident of wine diarrhea in the United States. Farming grapes in the United States cost wine producing companies a fortune, so they moved their grape farms to Guatemala. When farming moved to Guatemala, the incidence of diarrhea increased; it was discovered that cyclospora were the cause of the diarrhea, and it was tracked back to Guatemalan grape farms.
- Some bacteria produce symptoms by enterotoxins:
    - Staph aureus
        - Incubation period 6 hours
        - Vomiting (main symptom)
        - Fever
        - Some diarrhea
        - Abdominal pain (very severe and characteristic of this infection)
    - Bacillus cereus.
    - Clostridium perfringens (14 hours incubation)
- Some causative agents produce diarrhea via an inflammatory mechanism:
    - Enteropathogenic E. coli: mild inflammation causes mild diarrhea that does not contain blood
    - Rotavirus, norovirus, and HIV (mild inflammation)
    - Bloody diarrhea with high amounts of inflammation:
        - Salmonella
        - Shigella
        - Enteroinvasive E. coli
        - Ameba
        - Campylobacter jejuni
        - Yerseinia
- Differential diagnosis:
    - Ischemic vessels
    - Irritable bowel syndrome
    - Abscess
    - Carcinoid syndrome
    - Food allergies
    - Ingestion of non-digestible sugars
    - Acute alcohol ingestion
    - Medications

    - Small intestinal disease
- To locate the site of pathology causing diarrhea, we ask about the volume:
    - Small intestinal diarrhea is high in volume and water. Dehydration usually accompanies small intestinal disease
    - Colonic diarrhea: increased frequency of small amount of stools, bloody, and associated with urgency for stooling. E.g. ameba
- Lately, many of the causative agents of diarrhea have developed resistance. Resistance is more spread in certain countries. E.g. salmonella typhi in india
- E. coli can cause outbreaks. Some strains secrete toxins that cause bloody diarrhea. but without a fever. Some strains can be complicated with HUS (thrombocytopenia and renal failure). With E.coli infections, antibiotics are contraindicated as they make the disease worse. Treatment is usually supportive
- Rotavirus:
    - Named due its wheel-like structure
    - Vaccine is part of the national program
- Norovirus:
    - 2/3 of food-borne diarrhea
    - Vomiting
    - Diarrhea
    - Low grade fever
    - Nausea:
        - Most viral agents cause nausea due to their paralytic effect on the stomach
- Gastroenteritis is a disease that cannot be eradicated due to the high number of organisms that can cause it.
- Clinical case: 20 year old male patient presented with bloody diarrhea, old bloody diarrhea, tenesmus, and lower abdominal pain. What is your diagnosis?
    - Dysentery
    - Patients are usually: tachycardic, feverish, have abdominal pain, signs of sepsis, and dehydrated
    - Labs: hyponatremia (can present with normal Na levels), Creatinine increased, the patient has acidosis
    - Can be caused by Shigella…
    - Management: rehydration is the most important thing
- General management of acute diarrhea:
    - Acute diarrhea is mostly caused by viruses or bacteria. Parasitic causes are less common.
    - Stool culture is not important only 1 in 100 samples will yield a positive result.
    - When to order a stool culture?
        - When there is an outbreak
        - Cases of severe diarrhea only

        - Diarrhea more than one day
        - High fever
        - Severe dehydration
        - Sepsis
        - Blood in stool
        - Hemodynamically unstable patient (hypotensive)
        - During Outbreaks
    - Stool cultures are not usually useful; unless you ask the technician to look for a specific bacterium, the results will inform you about the presence of bacteria without specifying its type.
    - Rehydration can be life saving
        - 50-200 ml/kg/day
        - Normal saline…
    - Diet is important in management of diarrhea:
        - Food abstinence is not recommended. The patient needs calories to rebuild cells.
        - The patients should drink fruit juices, tea, soft easily digested food (bananas, apple sauce, rice, potatoes, crackers, soup)…
        - BRAT diet (bananas, rice, apple sauce, toast)
        - It is important that patients avoid dairy products. With infections, patients develop lactase deficiency. Ingesting dairy products will cause bloating and might worsen the symptoms of diarrhea.
        - Avoid carbonated drinks.
    - Contraindications of anti-motility agents:
        - Inflammatory diarrhea
        - Clostridium difficile infection (causes toxic megacolon)
        - E15 methicillin resistant Staph aureus.
        - Otherwise, it is okay to use them in small doses of anti-motility agents for traveler's diarrhea
        - In general it is better to avoid them
    - Antibiotics can be used with traveler's diarrhea as it is mostly bacterial
        - If the patient is febrile and dehydrated we give antibiotics
        - Fluoroquinolones are the most common antibiotics (ciprofloxacin)
    - If diarrhea lasts more than two weeks, it is mostly due to a parasitic cause (Giardia)

Parasitology

A **parasitic disease**, which is also known as parasitosis, is an infectious disease caused or transmitted by a parasite. Many parasites do not cause diseases. Parasitic diseases can affect practically all living organisms, including plants and animals, including mammals. In biology, parasitism is a non-mutual symbiotic relationship between species, where one species, the parasite, benefits at the expense of the other, the host.

Parasites falls into two major groups the endoparasites and the ectoparasites.
Endoparasites are classified into two main groups:
1. Protozoa, which are unicellular.
2. Metazoa (Metazoa is also called helminthes), which are multicellular.

In this sheet we are going to discuss the most common parasitic diseases that you may encounter in your infectious diseases' rotation in your fourth year as a medical student. This sheet was written from the references listed in the last page, since there are questions about parasitic diseases in the final exam.

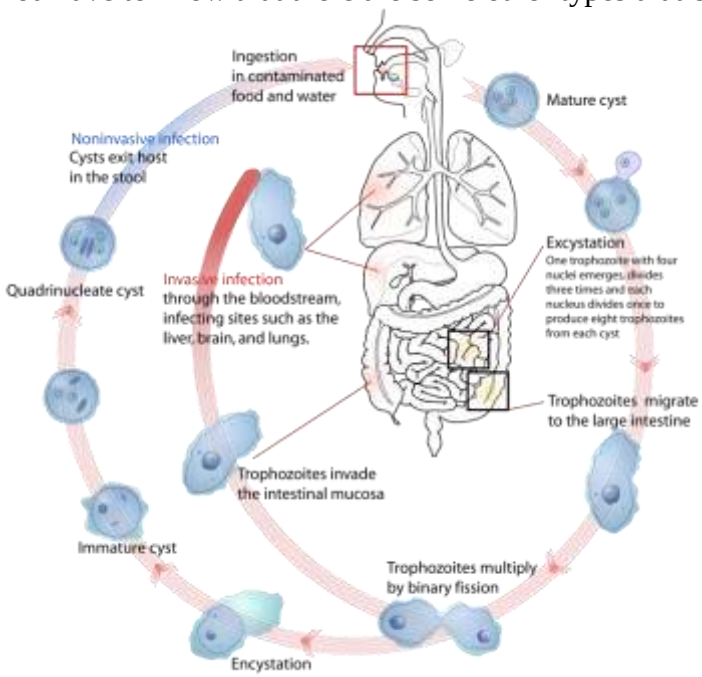
Amoeba

Amoeba is a protozoa endoparasite from Rhizopoda family. Lots of amoebae genera & species are free living; they live in the water and soil, and do **not** cause disease at all in the humans. There are 6 species which can inhabit the body of human beings. Out of these 6 species, there is only one species that is pathogenic and we're going to concentrate on, which is **Entamoebahistolytica**. The other five varieties can inhabit human's body but they do not cause significant diseases.

Since it is a protozoa, it spreads dirctly without an intermediate host. And Becausetrophozoites are very delicate, they die outside the body, so they can't transmit the disease, so they have to rely on something else or another form of the organism, which is known as the **cyst**, and that's the one that transmits the disease.

The six varieties of amoeba that are encountered in the body are:
1. entamoebahistolytica
The most important one, because as we said it's the pathological one and it's believed to be present in 10% of all the people in the world.
2. entamoeba coli: this is not pathogenic but it happens to be present in 30% of people.
We can differentiate it from the E.Histolatica by it's Cyst, the cyst of entamoebahistolytica is smaller and has only 4 nuclei, in coli it's larger and it has 8.
3. entamoebagingivalis: its present in the mouth in case of dental caries.

4. endolimax nana
5. iodamoebabutschlii
6. dientamoebafragilis
These are again amoebae which can be present in GI tract, in the large intestine, but they are not pathogenic
You have to know that there are some other types that can be there.



The incubation period for *E histolytica* infection is commonly 2-4 weeks but may range from a few days to years. The clinical spectrum of amebiasis ranges from asymptomatic infection to fulminant colitis and peritonitis to extraintestinalamebiasis, the most common form of which is amebic liver abscess.

Amebiasis is more severe in very young patients, in elderly patients, and in patients receiving corticosteroids. The clinical expression of amebiasis may be related to geography. For instance, amebic colitis is the predominant presentation in Egypt, whereas amebic liver abscesses predominate in South Africa.

Asymptomatic infections are common after ingestion of the parasite. *E dispar* does not cause invasive disease or antibody production. As many as 90% of *E histolytica* infections are also asymptomatic. The infection is self-limited but may be recurrent. It is not possible to distinguish between *E histolytica* and *E dispar* on clinical grounds; only antigen detection tests can make this distinction.

Amebic colitis

Amebic colitis is gradual in onset, with symptoms presenting over 1-2 weeks; this pattern distinguishes this condition from bacterial dysentery. Diarrhea is the most common symptom. Patients with amebic colitis typically present with cramping abdominal pain, watery or bloody diarrhea, and weight loss or anorexia. Fever is noted in 10-30% of patients. Intestinal amebiasis may mimic acute appendicitis.Rectal bleeding without diarrhea can occur, especially in children.

Fulminant amebic colitis is a rare complication of amebic dysentery (< 0.5% of cases). It presents with the rapid onset of severe bloody diarrhea, severe abdominal pain, and evidence of peritonitis and fever. Predisposing factors for fulminant colitis include poor nutrition, pregnancy, corticosteroid use, and very young age (< 2 years). Intestinal perforation is common. Patients may develop toxic megacolon, which is typically associated with the use of corticosteroids. Mortality from fulminant amebic colitis may exceed 40%.

Chronic amebic colitis is clinically similar to inflammatory bowel disease (IBD). Recurrent episodes of bloody diarrhea and vague abdominal discomfort develop in 90% of patients with chronic amebic colitis who have antibodies to *E histolytica*. Amebic colitis should be ruled out before treatment of suspected IBD because corticosteroid therapy worsens amebiasis.

Amebic liver abscess

Amebic liver abscess is the most common form of extraintestinalamebiasis.It occurs in as many as 5% of patients with symptomatic intestinal amebiasis and is 10 times as frequent in men as in women. Approximately 80% of patients with amebic liver abscess present within 2-4 weeks of infection. An estimated 95% of amebic liver abscesses related to travel develop within 5 months, though some may not manifest until years after travel to or residency in an endemic area.

The most typical presentation of amebic liver abscess is fever (in 85-90% of cases, in contrast to amebic colitis), right upper quadrant pain, and tenderness of less than 10 days' duration. Involvement of the diaphragmatic surface of the liver may lead to right-side pleuritic pain or referred shoulder pain. Acute abdominal symptoms and signs should prompt rapid investigation for intraperitoneal rupture.

Associated gastrointestinal (GI) symptoms occur in 10-35% of patients and include nausea, vomiting, abdominal distention, diarrhea, and constipation. Approximately 40% of patients who have amebic liver abscess do not have a history of prior bowel symptoms. Although 60-70% of patients with amebic liver abscess do not have concomitant colitis, a history of dysentery within the previous year may be obtained.In a recent study of routine colonoscopy in patients with amebic liver abscess, colonic involvement was noted in two thirds of cases.When colon was involved, right colonic lesion was universally present.

A small subset of patients with amebic liver abscess have a subacute presentation with vague abdominal discomfort, weight loss or anorexia, and anemia. Jaundice is unusual. Cough can occur. A history of alcohol abuse is common, but whether a causal relation exists is unclear.

Other manifestations of amebiasis

Ameboma, Pleuropulmonaryamebiasis, Cerebralamebiasis, Amebic peritonitis, Amebic pericarditis, Genitourinary amebiasis, Amebic appendicitis.

Physical Examination:

Patients with acute amebic colitis may present with lower quadrant abdominal tenderness. Fever is noted in only a minority of patients. Weight loss occurs in 40%. Dehydration is uncommon. Occult blood is nearly always present in stools (70-100%). Fulminant amebic colitis is commonly characterized by abdominal pain, distention, and rebound tenderness.

Amebic liver abscess may present with fever (85-90% of cases) and tender hepatomegaly (30-50%). Right lower intercostal tenderness may be elicited, particularly posteriorly (84-90%). Weight loss is noted in 33-50%. Breath sounds may be diminished at the right lung base, and rales may be heard. A small subset of patients has a subacute presentation with hepatomegaly, weight loss, and anemia. Jaundice is unusual (6-10%).

Treatment:

Most individuals with amebiasis may be treated on an outpatient basis. Several clinical scenarios may favor inpatient care, as follows:

- Severe colitis and hypovolemia requiring intravenous (IV) volume replacement
- Liver abscess that is of uncertain etiology or is not responding to empiric therapy
- Fulminant colitis requiring surgical evaluation
- Peritonitis and suspected amebic liver abscess rupture

Intestinal amebiasis may be mistakenly treated as if it were inflammatory bowel disease (IBD). Accordingly, in all patients with suspected IBD, lower gastrointestinal (GI) endoscopy should be performed before treatment with steroids is initiated.

Pharmacologic treatment:

Metronidazole is the mainstay of therapy for invasive amebiasis.Tinidazole has been approved by the US Food and Drug Administration (FDA) for intestinal or extraintestinalamebiasis. Amebic liver abscess of up to 10 cm can be cured with metronidazole without drainage.

Enterobiusvermicularis (pinworm), threadworm infection &oxyuriasis

They are very thin round worm, and they are a nematode Helminthes.

The females and males copulate and produce fertilized eggs. These eggs are not laid in the intestine. However, the pregnant female goes to the anal opening and lays the eggs on the skin outside the lumen.
Now if you have a look on the life cycle of it you could see that the laid eggs –on the perennial skin- are already "embryonated" which means that they are mature and infectious.
The problem here that these eggs well cause irritation to the perennial skin leading the patient to scratch. And this etching is called pleuritisani.

Now by that etching the patient could get the eggs below his nails, then he could either reinfect himself or infects other people. Usually this occurs with children so the people in danger of infection are other children in class or family members.

Signs and symptoms:

One third of individuals with pinworm infection are totally asymptomatic. The main symptoms are pruritus ani and perineal pruritus, i.e., itching in and around the anus and around the perineum, which occurs mainly during the night. Pinworms cannot damage the skin, and they do not normally migrate through tissues. However, in women they may move onto the vulva and into the vagina, which can cause vulvovaginitis.

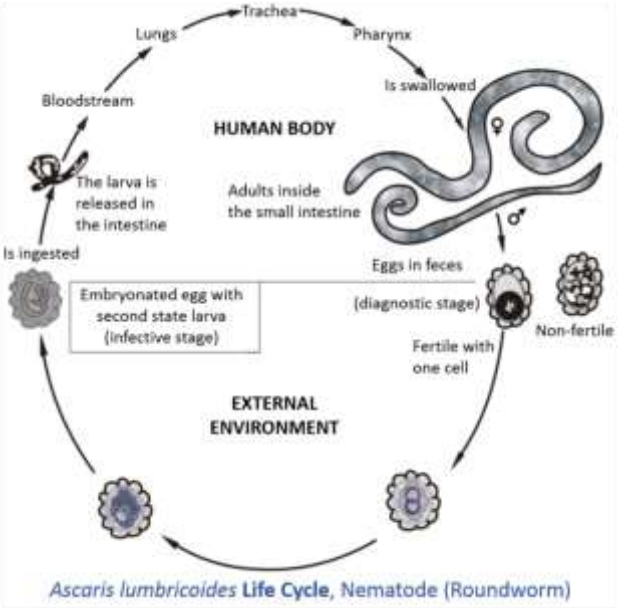
Diagnosis

Eggs are invisible to the naked eye, but they can be seen using a low-power microscope. Pinworms do not lay eggs in the feces, but sometimes eggs are deposited in the intestine. these eggs are look like coffee beans: one side is flat while the other is convex. And you can see the embryo in the middle.

Treatment :
Vermox \Mebendazole (we give two doses 10 days apart to eliminate the chance of reinfection).

Ascarislumbricoides

They are big worms, nematodes helminthes. These worms are very muscular, and they use these muscles to prevent flushing by peristalsis.
Males are 15-30cm long and females are 20-50cm long. The male's posterior end is curved ventrally and has a bluntly pointed tail.



Ascaris lumbricoides **Life Cycle**, Nematode (Roundworm)

Symptoms of Ascariasis :
Related to the number of worms: if the patient has only few worms in his gut the infection might pass without any symptoms or he might only have abdominal pain and discomfort.
As an infection it's not that serious, the worms will eventually die, and they will come out with feces. So the patient comes to your clinic panicked from these worms.
Sometimes it deteriorates the health of the patient if he is a child or immunosuppressed by the toxins released from it and causes anorexia , malnourishment ..etc

Diagnosis:
Fecal examination (you look for the eggs)
Treatment:
Piperazine , paralyzes the worm then it will be flushed out with feces

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Tapeworms (Cestodes)

1-Taenia Saginatum:
Intermediate host is the cattle, and the primary host is the human being.
2-Taenia Solium
Intermediate host is the pig and the primary host is human .

You can differentiate between T.saginatum and T.solium by their morphology and their life cycle.

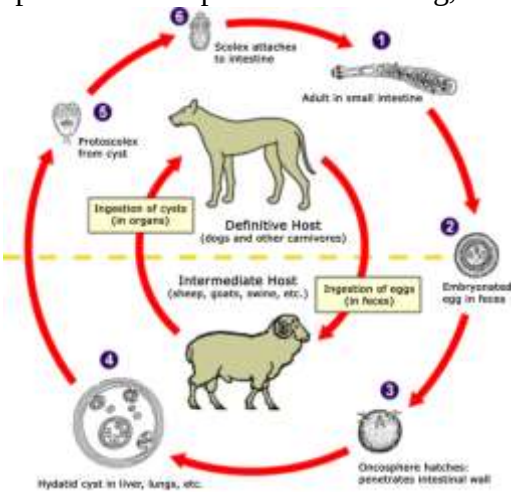
3-Diphyllobothrium Latum :
Fish tapeworm.
Largest tapeworm(up to 10 meters) that can infect humans.
Lives in small intestines and produces eggs: The eggs are Morphologically distinct: similar to those of trematodes, ovoid operculated on one end with a thickening on the other end. This is due to the adaptation that is needed to live inside aquatic interemediate hosts (Copepod in this case).
Symptoms:
present as 1 worm
live up to 20 years
Very minor symptoms
Deficiency of vitamin B12: these worms extend to the ileum and compete with the absorption of vitamin B12 (Megaloblastic anemia might occur) “this is a very important point, and it is a common exam question.”

Diagnosis:
Examination of feces(proglottids and the typical heads of the organism)

Hydatid disease

EchinococcusGranulosus
Small Tapeworm (1 cm), Cestode Helminthes. It is the smallest tapeworm that can affect human being.

The primary hosts are members of the canine family (foxes, wolves) and dogs, and the intermediate hosts proper are sheep, cattle, goats, pigs, etc. Humans can be accidental intermediate host, and in this case they are dead-end hosts.
The egg hatches in the small intestine of the intermediate host and releases an oncosphere that penetrates the intestinal wall and moves through the circulatory system into different organs,but the most common organs to be affected are the liver and the lungs. Once it has invaded these organs, the oncosphere develops into a cyst. The cyst then slowly enlarges, creating protoscolices and daughter cysts within the cyst. The definitive host then becomes infected after ingesting the cyst-containing organs
This cyst starts as a very small structure (about 1 cm in diameter) and it consists of a membrane and inside the membrane there is fluid which is yellowish in color and then it goes bigger and bigger. These cysts will be space occupying lesions living as tumors in the liver but we can have spread to other places like the lung, brain, skin, bone, subcutaneous tissue, and kidneys.



Clinical presentation
Manyhydatid cysts remain asymptomatic, even into advanced age. The parasite load, the site, and the size of the cysts determine the degree of symptoms. A history of living in or visiting an endemic area must be established. Also, exposure to the parasite through the ingestion of foods or water contaminated by the feces of a definitive host must be determined.
Secondary complications may occur as a result of infection of the cyst or leakage of the cyst: Minor leaks lead to increased pain and a mild allergic reaction characterized by flushing and urticaria.
Infection of the cyst can occur either as a primary infection or as a secondary infection following an episode of a leak into the biliary tree, a cystobiliary fistula.
Extremity pain with or without neurologic deficit is a sign of either bone or muscle involvement.

On physical examination, The most common sign is abdominal tenderness. Hepatomegaly may be present or a mass may be felt. Tender hepatomegaly is a sign of secondary infection of the cyst, especially when coupled with fever and chills. Ascites is rare, and splenomegaly can be the result of either splenic echinococcosis or portal hypertension. Jaundice could be a sign of biliary obstruction. Spider angiomas are a sign of cirrhosis of the liver, and Urticaria and erythema may be seen. Fever could be a sign of secondary infection or allergic reaction, and Hypotension is observed with anaphylaxis secondary to a cyst leak.

Treatment:
Medical management
Chemotherapeutic agents: Two benzimidazoles are used, albendazole and mebendazole.
Surgical management: surgery remains the primary treatment and the only hope for complete cure

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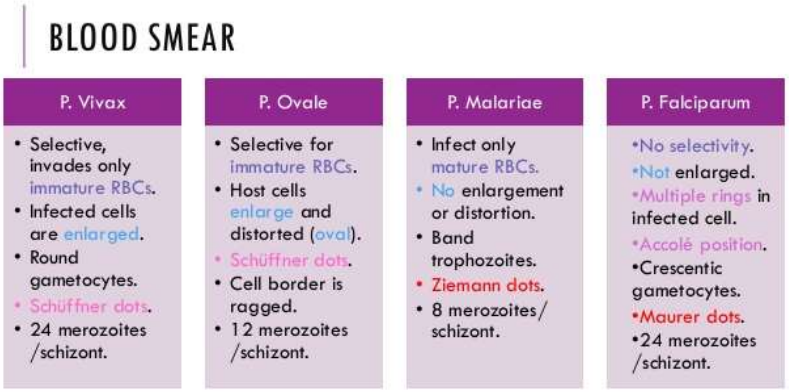
Malaria

It transmitted by a **female anopheles mosquito**.
Malaria is caused by a parasite known as *Plasmodium*, a coccidian which belongs to the group of *Apicomplexa*, so it is an intracellular parasite
The multiplication of Coccidia has two varieties:
Sexual and Asexual, depending on the host they are in,
 as in malaria; human is the intermediate host host where asexual multiplication takes place and is referred to as schizogony (the form of malaria is called schizont)
 while in mosquito which is the primary host, sexual multiplication takes place and is referred to as sporogony(results into sporozoites, which are transmitted to human and cause the infection).
The most important species are:
1- Plasmodium Vivax. (most common)
2- Plasmodium Falciparum.
3- Plasmodium Malaria.
4- Plasmodium Ovale.
IT IS VERY IMPORTANT TO Differentiate between these four species
this table doesn't include all of the important information



Treatment:
We already took the treatment in the pharmacology, but remember that in case of P.vivax or P.ovali we should give primaquine, chloroquine, or doxycycline because of hypnozoite state which may be present in the liver cells.

Filarial Worms

Wuchereriabancrofti
They reside in the lymphatics of the host, can survive for a few years (about 4 to 5), and produce microfilariae which can access the bloodstream.
The microfilariae mostly appear at night (10pm-2am), because the intermediate host is the mosquito, which usually flies at night. This is called the Diurnal rhythm.

Symptoms of filariasis
Non-inflammatory: More common in children
Inflammatory: There is an allergic inflammatory reaction to the adult worms which is associated with the lymphatics. The patient experiences malaise, fever, and lymphangitis
Obstructive disease: The lymphatics are scarred and obstructed, which means the lymph flows back. This causes the swelling of organs involved. One of the most common presentations is when the feet became very swollen and they look like elephant's leg, then the case is referred to as Elephantiasis.

Diagnosis
Can be achieved by observing clinical features, studying the degree of endemism, and more often, testing blood samples for the presence of microfilariae.
Treatment
Can be achieved by cleansing the skin, surgery, and the use of therapeutic drugs (DEC[diethylcarbamazine], ivermectin, albendazole).

Loa loa
They live in subcutaneous tissue, do not cause serious disease
They lay microfilariae which can access the blood, and appear after 2pm, since their intermediate host is the mango fly which flies in the afternoon

Symptoms of *Loa Loa*filariasis
- Crawling sensations under the skin, which are not very common.
- Allergic reaction: Red itchy swellings at the site of infection, called "Calabar Swellings"

Diagnosis
Can be achieved by testing a blood smear taken during the afternoon.
Treatment
Mentioned above.

Trypanosoma
Trypanosoma exists in two forms, one in the intermediate host (an insect) & another form in the primary host.
Trypomastigote: found in primary host (humans)
Epimastigote: found in intermediate host (fly or bug: tsetse fly or reduviid bug)
There are two types of trypanosome:
1) Trypanosomabrucei (causes sleeping sickness) present mainly in Africa
2) Trypanosomacruzi present in south America

Toxoplasma gondii

It’s an apicomplexa protozoa family member
Has two reproductive cycles; sexual in the primary host and asexual in the intermediate host, where it divides asexually by a special type of division known as enododyogeny
Human is an intermediate host, though considered as a dead end intermediate host
infection of toxoplasma is usually Asymptomatic
toxoplasmosis is a problem if the mother got infected with toxoplasmosa for the first time during pregnancy, then the infection can reach the fetus causing congenital toxoplasmosis.
In immunodeficient people when infected for the first time, they will be definitely ill & will exhibit severe symptoms; fever, lymph node enlargement, invasion of the CNS & neurological deficits

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Other endoparasites that you should read about:

Trichinellaspiralis
What is unique about this worm is that the same animal serves both as primary and intermediate host. This worm could exist in the intestine of the animal as an adult worm (nematode) and this stage is considered as a primary host but the intermediate stage which is the (larvea or a cyctedlarvea) actually happened to be in the tissues of that animal mainly the skeletal muscles.

LEISHMANIA
the sand fly is it’s intermediate host.
cutaneousleishmaniosis
When the human is sticked by a sand fly the site of injection will develop a lesion (usually a swelling) which becomes hard as it is made of inflammatory cells. so you will get a hard nodule which will ulcerate then become indurate chronic ulcer and after about one year it will heals leaving a nasty scar and you will get solid immunity .
The second type of leishmaniosis is the **diffuse cutaneous leshmaniosis**(mostly associated with L.aethiopica) , its named diffuse because there is a spreading of the lesions on the skin unlike the previous type . Actually the immune response is responsible for this dissemination
The third type is the **muco cutaneous leishmaniosis**, it usually present in America and caused by leishmaniabraziliensis
The last type is **visceral leishmaniosis**(or kalaazar "black disease" because of the hyperpegmintation of the skin that is one of its symptoms)

The species that lives in cold environments like skin 34-35 C (L.tropica ,L.aethiopica) cause cutaneous leishmaniosis. But other species like **L.denovani ,L.chagasi , L.infantum**can live in cold tempreture and are **more resistant to cidal actions of the serum**, so they can live inside the macrophages in **viscera** (liver , spleen , bone marrow) and cause visceral leishmaniosis.

Ectoparasites:

Lice

Head Lice

- The egg of a louse is called a nit. They can be found stuck to the hair, and shine under fluorescent light which helps detect them.
- They are confined to the head, and are attracted to moist or damp areas; such as behind the ears.
- Head lice are not a hygienic issue; they can easily be transmitted by close contact with an infected person. They do not transmit disease either.

Symptoms

- Itchiness.
- Scratching may lead to excoriation of the skin (abrasions; skin is worn off) which can be secondarily infected by bacteria

Pubic Lice

- Commonly called "Crabs" because of their slightly similar appearances, they affect pubic hair, sometimes eyelashes and brows, and can be considered an STD in some cases.
- They are not vectors for other diseases. Their only consequences are itchiness and excoriation of the skin.

***Cimexlectularius*(Bed Bugs)**

- Nest in cracks in walls, wood, furniture, or anything similar.
- They are not a hygienic issue.
- They feed on blood. Their bites are similar to mosquito bites, only larger and nastier, and might cause skin rashes and allergic symptoms.
- Do not transmit diseases, but are a nuisance because of their bad smell.
- Acquisition is by close contact.

References:

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Wikipedia

-This sheet was written in July, 2016

GOOD LUCK ALL :D