

Biology 6 – Test 5 Study Guide

Chapter 19 – HIV and AIDS

A. Discovery

- a. 1981: high rate of rare Kaposi's sarcoma and pneumonia in immunocompromised group in Los Angeles. Both rare.
- b. 1983: virus discovered that infects Helper T cells.
- c. Origin
 - i. Simian virus in monkeys and chimps.
 - ii. Estimation that virus made jump to humans in 1930s during butchering of food in Sub-Saharan Africa.
 - iii. Oldest case is man who died in 1959 in Kinshasa, Congo.

B. HIV

- a. Strains
 - i. HIV-1 – most virulent. Clade B is most common in US.
 - ii. HIV-2 – more rare and less virulent. More related to SIV
- b. Structure (Fig. 19.12)
 - i. Retrovirus w/ two strands of RNA
 - ii. Reverse transcriptase for conversion to DNA, integrase for integration
 - iii. Core and protein coat
 - i. Envelope with gp120 for attachment to CD4 receptor on T_H cells, macrophages, dendrites.
- c. Life cycle (movie)
 - i. Attachment and entry via gp120
 1. Binding to CD4 (normally for macrophage contact).
 2. Also uses other chemokine receptors (e.g. CCR5 and CXCR4). Other potential receptors to be discovered.
 - ii. Reverse transcription and integration.
 - iii. Expression. Proteases must process viral proteins before assembly. E.g. gp160 is cleavage into gp120 and gp41 (another envelope protein).
 - iv. Assembly and release.

C. Pathology

- a. Transmission (Fig. 19.17)
 - i. Bodily fluids – blood (highest concentration), breast milk, placental transfer.
 - ii. Modes of transmission - sexual contact, nursing, transfusions, injecting drug use
- b. Types of infection (Fig. 19.13)
 - i. Latent
 1. Provirus is integrated but is not actively expressed.
 2. Some latent virions can be produced but will not exit cell. Remain in vacuoles or can move to new cells during cell-cell fusion. (Fig. 19.14)
 3. Can persist for decades, especially in memory cells.
 - ii. Active – high expression.
- c. Pathogenesis (Fig. 19.15)
 - i. Acute – flu-like symptoms. Spike in viral load, crash in CD4 T cell population. 2 weeks.
 - ii. Asymptomatic – no outward symptoms. Seroconversion. Crash in viral load, recovery of T cells but slow decline over time. 2-10 years.
 - iii. Symptomatic – opportunistic infections (e.g. persistent yeast infection). T cell population getting low. Months to a few years.

- iv. AIDS – Severe infections, pneumonia, Kaposi’s sarcoma. Significant loss of both B and T cells. Viral load increases. Months – death.
 - d. Pathogenicity
 - i. Latent infection – very little or no RNA and protein made for detection.
 - ii. High mutation rate- reverse transcriptase is very error-prone. gp120 changes constantly.
 - iii. Immune suppression- infects the immune system itself.
 - e. Epidemiology
 - i. 25 million dead (1.8 million in 2009), 33 million living (2.6 million incidences in 2009)
 - ii. Present distribution (Fig. 19.16)
 - 1. US – 1 million. Usually through injecting drugs and homosexual males.
 - 2. Sub-Saharan Africa – 30 million. Some populations are 25% infected. Heterosexual contact.
 - 3. Asia – 5 million. Numbers are expected to explode. Heterosexual contact, sex workers. Clades A/E is especially adapted to heterosexual transmission through mucous membranes.
 - f. Diagnostics
 - i. Detect antibodies to HIV – ELISA, Western blot
 - ii. Detect viral load in plasma – PCR, nucleic acid hybridization
- D. Therapies
- a. Targets of HIV life cycle
 - i. Nucleoside analogs – mimic dNTPs that block reverse transcription. E.g. AZT, ddI.
 - ii. Inhibitors of reverse transcriptase – these drugs target RT itself.
 - iii. Blockers of integrase – small oligonucleotides block the integration reaction.
 - iv. Protease inhibitors – block protease to prevent gp160 cleavage.
 - v. Cocktails (combination of drugs) work the best, such as HAART (highly active antiretroviral therapy).
 - b. Boost immunity
 - i. Thymic and adrenal hormones can increase numbers of T cells.
 - ii. IL-2 increases CD4 cells.
 - c. Virus decoys – create a protein or antibody that will bind up HIV and prevent it from binding a cell. Target gp41 and gp120.
 - d. Vaccine – problem of developing because of high mutation rate of HIV.

Chapter 21 – Diseases of Skin

- A. Overview
 - a. Skin structure review (Fig. 21.1)
 - i. Epidermis, dermis, subcutaneous layers.
 - ii. Dermal layers contain vessels, nerves, hair follicles and glands.
 - b. Skin lesions (Fig. 21.2)
 - i. Vesicles – small, raised, fluid-filled. (e.g. chickenpox)
 - ii. Bullae – large, raised, fluid-filled. (e.g. impetigo)
 - iii. Macules – flat, reddish, no fluid. (e.g. measles, ringworm)
 - iv. Papules (pustules) – raised, pus-filled. (e.g. dermatitis)
- B. Chickenpox/shingles – e.g. of viral infection (Fig. 21.10)
 - a. Etiology – Varicella zoster in initial infection. Herpes zoster in recurrence.
 - b. Pathogenicity – latent in nerve cells in spinal cord.

- c. Pathogenesis
 - i. Chickenpox
 - 1. 2 week incubation, vesicles form on skin for 3-4 days followed by rupture and scabbing. Mild symptoms.
 - 2. Complications: Reye's Syndrome – vomiting, brain damage. Not understood, but complicated by aspirin.
 - ii. Shingles
 - 1. Rare recurrence of zoster is shingles. Localized vesicles at skin along nerve routes, pain, mild symptoms. 17% mortality rate in immunocompromised patients.
 - 2. Complications: Postherpetic neuralgia – nerve damage. Long-term pain.
 - d. Epidemiology
 - i. Transmission – direct contact and droplet. Highly contagious
 - ii. Chickenpox occurs in children (usually 5-9yr), shingles in adults (>45yr)
 - iii. In US, 95% have had chickenpox, 20% get shingles.
 - e. Treatment
 - i. Shingles: antiviral drugs – e.g. acyclovir, valtrex
 - f. Vaccines – 1995 – live attenuated vaccine available.
- C. *Pseudomonas dermatitis* – e.g. of bacterial infection
- a. Etiology – *Pseudomonas aeruginosa*. G- rod, opportunistic, lives in moist environments.
 - b. Pathogenicity
 - i. Biofilm – allows growth on medical equipment and on target tissues
 - ii. Toxins
 - 1. Endotoxin – LPS component
 - 2. Exotoxins – has both A-B and membrane-disrupting types
 - iii. Enzymes – proteases and hemolysins to aid in invasion
 - c. Pathogenesis – up to 2 days incubation, papules around hair follicles for 2 weeks.
 - i. Complications: otitis externa (swimmers' ear) and toxemic effects if secondary.
 - d. Epidemiology
 - i. Transmission – direct contact and indirect through water/moist objects. Hot tub rash! Nosocomial too.
 - ii. Prone to outbreaks (e.g. hot tubs) and constant in hospitals
 - iii. Case study handout.
 - e. Treatment – antibiotics
 - f. Prevention – chlorination of pools/tubs
- D. Dermatormycosis (ringworm or tinea) – e.g. of fungal infection (Fig. 21.16)
- a. Etiology – fungal *Trichophyton*, *Microsporum*, *Epidermophyton* spp.
 - b. Pathogenicity – lives off keratin in skin and nails. Athlete's foot, jock itch, ringworm just indicate where on body.
 - c. Pathogenesis – 1-2 weeks incubation, circular, macules for 2 weeks. If chronic, may last for months
 - d. Epidemiology
 - i. Transmission – direct contact and with fomites.
 - ii. Children and immunocompromised are prone to scalp ringworm
 - e. Treatment – topical fungicides
- E. Pediculosis (lice) – e.g. of animal infections (Fig. 21.19)
- a. Etiology – *Pediculus humanus*. Lice feed on head or body. Eggs (nits) are laid on hair shaft. A louse can live for 1 month.
 - b. Pathogenicity – jump from scalp to scalp
 - c. Pathogenesis – Itching due to allergic reaction to saliva. May develop after several weeks.

- d. Epidemiology
 - i. Transmission – direct contact, especially head to head.
 - ii. Children are susceptible. No correlation with hygiene.
- e. Treatment – topical insecticides, combing.

Chapter 22 – Diseases of the Nervous System

- A. Overview of nervous system
 - a. Central nervous system (CNS) vs Peripheral (PNS) (Fig. 22.1)
 - b. Meninges – covers spinal cord. Has spinal fluid in subarachnoid space (Fig. 22.2)
- B. Poliomyelitis – e.g. of viral
 - a. Etiology – poliovirus: single-stranded RNA
 - b. Pathogenicity – inhibition of protein synthesis in cell prevents recognition by phagocytes
 - c. Pathogenesis – infects GI tract → lymph → blood → nerves.
 - i. GI tract is infected – mild symptoms occur in a few days.
 - ii. Viremia is when virus enters bloodstream. Most patients do not progress further.
 - iii. Paralysis after several weeks if virus enters CNS. Death occurs if motor nerves to diaphragm fail.
 - d. Epidemiology – widely eradicated in the west with occasional breakouts.
 - i. Transmission – vehicle: through feces-contaminated water.
 - ii. Children under 5 most susceptible
 - iii. 0.5% infected get paralysis
 - iv. Occasional outbreaks but widely eradicated in west. Most cases in Asia, Africa.
 - e. Vaccines (Fig. 22.11)
 - i. No viable treatments
 - ii. 1954 – Salk vaccine (inactivated) (IPV)
 - iii. 1963 – Sabin vaccine (attenuated) (OPV) – more effective but no longer used.
Few cases of mutants. Now have returned to IPV
- C. Bacterial Meningitis – e.g. of bacterial
 - a. Etiology – 50 kinds, but most common are *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Neisseria meningitidis*. Opportunistic. (Fig. 22.3)
 - b. Pathogenicity - capsulation evades phagocytosis. Produce endotoxins.
 - c. Pathogenesis – hours to few days incubation, mild symptoms first but if moves to CSF, can cause convulsions, coma.
 - d. Epidemiology
 - i. Transmission – direct contact, sometimes droplet
 - ii. Affects mostly children. NM in college dorms and SP in adults
 - e. Treatments – antibiotics. Spinal tap to ID organism.
 - f. Vaccines – conjugated vaccines exist for all. Hib lower because of use.
- D. Trypanosomiasis (sleeping sickness) – e.g. of protozoan infection
 - a. Etiology – Protozoan *Trypanosoma brucei* by Tsetse fly vector.
 - b. Pathogenicity – high antigenic variation of surface proteins, sheds coats during life cycle (Fig. 22.16)
 - c. Pathogenesis – infects blood. Mild symptoms progress to affect kidneys, lymph, cardiovascular. When in CNS, then listlessness.
 - i. Gambiense – chronic, takes years
 - ii. Rhodesiense – acute, takes months
 - d. Epidemiology
 - i. Transmission – vector – tsetse fly bite.

- ii. Occurrence – Sub-Saharan Africa. 0.5 million prevalence, 30,000 incidences/year.
- e. Treatments
 - i. Chemotherapy – drugs that inhibit growth (e.g. suramin)
 - ii. Prevention – pesticides to eliminate fly
- f. Vaccine – underdevelopment but difficult because of antigenic variation and cost.

Chapter 23 – Diseases of the Circulatory System

A. Overview of the Circulatory System

- a. Vessels (Fig. 23.1)
 - i. Arteries → arterioles → capillaries → venules → veins
 - ii. Arteries and arterioles bring blood away from heart.
 - iii. Capillaries allow exchange of materials with tissues
 - iv. Veins and venules bring blood to the heart
- b. Heart
 - i. The main pump of the circulatory system
 - ii. Has two thin-walled atria that pump blood into the ventricles
 - iii. Has thick-walled ventricles that pump blood to all other body organs
- c. Blood
 - i. Plasma – noncellular portion containing water, salts, nutrients, etc.
 - ii. Cells – red, white, and platelets.

B. Bacterial Endocarditis – bacterial

- a. Etiology – mostly *Streptococcus* and *Staphylococcus* sp. Sometimes fungi
- b. Pathogenicity
 - i. Opportunistic in patients with heart defects.
 - ii. Clotting and biofilm production on heart valves
- c. Pathogenesis
 - i. Deformed valve gives greater chance of clots forming (Fig. 23.4)
 - ii. If bacteremia occurs (circulatory infection), biofilm (vegetation) may form at clots.
 - iii. Vegetation leads to continuous bacteremia, metastasis, and clots elsewhere in the circulatory system.
 - iv. Mild symptoms, heart murmur, swelling, pain.
- d. Epidemiology
 - i. Occurrence – generally in older adults with heart defects from autoimmune diseases, congenital defects, or surgery.
 - ii. Link with dental procedures or surgery due to risk of bacteremia
- e. Treatment
 - i. Antibiotics

C. Filariasis – animal

- a. Etiology – roundworm *Wuchereria bancrofti*
- b. Pathogenicity – thick outer walls are resistant to immune attack
- c. Pathogenesis
 - i. Life cycle include bite of mosquitoes, transferring microfilariae.
 - ii. Larvae will grow and migrate to lymphatic vessels.
 - iii. After ~6 months, adults will mate to produce more microfilariae.

- iv. Symptoms: swelling that can lead to elephantiasis.
- d. Epidemiology
 - i. Transmission – by mosquito bite. Microfilariae circulate at night, when mosquitoes are most active.
 - ii. Occurrence
 - 1. Found in tropical regions of Africa, Asia, and Central/South America.
 - 2. 120 million infected
- e. Treatment
 - i. Diagnosis: blood smears to detect microfilariae.
 - ii. Treated with drugs such as metronidazole. Pressure wrapping can relieve swolleness.
 - iii. Prevention by mosquito eradication.

Chapter 24 – Diseases of the Respiratory System

- A. Overview of Respiratory System
 - a. Upper – everything above the bronchi (Fig. 24.1)
 - i. Mouth/nasal passage, pharynx, larynx, trachea
 - b. Lower – bronchi and below (Fig. 24.2)
 - i. Bronchi, alveoli
- B. Influenza (Flu) - Viral
 - a. Etiology – Influenzavirus (ssRNA minus strand)
 - i. Structure (Fig. 24.16)
 - 1. 2-8 minus strand ssRNA in genome
 - 2. H-spikes – hemagglutinin (refers to clumping with antibody) used for attachment to host cells.
 - 3. N-spikes – neuraminidase cleaves sialic acid to allow budding during exit.
 - ii. Strains (Tab. 24.2)
 - 1. A is most virulent. High mutation rate. Crosses species more easily
 - 2. B is moderately virulent and confined to geographic regions. Lower mutation rate and only in humans.
 - 3. C has low virulence. Lacks N spikes. Limited human reservoirs.
 - b. Pathogenicity
 - i. High antigenic variation in spikes.
 - ii. Antigenic drift – mutations causing change.
 - iii. Antigenic shift - recombination and assortment of RNA genomes between variants may explain species crossing (e.g. bird flu).
 - c. Pathogenesis
 - i. Attacks epithelial lining of upper respiratory tract, especially ciliated mucosal cells. These cells become more prone to secondary infections
 - ii. Very rapid onset, usually in hours, and mild symptoms for days
 - iii. Complications: bronchitis and pneumonia in immunocompromised, old, and young.
 - d. Epidemiology
 - i. Transmission – droplet, highly contagious
 - ii. Generally affects immunocompromised, old, and young more.
 - iii. 50,000-70,000 die every year in US. ¼ to ½ million worldwide.

- iv. Can be pandemic and seasonal.
 - v. Famous 1918 pandemic killed 20 million. Current bird flu is similar to progression of 1918 flu.
 - e. Treatments – antiviral drugs. E.g. amantadine blocks uncoating.
 - f. Vaccines
 - i. Current strains are used to make subunit vaccines (antigens). Usually use H-spike.
 - ii. Attenuated viruses are available as a spray: FluMist
- C. Tuberculosis - bacterial
- a. Etiology – *Mycobacterium tuberculosis*(Fig. 24.9)
 - b. Pathogenicity – attacks phagocytes. Can escape phagocytosis due to waxy cell walls. Tubercles and slow replication time prevents immune detection.
 - c. Pathogenesis
 - i. Replication time is very slow (~20 hrs)
 - ii. Symptoms can take months to years to show up and can last for years.
 - iii. Stages (Fig. 24.10)
 - 1. Infection begins inside macrophage at the aveoli. This attracts additional macrophages to site.
 - 2. A tubercle is formed to wall-off lesion.
 - 3. Tubercles enlarge as bacilli are released. Fills with liquid and air.
 - 4. Rupture releases bacilli and liquids into bronchioles. Leads to coughing and further destruction of aveoli.
 - iv. Infection can occur at other sites and cause casts due to destruction of tissues and fusion
 - d. Epidemiology
 - i. Transmission – droplet, highly contagious.
 - ii. Was a turn of the century disease (1/7 died of TB)
 - iii. Worldwide 1/3 infected. 9.4 million incidences and 1.7 million died in 2009.
 - iv. US 10-12 million infected. 11,000 incidences each year. Highest in immigrant groups (Fig. 24.12).
 - e. Treatments and diagnosis
 - i. Skin test – protein antigen is injected under skin and T cell hypersensitivity reaction shows up as rash. Follow up with x-ray and lab tests.(Fig. 24.11)
 - ii. Treat with antibiotics (e.g. isoniazid and rifapin). Tough to treat because hidden in tubercle and resistance. New drugs against lipid metabolism of pathogen are being developed.
 - f. Vaccine – attenuated bovine strain. Does not work well in adults and will give false-positive in skin tests.

Chapter 25 – Diseases of the Digestive System

- A. Overview of Digestive System
 - a. GI tract (Fig. 25.1)
 - i. Mouth → pharynx → esophagus → stomach → small intestine → large intestine
 - ii. Organs: Liver, pancreas, gall bladder
 - b. Tooth (Fig. 25.2)
 - i. Crown → root → neck
 - ii. Enamel → dentin → pulp
- B. Hepatitis - viral
 - a. Etiology – hepatitis virus A-E. Now a G and maybe H (Fig. 25.16)

- b. Pathogenicity – high variation in spikes.
 - c. Pathogenesis
 - i. Hep A – 2-6 weeks incubation, mostly subclinical with mild symptoms for days to weeks. Sometimes jaundice and rare liver damage.
 - ii. Hep B – 4-26 weeks incubation, mostly subclinical with greater chance of progression to liver damage. Can lead to cirrhosis and cancer. Can become chronic.
 - iii. Hep C – 2-22 weeks incubation, similar to B but more likely to be chronic (85%) and lead to cirrhosis and cancer.
 - d. Epidemiology (Fig. 22.15)
 - i. Transmission – fecal-oral for Hep A, parenteral for Hep B, C. Hep B and C are spread by sexual contact and blood/IDU.
 - ii. Hep A – high occurrence (maybe 50% get infected). Underreported.
 - iii. Hep B – high in hospital workers (10,000 incidence/year). High in young adults (130,000 estimated). 1.25 million carriers.
 - iv. Hep C – high in transfused patients. 100,000 incidences/year.
 - e. Treatments
 - i. Hep A – none. Sometimes antibodies.
 - ii. Hep B – interferon, nucleoside analogs
 - iii. Hep C – interferon, some antiviral drugs.
 - f. Vaccines – attenuated for Hep A and subunit for Hep B. None for Hep C
- C. Tooth Decay - bacterial
- a. Etiology – usually initiated by *Streptococcus mutans* but hundreds of others also involved
 - b. Pathogenicity
 - i. Plaque (biofilm) formation
 - 1. Proteins in saliva coat tooth to form *pellicle*
 - 2. *S. mutans* sticks to pellicle
 - 3. Dextran is made (glucose polymer)
 - 4. More bacteria stick to teeth
 - 5. Lactic acid begins to be produced
 - ii. Lactic acid buildup slowly decays enamel of teeth. Tartar is a type of plaque.
 - iii. *S. mutans* prefers crevices
 - c. Pathogenesis
 - i. Months-years.
 - ii. Plaques begin to form in 24 h.
 - iii. Decay passes through enamel, dentin, and pulp. Abscesses form (Fig. 25.4)
 - iv. Gum disease (periodontal) is similar. (Fig. 25.5)
 - 1. Gingivitis is early stage, periodontitis is chronic
 - d. Epidemiology – common! Probably 100% incident of at least minor decay. Association with sucrose consumption.
 - e. Treatments – fillings, pulling teeth. For gum disease, antibacterial mouthwashes, antibiotics, and surgery.
 - f. Prevention
 - i. Lower sucrose – sucrose is broken down to use in formation of dextran.
 - ii. Fluoridated water – strengthens enamel.
 - iii. Brushing and flossing – removes plaques. Allows better saliva access.
- D. Giardiasis – protozoan
- a. Etiology – *Giardia lamblia*(Fig. 25.18)
 - b. Pathogenicity – life cycle includes encystation in small intestine. Adults feed on mucous.

- c. Pathogenesis – days incubation. Weeks to months of mild symptoms plus diarrhea, cramps, weight loss, dehydration. Flatulence and odor due to H₂S gas production. “Montezuma’s Revenge,” “backpacker’s diarrhea”
- d. Epidemiology
 - i. Transmission – fecal-oral by vehicle (water) and fomites.
 - ii. Effects 2% of adults and 6-8% of children in developed countries, nearly 33% of people in developing countries
- e. Diagnosis
 - i. Stool samples show presence of mucous sheets and cysts.
 - ii. ELISA is now used.
- f. Treatment – antiprotozoan drugs (e.g. metronidazole)

Chapter 26 – Diseases of the Reproductive System

- A. Overview of the Reproductive System (Fig. 26.1-3))
 - a. Males have longer urethras than females.
 - b. Females have greater separation of urinary and sex organs.
 - c. Females have closer proximity of anus and urinary openings.
- B. Genital Herpes - viral
 - a. Etiology – Herpes simplex virus 2 (HSV-2), a dsDNA virus. Sometimes HSV-1 (usually oral).
 - b. Pathogenicity – antigenic variation, latency in nerve ganglia.
 - c. Pathogenesis (Fig. 26.13)
 - i. Incubation less than 1 week, vesicles around affected areas for a few weeks. Females are more affected because of greater mucosal surface area. Complications in females: miscarriages, passing to child, greater risk of HIV.
 - ii. Migrates to nearby ganglia and remains latent. May recur frequently (88% for HSV-2, 50% for HSV-1) and travels down nerves to original site of infection.
 - iii. Neonatal herpes
 - 1. Can be passed by mother or contaminated hospital equipment.
 - 2. Very serious or lethal to infant, especially when neural/visceral.
 - d. Epidemiology
 - i. Transmission – direct, sexual contact especially during symptoms. However, there are “shedders” that can spread virus without outbreaks.
 - ii. Prevalence – 67 million in US.
 - iii. One out of six people between the ages of 14-19 years old have genital HSV-2 in the US.
 - e. Treatment/Prevention
 - i. Condoms may not protect if lesions are not covered.
 - ii. Antivirals for treatment (acyclovir)
 - iii. Vaccines: none yet, but some under development. Problem is most people already harbor HSV.
- C. Gonorrhea - bacterial
 - a. Etiology – *Neisseria gonorrhoeae*. G- diplococcus. (Fig. 26.7)
 - b. Pathogenicity
 - i. Attaches to mucosal cells using fimbriae.
 - ii. Invades space between cells. Produces endotoxins to damage mucosa.
 - iii. Produces proteases to cleave structural proteins and to cleavage IgA.
 - iv. Resists phagocytosis.
 - c. Pathogenesis – fast, days. (Fig. 26.6)

- i. Affects eyes, rectum, urethra, genitals. Inflammation and pus forms at infection sites. 2-7 days incubation, symptoms within 2 weeks.
 - ii. Males: lower rate of infection (20-30%). Many infected are asymptomatic. Mild symptoms of pain, discharge with rare progression to blocked urethra, sterility.
 - iii. Females: higher rate (60-90%) due to greater surface area. Many asymptomatic. More pronounced symptoms (rash, soreness, bleeding, discharge). Can lead to systemic diseases.
 - d. Epidemiology
 - i. Transmission – direct – sexually transmitted.
 - ii. In sexually active teens and adults.
 - iii. Incidence is decreasing, but still about 300,000 cases a year in US. (Fig. 26.5)
 - e. Treatments
 - i. Antibiotics – now resistant to penicillin. Second-line drugs available but resistance rising.
 - ii. Prevention – use of condoms.
- D. Candidiasis – fungal (yeast infection)
 - a. Etiology – *Candida* spp. especially *albicans*. (Fig. 21.17)
 - b. Pathogenicity – opportunistic during immunocompromised state or suppressed normal flora. This can occur by stress, other diseases, antibiotics etc.
 - c. Pathogenesis – 1 week incubation, 2 weeks of itching, discharge. Males can get UTI. Thrush occurs in the oral cavity with white lesions.
 - d. Epidemiology
 - i. Transmission – none
 - ii. Occurrence – most women get it in their lifetime (about ½ by age 25)
 - e. Treatment – antifungal (miconazole)