

Digestive System Infections

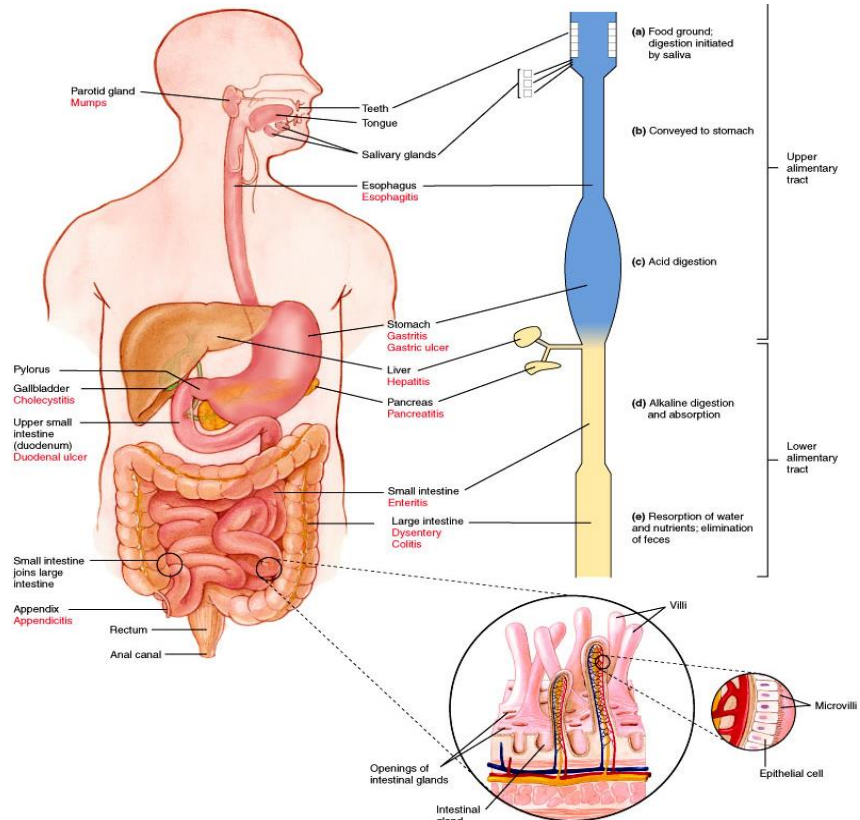
Lecture 22 – Dr. Gary Mumaugh

Digestive System Infections

- Sometimes referred to as the alimentary or gastrointestinal tract
- Passageway running from mouth to anus
- One of the body's boundaries from the environment
- Major route of microbial invasion
- Main purpose to provide nutrients to body
- Divided into upper and lower tracts

Normal Microbiota

- Important in protecting body from invasion
- Flora of digestive tract mainly found in the oral cavity and intestines
 - Esophagus has very little flora
 - Normal stomach is devoid of microorganisms
 - Killed by stomach acid
- Mouth
 - Relatively few species colonize oral cavity
 - Streptococcal species most common
 - Host limits number of bacteria on mucous membranes
 - Membrane cells constantly shedding
 - Teeth are nonshedding surface
 - Large numbers of bacteria can collect and form biofilm
 - Masses of bacteria termed dental plaque
- Intestines
 - Small number of bacteria colonize upper small intestine
 - Large intestine contains very high numbers of organisms
 - Approximately 10^{11} bacteria per gram of feces
 - That is 100 billion bacteria!!!!
 - High population is due to abundance of nutrients in feces
 - *Escherichia coli* and other enterobacteria predominate bacterial population
 - Important source of opportunistic infections
 - Especially of the urinary tract
 - Normal intestinal flora prevent pathogenic colonization of large intestine

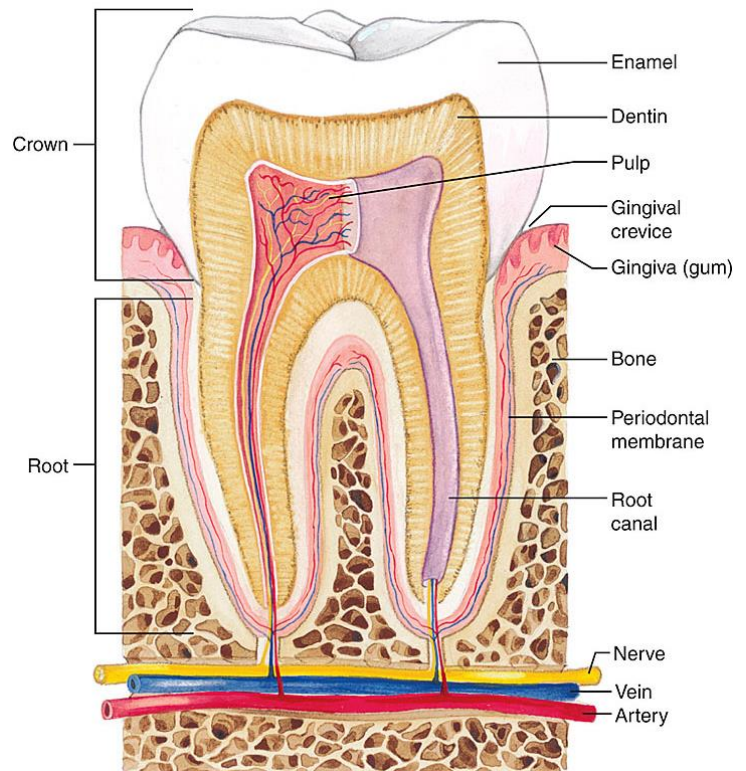


Bacterial Disease of the Upper Digestive Tract

- Most common human diseases occur in the mouth
 - Bacterial infections of the stomach are very uncommon
 - When infections do occur they can lead to ulcers
- Oral flora can enter bloodstream during dental procedures
 - Can cause sub-acute bacterial endocarditis
- Chronic gum infections play role in arterial sclerosis and arthritis

Dental Caries

- Most common infectious disease of human beings
 - Main reason for tooth loss
 - As many as 47% of US population over 65 have lost all of their teeth
- Symptoms
 - Usually advanced before symptoms arise
 - Once symptoms develop they include
 - Throbbing pain
 - Noticeable discoloration, roughness or defect in tooth
 - Tooth can break while chewing
- Causative Agent
 - *Streptococcus mutans* most common cause
 - Colonize teeth
 - Cannot colonize mouth
 - Thrive in acidic conditions
 - Produce lactic acid from sugar metabolism
 - Produce glucans from sucrose
 - Glucans essential for production of dental caries
- Pathogenesis
 - Begins with adherence of bacteria to specific receptors on teeth
 - Production of glucans from dietary sucrose
 - Glucans bind organisms together and to tooth
 - Formation of cariogenic plaque
 - Dietary sugar produces drop in pH
 - Acidic environment causes calcium phosphate in teeth to dissolve, which creates pits and fissures for colonization



Dental Caries

- Prevention
 - Restriction of dietary sucrose
 - Most important preventative
 - Fluoride required for teeth to resist acid
 - Makes tooth enamel harder
 - More resistant to dissolving in acid
- Treatment
 - Mechanical removal of plaque via tooth brushing and dental floss
 - Reduces incidence by approximately 50%
 - Pits and fissures prevented by application of sealant
 - Treatment of caries requires drilling out cavity and filling with amalgam



Periodontal Disease

- Symptoms
 - Majority of cases asymptomatic
 - Common symptoms
 - Bleeding gums
 - Gum sensitivity
 - Bad breath
 - Teeth become loose and discolored
 - Receding gums
- Causative agent
 - Caused by dental plaque
 - Plaque forms at area where gum joins the teeth
 - Numerous bacteria reside in plaque
- Pathogenesis
 - Plaque forms on teeth at gum line often referred to as tartar
 - Extends into gingival crevice
 - Inflammation occurs known as gingivitis
 - Progression results from enzymes released from organisms that weaken gingival tissue
 - Causes gingival crevice to widen and deepen
 - Gram-negative organisms increase and release exotoxins
 - Exotoxins attack host tissue
 - Membranes and bone softens - Tooth may be lost

Periodontal Disease

- Epidemiology
 - Mainly disease of individuals over 35
 - 90% of those over 65 have some periodontal disease
 - People with underlying immunodeficiency often have severe disease
- Prevention and treatment
 - Careful flossing and brushing
 - Combined with semi-annual polishing and cleaning
 - Treated by cleaning out inflamed gingival crevice and removing plaque
 - In advanced cases surgery is usually required

Trench Mouth

- Also known as Vincent's disease or acute necrotizing ulcerative gingivitis
 - ANUG
- Condition distinct from other forms of periodontitis
- Disease rampant during World War I
 - Soldiers in trenches unable to attend to proper mouth care
- Symptoms
 - Abrupt onset
 - Fever
 - Bleeding and painful gums
 - Foul odor
- Causative agent
 - Oral spirochete from *Treponema* genus
 - Bacteria most likely work synergistically with other anaerobic bacteria of mouth
- Pathogenesis
 - Precise mechanism unknown
 - Presumed to act with other bacteria to destroy tissue
 - Plaque is always present
 - Bacterial invasion causes necrosis and ulceration
 - Mainly of gums between teeth
- Prevention and Treatment
 - Control directed at daily brushing and flossing
 - With semi-annual cleaning
 - Antibacterial treatment directed at spirochetes and anaerobic bacilli
 - Relieves acute symptoms
 - Must be followed up with extensive plaque removal
- Epidemiology
 - Can occur at any age
 - Arises in association with poor dental hygiene
 - Stress, malnutrition or immunodeficiency may contribute
 - Disease not contagious



Important Infections of the Teeth and Gums

	Dental Caries	Periodontal Disease	Trench Mouth
Symptoms	None until advanced disease. Late: discoloration, roughness, broken tooth, throbbing pain	Most cases asymptomatic until advanced disease. Bleeding, sensitive gums, bad breath, loosening of the teeth. Receding gums with exposed discolored tooth roots.	Abrupt onset of fever, painful bleeding gums, and a foul mouth odor
Incubation period	1 to 24 months before cavity is detectable	Months or years	Undetermined
Causative agent	Dental plaque populated with <i>Streptococcus mutans</i>	Dental plaque, cariogenic or not	Probably a spirochete of the genus <i>Treponema</i> acting with <i>Fusobacterium</i> , <i>Prevotella</i> , or other anaerobes
Pathogenesis	Bacteria in plaque produce acid from dietary sugars; slowly dissolves the calcium phosphate crystals composing the tooth; sucrose critical for cariogenic plaque formation.	Plaque forms at the gum margins and gradually extends into the gingival crevices. Bacterial products incite an inflammatory response. The crevices widen and deepen, and the proportion of anaerobes increases. Toxins and enzymes weaken the tissues holding the teeth and cause them to become loose.	The spirochetes and certain other anaerobes act synergistically to cause death of tissue, ulceration, and tissue invasion by spirochetes.
Epidemiology	Worldwide distribution, incidence depending on dietary sucrose, natural or supplemental fluoride. The young are more susceptible than the old.	Primarily a disease of those older than 35 years. Immunodeficient individuals are at increased risk of severe disease.	All ages are susceptible in association with poor mouth care, malnutrition, or immunodeficiency. It is not contagious.
Prevention and treatment	Restriction of dietary sucrose, supplemental fluoride, mechanical removal of plaque, sealing pits and fissures in childhood teeth.	Avoid buildup of plaque. Surgical treatment in severe cases to expose tooth roots and remove plaque and calculus.	Avoid buildup of plaque. Antibiotic treatment acutely, followed by removal of plaque and calculus.

***Helicobacter pylori* Gastritis**

- Symptoms
 - Range from belching to vomiting
 - Most are asymptomatic
 - Symptoms occur when infection is complicated with ulcers or cancer
 - Symptoms include
 - Abdominal pain
 - Tenderness
 - Bleeding
- Causative Agent
 - *Helicobacter pylori*
- Pathogenesis
 - Bacteria survive extreme acidity of the stomach
 - Able to neutralize environment
 - Organism uses flagella to corkscrew through mucosal lining
 - Inflammatory response begins
 - Mucus production decreases
 - Without mucus stomach lining not protected from acidic environment
 - Infection persists for years - Possibly for a lifetime

***Helicobacter pylori* Gastritis**

- Epidemiology
 - Infections tend to cluster in families
 - Transmission most likely fecal-oral route
 - Flies also capable of transmission
 - 20% of US population infected
 - Incidence increases with age
 - Almost 80% of those over 75 infected
 - Rates highest in lower socioeconomic groups
- Prevention and Treatment
 - No proven prevention measures
 - Infection can usually be eradicated with combined antibiotic treatment
 - Medication is also used to inhibit production of stomach acid

Symptoms	Initial infection: range from belching to vomiting. Localized abdominal pain and tenderness, bleeding when complicated by ulcer or cancer
Incubation period	Usually undetermined
Causative agent	<i>Helicobacter pylori</i> , a spiral, Gram-negative, microaerophilic bacterium, with sheathed flagella
Pathogenesis	Organisms survive the acidity of stomach juices by producing a powerful urease. Upon reaching the layer of mucus, they penetrate to the epithelial surface, where bacterial products incite an inflammatory response. Thinning of the mucus layer occurs, and 10% to 20% of infected individuals develop ulcerations. Only a small percentage develop cancer, but more than 90% of individuals with stomach cancers are infected with <i>H. pylori</i> .
Epidemiology	Probably fecal-oral transmission. Progressive increase with age, reaching almost 80% of those over 75.
Prevention and treatment	No proven preventive. Most infections are cured using two antibiotics together, plus a medication to suppress stomach acid.

Herpes *Simplex*

- Extremely widespread disease
 - Many manifestations
 - Most common form begins in mouth and throat
 - Infection persists for life
 - Virus transmissible with saliva
 - Disease usually insignificant
 - Disease can be fatal in immunodeficiency
- Symptoms
 - Typically begin during childhood
 - Common symptoms include
 - Fever and blisters
 - First lesions which lead to ulcers
 - Ulcers in mouth and throat
 - Generally painful
 - Heal without treatment
 - After healing disease becomes latent
 - Symptom recurrence usually begins on the lips
 - Marked by tingling, itching, burning or painful sensation
- Causative Agent - Herpes simplex virus (HSV)
 - Two types
 - HSV 1
 - Occurs mainly on the lips and mouth
 - HSV 2
 - Responsible for most genital infections
- Pathogenesis
 - HSV 1
 - Virus multiplies in epithelial cells of mouth or throat
 - Blisters form
 - Blisters contain large numbers of mature virions
 - Some virions carried to lymph vessels and nodes
 - Immune response produced limits infection
 - Some virions enter sensory nerves as latent virus
 - Latent virus can become infectious
 - Viruses are carried by nerves to skin or mucous membranes
 - Viruses produce recurrent disease
 - Stress can precipitate recurrences



Herpes *Simplex*

- Epidemiology
 - Extremely widespread
 - Infects 90% of some US inner-city populations
 - Estimated 20% to 40% of Americans suffer from recurrent disease
 - Transmitted by close physical contact
 - Virus can survive for several hours on plastic and cloth
 - Greatest risk of transmission is contact with lesion or infected saliva
- Prevention and Treatment
 - Antivirals such as acyclovir have proved effective in treatment

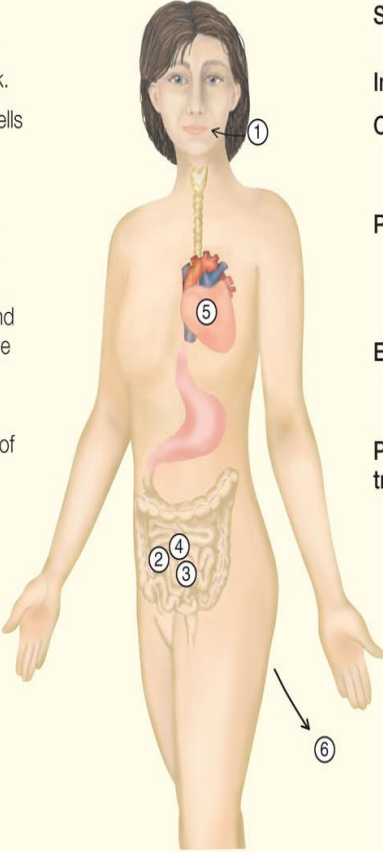
Symptoms	Initial infection: fever, severe throat pain, ulcerations of the mouth and throat. Recurrences: itching, tingling or pain usually localized to the lip, followed by blisters that break leaving a painful sore, which usually heals in 7 to 10 days
Incubation period	2 to 20 days
Causative agent	Herpes simplex virus (HSV), usually type 1
Pathogenesis	The virus multiplies in the epithelium, producing cell destruction and blisters containing large numbers of infectious virions. An immune response quickly limits the infection, but non-infectious HSV DNA persists in sensory nerves. This DNA becomes the source of infectious virions that are carried to the skin or mucous membranes, usually of the lip, causing recurrent sores.
Epidemiology	Widespread virus, transmitted by close physical contact. The saliva of asymptomatic individuals is commonly infectious.
Prevention and treatment	Acyclovir, penciclovir, and similar medications that target HSV DNA polymerase can shorten the duration of the illness or prevent recurrences. Sunscreens are helpful in preventing recurrences due to ultraviolet exposure.

Cholera

- Symptoms
 - Classic example of severe diarrhea
 - Can amount to loss of 20 liters of fluid per day
 - Often termed “rice water stools” due to appearance
 - Vomiting common in most cases
 - Usually occurs at the onset of disease
 - Many suffer muscle cramps
 - Caused by loss of fluid and electrolytes
- Causative Agent - *Vibrio cholerae*
 - Tolerates high alkaline environment
- Pathogenesis
 - Large numbers must be ingested to effect disease due to sensitivity to stomach acid
 - In small intestine, organisms adhere to epithelial lining and multiply there
 - Bacteria-produced toxin
 - Cholera toxin - Responsible for symptoms

Cholera

- Epidemiology
 - Fecal contamination of water most common source of transmission
 - Crabs and vegetable fertilized with human feces have also been implicated
 - Person with cholera may discharge at least 1 million bacteria per milliliter of feces
- Prevention
 - Depends largely on
 - Adequate sanitation
 - Availability of safe water supplies
 - Travelers should cook food immediately before eating
 - Fruit should be peeled personally
 - Avoid ice unless made with boiled water
- Treatment
 - Treatment depends on rapid replacement of fluids and electrolytes
 - Essential before irreversible damage to vital organs
 - Replacement of fluids and electrolytes decreases mortality to less than 1%



① *Vibrio cholerae*, the causative bacterium, enters the mouth with fecally contaminated food or drink.

② The bacteria attach to epithelial cells of the small intestine.

③ *V. cholerae* toxin enters the cells and prevents them from down-regulating secretion of water and electrolytes.

④ The epithelial cells pump water and electrolytes from the blood into the intestinal lumen, causing watery diarrhea.

⑤ Shock and death occur because of fluid loss from the circulatory system, unless the fluid can be replaced.

⑥ The bacteria exit the body with feces.

Symptoms	Abrupt onset of massive diarrhea, vomiting, muscle cramps
Incubation period	Short, generally 12 to 48 hours
Causative agent	<i>Vibrio cholerae</i> , a curved, alkali and salt tolerant, Gram-negative rod bacterium
Pathogenesis	Heat-labile exotoxin causes excessive secretion of water and electrolytes by the intestinal epithelium; leads to dehydration and shock.
Epidemiology	Ingestion of fecally contaminated food or water; sometimes natural sources associated with marine crustaceans.
Prevention and treatment	Purification of water, careful handwashing; vaccination. Treatment: Rehydration with a solution of electrolytes and glucose, given intravenously in severe cases; or similar electrolyte solution containing a glucose source given by mouth in milder cases.

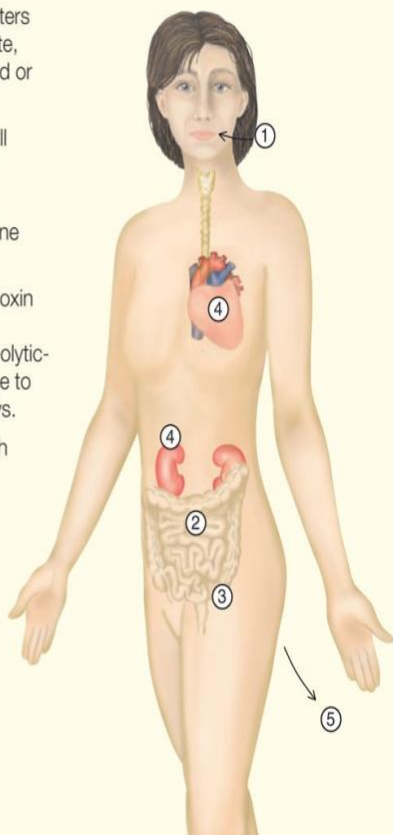
Shigellosis

- Symptoms
 - Classic symptom is dysentery
 - Other symptoms include
 - Headache
 - Vomiting
 - Fever stiff neck
 - Convulsions (rare)
 - Joint pain
 - Commonly fatal for infants in developing countries
- Causative Agent - Four species of *Shigella*
 - *S. flexnuri*
 - *S. boydii*
 - *S. sonnei*
 - *S. dysenteriae*
- Pathogenesis
 - First step is phagocytosis of bacteria by cells in the large intestine
 - Cells transport bacteria beneath epithelium
 - Bacteria adhere to specific receptors near base of epithelial cells
 - Bacteria multiply at high rate
 - *S. dysenteriae*
 - Rarely encountered in United States
- Epidemiology
 - Generally has a human source of transmission
 - Transmitted fecal-oral route
 - Organism not easily killed by stomach acid
 - Does not have a high infecting dose
 - As few as 10 organisms
 - Transmission occurs most often as a result of overcrowding
 - Also common in day cares and among homosexual men
 - Contaminated food and water also responsible for outbreaks
- Prevention and Treatment
 - Controlled by sanitary measures and surveillance of food handlers and water supplies
 - No vaccine available
 - Most important treatment is fluid and electrolyte replacement
 - Antimicrobials often used to shorten duration of symptoms
 - Also shorten time bacteria discharged in feces
 - However 20% of *Shigella* species are resistant to antibiotics of choice

***Escherichia coli* Gastroenteritis**

- Symptoms
 - Depends largely on virulence of infecting strain
 - Symptoms can range from vomiting and a few loose stools to profuse watery diarrhea to severe cramps and bloody diarrhea
 - Fever not usually prominent
 - Recovery usually occurs within 10 days
- Pathogenesis
 - Possesses two important virulence factors
 - Production of enterotoxin
 - Adherence to cells of small intestine
 - Virulence factors foster spread
- Causative Agent
 - Most diarrhea causing *E. coli* fall into four groups
 - Enterotoxigenic *E. coli* (ETEC)
 - Most common cause of traveler's diarrhea
 - Enteroinvasive *E. coli* (EIEC)
 - Disease closely resembles that of *Shigella* species
 - Enteropathogenic *E. coli* (EPEC)
 - Causes outbreaks in hospital nurseries and bottle fed infants in developing countries
 - Enterohemorrhagic *E. coli* (EHEC)
 - Often produces severe illness due to production of potent group of toxins
 - Toxins closely related to Shiga toxins
 - Most common strain O157:H7
- Epidemiology
 - Epidemics occur from
 - Person-to-person spread
 - Contaminated food and water
 - Unpasteurized milk and juices
 - Humans, domestic and wild animals all sources of pathogenic strains
- Prevention and Treatment
 - Prevention directed at
 - Hand washing
 - Pasteurization of drinks
 - Proper food preparation
 - Treatment includes
 - Replacement of fluids and electrolytes
 - Infants may require antibiotics
 - Antibiotics tend to prolong disease in adults
 - Traveler's diarrhea can be controlled with bismuth preparations
 - Pepto-Bismol

Escherichia coli Gastroenteritis

<p>① Pathogenic strain of <i>E. coli</i> enters the body by the fecal-oral route, usually with contaminated food or beverage.</p> <p>② Most strains colonize the small intestine and produce watery diarrhea.</p> <p>③ Others invade the large intestine and cause dysentery.</p> <p>④ Some strains produce Shiga toxin which is absorbed by the bloodstream and causes hemolytic-uremic syndrome with damage to red blood cells and the kidneys.</p> <p>⑤ The bacteria exit the body with feces.</p>		<p>Symptoms Vomiting and diarrhea; sometimes dysentery</p> <p>Incubation period 2 hours to 6 days</p> <p>Causative agent <i>Escherichia coli</i>, certain strains</p> <p>Pathogenesis Various mechanisms; attachment to small intestinal cells allows colonization; some strains produce one or more enterotoxins; some strains, resembling shigellas, invade large intestinal epithelium; others cause host cell membrane thickening and loss of microvilli, and may produce Shiga toxin.</p> <p>Epidemiology Common in travelers; can be foodborne or waterborne; fecal-oral route transmission, sometimes animal source.</p> <p>Prevention and treatment Sanitary precautions including careful handwashing; pasteurization of drinks, thorough cooking of meats; replacement of fluid loss; bismuth compounds for treatment.</p>
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Salmonellosis

- **Symptoms**
 - Generally characterized by
 - Diarrhea
 - Abdominal pain
 - Nausea
 - Vomiting
 - Fever
 - Symptoms vary depending on virulence of strain and number of infecting organisms
 - Symptoms are generally short-lived and mild
- **Causative Agent** - *Salmonella* species
- **Pathogenesis**
 - Bacteria sensitive to stomach acid
 - Large number required for infection
 - Bacteria adhere to receptors on epithelial cells of lower small intestine
 - Cells take up bacteria through phagocytosis
 - Bacteria multiply within phagosome discharged through exocytosis
 - Inflammatory response increases fluid secretion resulting in diarrhea

Salmonellosis

- Pathogenesis
 - Some strains of serotype *Salmonella* Typhi are not easily eliminated
 - Organisms cross membrane and resist killing by macrophages
 - Bacteria multiply within macrophages then carried to bloodstream
 - Organisms are released when macrophages die and invade tissues
 - Can result in abscess, septicemia and shock
- Epidemiology
 - Bacteria can survive long periods in the environment
 - Children are commonly infected
 - Generally by household pets such as turtles, iguanas and baby chicks
 - Most cases have an animal source
 - Enteric fevers, such as those caused by *Salmonella* Typhi are generally the exception
 - “Typhoid Mary” notorious carrier
 - Caused at least 53 cases over 15 years
- Prevention and Treatment
 - Control depends on reporting cases and tracing source of outbreak
 - Adequate cooking kills bacterium
 - Vaccine available for prevention of typhoid fever
 - Vaccine 50% to 75% effective
 - Surgical removal of gallbladder eliminates carrier state

Symptoms

Diarrhea and vomiting; rarely, prolonged fever, headache, abdominal pain, abscesses, and shock

Incubation period

Usually 6 to 72 hours; can be 1 to 3 weeks in typhoid fever

Causative agent

Salmonella enterica, motile, Gram-negative, enterobacteria

Pathogenesis

Invasion of the lining cells of lower small and large intestine, with penetration to underlying tissues; body's inflammatory response causes increase in fluid secretion. Sometimes survival within macrophages and spread throughout the body, destruction of Peyer's patches.

Epidemiology

Ingestion of food contaminated by animal feces, especially poultry. Human fecal source in typhoid fever-like illnesses.

Prevention and treatment

Adequate cooking and handling of food; attenuated vaccine against typhoid fever. Usually no antimicrobial advised unless invasion of tissues or blood occurs.

Campylobacteriosis

- Bacteria recognized in 1972
- Leading cause of bacterial diarrhea in United States
 - Estimated 2.4 million cases annually
 - Less than 1,000 fatalities
 - Fatalities mostly in elderly and immunocompromised
- Symptoms
 - Fever
 - Vomiting
 - Diarrhea
 - Abdominal cramps
 - Dysentery
 - Occurs in only about 50% of cases
- Causative Agent - *Campylobacter jejuni*
- Pathogenesis
 - Small infecting dose
 - Only 500 organisms required to initiate disease
 - Organisms penetrate intestinal epithelium
 - Bacteria multiply in and under cells and initiate inflammation
 - Sequel of disease is Guillain-Barré Syndrome
 - Begins abruptly within about 10 days of onset of diarrhea
 - Tingling of the feet leads to progressive paralysis of the legs, arms and rest of body
 - Most patients require hospitalization
 - 80% recover completely, 5% mortality despite treatment
- Epidemiology
 - Numerous foodborne and waterborne outbreaks have been reported
 - Most cases sporadic
 - Causative agent lives in intestine of variety of domestic animals
 - Particularly poultry
 - One drop of juice from raw chicken can contain infective dose
 - Epidemics have occurred due to unpasteurized milk
 - Person-to-person spread rare
- Prevention and Treatment
 - Prevention directed at proper treatment of water and food
 - Pasteurization of drinks and proper cooking and handling of raw food
 - Most cases of campylobacteriosis subside without antimicrobial treatment in about 10 days
 - Erythromycin recommended for severe cases

Hepatitis A

- Symptoms
 - Fatigue
 - Fever
 - Loss of appetite
 - Nausea
 - Right side abdominal pain
 - Dark-colored urine and clay colored feces
 - Jaundice
 - Children less than 6 years old are generally asymptomatic nearly 70%
 - 20% of adults require hospitalization
 - Full recovery in about 2 months
 - Formally called infectious hepatitis
- Pathogenesis
 - Transmission from ingestion of contaminated food or water
 - Ingested virus reaches liver by unknown route
 - Liver main site of viral replication
 - Only tissue know to be damaged
 - Virus is released into bile
 - Virus-laden bile eliminated in feces
- Epidemiology
 - Spreads via fecal-oral route
 - Many outbreaks originated from restaurants
 - Due to infected food handler
 - Raw shellfish frequent source of infection
 - Low socioeconomic groups make up high percentage of infected
 - High risk groups include people in day care and nursing homes and homosexual men
- Causative Agent - Hepatitis A virus (HAV)
- Prevention and Treatment
 - Vaccine available since 1995
 - Indicated for travelers to deprived regions, homosexual men, sewer workers and healthcare workers
 - Gamma globulin contains HAV antibody, can be given to individuals that have been exposed
 - Afford short term protection if given within 2 weeks of exposure

Hepatitis B

- Symptoms
 - Similar to hepatitis A
 - Symptoms for hepatitis B more severe
 - Causes death in 1% to 10% of hospitalized cases
 - Formerly known as serum hepatitis
- Causative Agent - Hepatitis B virus (HBV)

Hepatitis B

- Pathogenesis
 - HBV carried in liver
 - Mechanism of liver damage unknown
 - Damage most likely results from immune response
 - Virus replicates via reverse transcriptase
 - Viral DNA transported to host nucleus
 - Host mRNA makes RNA copy
 - RNA copy transcribed by viral reverse transcriptase
 - New DNA copy is genome for new virus
 - New viruses bud from host cell
- Epidemiology
 - Progressive rise in reported cases between 1965 and 1985
 - Incidence appears to have plateaued
 - HBV spread mainly by blood, blood products and semen
 - Carriers are of major importance - Often unaware of infection
 - Risk factors for infection include
 - Sharing needles
 - Tattooing and piercing with contaminated instruments
 - Shared toothbrushes, razors and towels
 - Sexual intercourse responsible for nearly 50% of cases in United States
- Prevention and Treatment
 - Vaccine approved in 1980s
 - New more effective vaccine available since 1986
 - Vaccination against HBV can help prevent liver cancer caused by the virus
 - Passive immunization with HBIG (hepatitis B immune globulin) offers immediate protection
 - No curative treatment

Hepatitis C

- Symptoms
 - Same as hepatitis A and hepatitis B
 - Generally milder
 - 65% are asymptomatic - 25% have jaundice
- Causative Agent - Hepatitis C virus
- Pathogenesis
 - Few details known
 - Infection transmitted via contact with infected blood
 - Incubation period average 6 weeks
 - Over 80% develop chronic infections
 - Virus infects the liver
 - Incites inflammatory and immune responses
 - Disease comes and goes
 - Individuals have times of near normalcy
 - 10% to 20% will develop cirrhosis or liver cancer

Hepatitis C

- Epidemiology
 - Mechanism of exposure not always obvious
 - Risk factors include
 - Sharing toothbrush, razors, towels
 - Tattooing and piercing with unclean instrument
 - Sharing syringes
 - 60% of US cases due to sharing needles
 - Transmission via intercourse most likely rare
 - Can occur if multiple partners
- Prevention and Treatment
 - No vaccine for HCV
 - Vaccination against A and B seem to give some protection
 - Avoidance of alcohol to limit effect on liver
 - No satisfactory treatment
 - Some are helped by interferon therapy

	Hepatitis A	Hepatitis B	Hepatitis C
Causative agent	Non-enveloped, single-stranded RNA picornavirus, HAV	Enveloped, double-stranded DNA hepadnavirus, HBV	Enveloped, single-stranded RNA flavivirus, HCV
Mode of spread	Fecal-oral	Blood, semen	Blood, possibly semen
Incubation period	3 to 5 weeks (range, 2 to 7 weeks)	10 to 15 weeks (range, 6 to 23 weeks)	6 to 7 weeks (range, 2 to 24 weeks)
Prevention	Inactivated vaccine; immune globulin	Subunit-vaccine; immune globulin	No vaccine
Comments	Usually mild symptoms, but often prolonged; full recovery; no long-term carriers; combined hepatitis A and hepatitis B vaccine available	Symptoms often more severe than hepatitis A; progressive liver damage in 1% to 6% can lead to cirrhosis and cancer, chronic carriers; can cross the placenta; combined hepatitis A and hepatitis B vaccine available	Usually few or no symptoms; progressive liver damage or cancer in 10% to 20% of cases; chronic carriers
	Hepatitis D	Hepatitis E	Hepatitis G
Causative agent	Defective single-stranded RNA virus, HDV	Non-enveloped, single-stranded RNA calcivirus, HEV	Single-stranded RNA flavivirus
Mode of spread	Blood, semen	Fecal-oral	Blood, possibly semen
Incubation period	2 to 12 weeks	2 to 6 weeks	Weeks
Prevention	No vaccine	No vaccine	No vaccine
Comments	Prior or concurrent HBV infection necessary; can cause worsening of hepatitis B; can cross the placenta	Similar to hepatitis A, except severe disease in pregnant women, same or related virus in rats	Usually mild symptoms; persistent viremia for months or years

Giardiasis

- Symptoms
 - In epidemic approximately two-thirds of exposed individuals develop symptoms
 - Incubation period 6 to 20 days
 - Symptoms range from mild to severe
 - Indigestion and nausea to vomiting and explosive diarrhea
 - Symptoms usually disappear within 4 weeks
 - Some cases become chronic
- Causative Agent - *Giardia lamblia*
- Pathogenesis
 - Cyst responsible for infection
 - Due to fact they resist stomach acid
 - Trophozoites attach to epithelium of small intestine and move to large intestine
 - In severe cases, organism may cover entire intestinal surface
 - Interfere with proper functioning of intestine
 - Results in malnutrition, bulky feces and excess gas
- Epidemiology
 - Single human stool can carry 300 million cysts
 - Only 10 required for infection
 - Cysts can survive in cold water up to 2 months
 - Chlorination often ineffective against cysts
 - Filtration required
 - Transmission risk increases with risky behavior
 - Anal and oral sex
 - Transmission usually via fecal-oral route
 - Particularly in contaminated water
 - Sources of organism include
 - Beavers, raccoons, muskrats, dogs, cats, humans
- Prevention and Treatment
 - Filtration of water
 - Organisms sensitive to heat
 - Boil water 1 minute before using
 - Good hygiene practices

Amebiasis

- Symptoms
 - Commonly asymptomatic
 - Symptoms range from chronic diarrhea to acute dysentery and death
- Causative Agent
 - *Entamoeba histolytica*
- Pathogenesis
 - Infection begins with ingestion of mature cysts
 - Organisms released from cyst in small intestine
 - Feed non intestinal mucus and bacteria
 - Some strains produce toxic enzyme which kills epithelium and can lead to abscess
 - Irritating effect of feeding organisms causes intestinal cramps
 - In presence of intestinal ulceration diarrheal fluid is often bloody
 - This condition referred to as amebic dysentery
- Epidemiology
 - Distributed worldwide
 - More common in tropical areas
 - Human only reservoir
 - Transmission fecal-oral route
 - Mainly associated with poverty, male homosexuality and migrant workers
- Prevention and Treatment
 - Depends on
 - Sanitary measures
 - Avoiding fecal contamination of food and drinking water
 - Metronidazole, paromomyacin are effective treatments

Symptoms	Diarrhea, abdominal pain, blood in feces
Incubation period	2 days to several months
Causative agent	<i>Entamoeba histolytica</i> ; a protozoan of the group Sarcodina
Pathogenesis	Ingested cysts liberate trophozoites in the small intestine; upon reaching the large intestine, trophozoites feed on mucus and cells lining the intestine; enzymes are produced that allow penetration of the intestinal epithelium, sometimes the intestinal wall and blood vessels, and thence to the liver and other organs, resulting in abscesses.
Epidemiology	Ingestion of fecally contaminated food or water; disease associated with poverty, homosexual men, and migrant workers.
Prevention and treatment	Good sanitation and personal hygiene. Treatment: metronidazole, paromomyacin.

