Microbial Diseases of the Cardiovascular and Lymphatic Systems

Chapter 23
I. Structure and Function of the Cardiovascular and Lymphatic systems

• A. INTRODUCTION
  – The heart, blood, and blood vessels make up the cardiovascular system.
  – Lymph, lymph vessels, lymph nodes, and lymphoid organs constitute the lymphatic system.

• B. Structure and Function of the Cardiovascular System
  – The heart circulates substances to and from tissue cells.
  – Blood is a mixture of plasma and cells.
  – Plasma transports dissolved substances. Red blood cells carry oxygen. White blood cells are involved in the body’s defense against infection.

Human cardiovascular system
I. Structure and Function of the Cardiovascular and Lymphatic systems

C. STRUCTURE AND FUNCTION OF THE LYMPHATIC SYSTEM

- Fluid that filters out of capillaries into spaces between tissue cells is called interstitial fluid.
- Interstitial fluid enters lymph capillaries and is called lymph.
- Vessels called lymphatics return lymph to the blood near the right side of the heart.
- Lymph nodes contain fixed macrophages (dendritic cells), B cells, and T cells.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• A. Septicemia, Sepsis, and Septic Shock
  – The growth of microorganisms in blood is called septicemia. Signs include lymphangitis (inflamed lymph vessels). *Red streaks, see picture in text.*
  – Septicemia can lead to septic shock, characterized by decreased blood pressure.
  – Septicemia usually results from a focus of infection in the body, e.g. UTI, pneumonia.
  – Gram-negative rods are usually implicated. Endotoxin causes the symptoms associated with a severe drop in blood pressure. 250K deaths/yr.
  – *Staph aureus*, a gram positive cocci, may cause a toxic sepsis especially in dialysis patients.
  – Puerperal sepsis begins as a uterine infection following childbirth or abortion; it can progress to peritonitis or septicemia.
  – *Streptococcus pyogenes* is the most frequent cause.
Lymphangitis, a sign of sepsis. Note the clearly defined inflamed lymph vessels.
Short-term, triple-lumen central venous catheter. The ends from which the catheter is accessed are usually referred to as the hub(s). After the catheter is inserted, the tip will reside within the bloodstream.
Possible routes by which microorganisms gain access to the blood stream.
Blood culture bottles for the BACTEC 9240 continuous monitoring instrument.
The BACTEC – continuous monitoring blood culture system.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• B. Bacterial Infections of the Heart
  – The inner layer of the heart is the endocardium.
  – Subacute bacterial endocarditis is usually caused by alpha-hemolytic streptococci, staphylococci, or enterococci.
    • Develops slowly
    • The infection arises from a focus of infection, such as a tooth extraction.
    • Preexisting heart abnormalities are predisposing factors.
    • Signs include fever, anemia, and heart murmur.
  – Acute bacterial endocarditis is usually caused by Staphylococcus aureus.
    • The bacteria causes rapid destruction of heart valves.
A case of subacute endocarditis showing a diseased mitral valve and associated structures. Symptoms include fever, heart murmur from poor mitral valve function.
Vegetations of bacterial endocarditis. Lighter area indicates vegetations.
Infectious Diseases - Bacterial endocarditis. Widespread skin lesions may sometimes be seen, especially in acute staphylococcal endocarditis. This patient showed numerous ecchymoses of his hands and feet during the course of staphylococcal septicemia, and signs of aortic valve involvement soon appeared.
Hemorrhagic vasculitic lesion of *S. aureus* endocarditis.
Two diagnostically important peripheral manifestations of infective endocarditis are splinter hemorrhages (left) and Osler’s nodes (right). These painful erythematous nodular lesions may be due to deposition of immune complexes in blood vessel walls, but bacteria have been cultured from them in a few instances, suggesting that they may be embolic in nature.
C. Rheumatic Fever – *Streptococcus pyogenes*

– Rheumatic fever is an autoimmune complication of streptococcal infections.
– Rheumatic fever is expressed as arthritis or inflammation of the heart. It can result in permanent heart damage.
– Antibodies against group A beta-hemolytic streptococci (*S. pyogenes*) react with streptococcal antigens deposited in joints or heart valves or cross-react with the heart muscle.
– Immune reaction to group M protein in Strep is the cause.
– Rheumatic fever can follow a streptococcal infection, such as streptococcal sore throat. Streptococci might not be present at the time of rheumatic fever.
– Prompt treatment of streptococcal infections can reduce the incidence of rheumatic fever. Before antibiotics this was a leading killer of children.
– Penicillin is administered as a preventive measure against subsequent streptococcal infections.
Pediatrics – Erythema marginatum – Numerous erythematous rings with pale centers of normal skin are present on the trunk and limbs. Some of these lesions have coalesced to form an irregular and changing pattern. This rash is an occasional pattern of rheumatic fever.
Growth of a β-hemolytic Streptococcus on blood agar showing large zones of clear hemolysis around small transparent colonies.
A subcutaneous nodule (often near a joint) caused by rheumatic fever, an autoimmune complication of some *S. pyogenes* infections.
D. Tularemia – *Francisella tularensis*

- Tularemia is caused by *Francisella tularensis*. The reservoir is small wild mammals, especially rabbits.
- Signs include ulceration at the site of entry, followed by septicemia and pneumonia.
- Humans contract tularemia by handling diseased carcasses, eating undercooked meat of diseased animals, being bitten by certain vectors (such as deer flies), or inhaled. Maybe used as a bio weapon.
- *F. tularensis* is resistant to phagocytosis, so is a problem in chemotherapy.
- Laboratory diagnosis is based on an agglutination test on isolated bacteria.
Tularemia cases reported by county. 1347 cases total.
CDC:2002
Francisella tularensis seen in tissue specimens. A, Section of an infected liver. The capsular surface shows multiple white, stellate areas of necrosis.
Figure 34-14.8,
Gram stain from broth culture of *F. tularensis*.
E. Brucellosis (Undulant Fever) - *Brucella abortus*

- Brucellosis can be caused by *Brucella abortus*, *B. melitensis* (goats), and *B. suis* (swine).
- *B. abortus* most common in US. Get from unpasteurized milk or diseased animal tissue. Mainly in vets, farmers, meat packers. Enters skin, mucous membranes, or GI tract.
- Domesticated animals (cattle, pigs, goats, and camels) and some wild stocks constitute the reservoir.
- The bacteria enter through minute breaks in the mucosa or skin, reproduce in macrophages, and spread via lymphatics to liver, spleen, or bone marrow.
- Signs include malaise and fever that spikes each evening (undulant fever). 104°
- A vaccine for cattle is available.
- Diagnosis is based on serological tests.
Culture of *Brucella melitensis* on sheep's blood agar after 48 hours. Requires specially enriched media and increased CO$_2$. 
Gram stained film of *Brucella abortus* showing short coco-bacillary Gram-negative organisms. These are often morphologically indistinguishable from similar organisms such as *Bordetella*. 
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• F. Anthrax – *Bacillus anthracis*
  – *Bacillus anthracis* causes anthrax. In soil, endospores can survive for up to 60 years.
  – Grazing animals acquire an infection after ingesting the endospores.
  – Human gastrointestinal anthrax (50% mortality) caused by ingestion of endospores has been reported.
  – Humans contract anthrax by handling hides from infected animals. The bacteria enter through cuts in the skin (20% mortality) or through the respiratory tract (100% mortality). Can be used as a bio weapon.
  – Entry through the skin results in a pustule that can progress to septicemia. Entry through the respiratory tract can result in pneumonia and quick death.
  – Diagnosis is based on isolation and identification of the bacteria.
Pustule of cutaneous anthrax. When the pustule breaks open, a depressed ulcerated lesion forms. Note the black eschar (scab) that forms. Anthrax derives from the Latin word for coal.
Infectious diseases - Anthrax. In the Western World this is a rare occupational disease associated with contact with imported hides, wool, and hair. The cutaneous form begins as a small papule and is soon surrounded by vesicles (a blister-like eruption on the skin containing serous fluid). The central area ulcerates and dries to form a black eschar. There is much surrounding edema. Later the eschar (a slough esp. one followed by a cauterization or wound) spreads to cover the previously vesicular area.
Peripheral blood films collected from a cow dying of anthrax. The preparations are stained with methylene blue.
Impression colony of *Bacillus anthracis* stained with methylene blue, demonstrating the "medusa-head" appearance of the colony edge. x12 and x75.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• G. Gangrene – *Clostridium perfringens*
  – Soft tissue death from ischemia (loss of blood supply) is called gangrene.
  – Microorganisms grow on nutrients released from gangrenous cells.
  – Ferments carbohydrates and produces $\text{CO}_2 + \text{H}_2$ gases. Organisms produce exotoxins and enzymes that further interfere with blood supply and favor spread of infection. Spreads area of necrosis and toxemia and death may occur.
  – Gangrene is especially susceptible to the growth of anaerobic bacteria such as *Clostridium perfringens*, the causative agent of gas gangrene.
  – *C. perfringens* can invade the uterine wall during improperly performed abortions.
  – Debridement, hyperbaric chambers, and amputation are used to treat gas gangrene.
Appearance of toes with gangrene caused by Clostridium perfringenes. Preexisting necrotic tissue (black areas), due to poor circulation, lead to anaerobic conditions necessary for *Clostridium* growth.
Clostridial myonecrosis (gas gangrene).
Diabetic foot infection with soft-tissue gas formation.
Hyperbaric chambers are used to treat gas gangrene. Usually available only in major medical centers.
Pure growth of *C. perfringens* on Brucella Agar incubated anaerobically. Double zone of hemolysis. Inner zone complete beta-hemolysis; outer zone of partial hemolysis.
Gram-positive non-sporing bacillus, *Clostridium perfringens* grown from a gas gangrene lesion.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• **Diseases Caused by Bites and Scratches**
  – *Pasteurella multocida*, introduced by the bite of a dog or cat, can cause septicemia.
  – Anaerobic bacteria such as *Clostridium*, *Bacteroides*, and *Fusobacterium* found in the mouth infect deep animal bites (including human bites).
  – Cat-scratch disease is caused by *Bartonella henselae*. Found in up to 50% of all cats.
  – *Initial sign is a reddish purple papule at infection site.*
Human bite infection
Appearance of *Bacteroides fragilis* on a KVLB/BBE agar biplate. Browning of the BBE medium is due to esculin hydrolysis.
Gram-stained appearance of *Bacteroides* sp.
Animal bite caused by *Pasteurella* sp.
Pasteurella multocida colonies on sheep's blood agar.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• I. Vector-Transmitted Diseases
  – 1. Plague - Yersinia pestis
    • Plague is caused by Yersinia pestis. The vector is usually the rat flea (Xenopsylla cheopis).
    • Normally a disease of rats. Transmitted one to another by rat flea. European rats introduced into US many years ago are primary reservoirs. In far West and Southwest is endemic in wild rodents, esp. ground squirrels, prairie dogs & chipmunks. If host dies the rat flea seeks a replacement. Another rodent or human. Plague infected flea needs to feed because growth of bacteria blocks the flea’s digestive tract and the blood the flea ingests is quickly regurgitated. Don’t always need arthropod vector. Contact from the skinning infected animals, scratches of domestic cats and similar incidents have been reported as causing plague.
    • Reservoirs for plague include European rats and North American rodents.
    • Signs of bubonic plague include bruises on the skin and enlarged lymph nodes (buboes).
    • The bacteria can enter the lungs and cause pneumonic plague.
    • Pneumonic form especially dangerous. Highly infective due to airborne droplet transmission. Untreated mortality rate is 50-75% and pneumonia is near 100% Organism can survive inside cells.
    • Laboratory diagnosis is based on isolation and identification of the bacteria.
    • Antibiotics are effective in treating plague, but they must be administered promptly after exposure to the disease.
    • Streptomycin and tetracycline can be used prophylactically in those who have been exposed.
A case of bubonic plague showing bubos (swollen lymph nodes) on the thigh, an indication of systemic infection.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• I. Vector-Transmitted Diseases
  – 2. Relapsing Fever – *Borrelia sp.*
    • *Relapsing fever is caused by Borrelia species and transmitted by soft ticks.*
    • *The reservoir for the disease is rodents.*
    • *Signs include fever, jaundice, and rose-colored spots.*
      *Relapses recur three or four times after apparent recovery due to antigenic variants.*
    • *Laboratory diagnosis is based on the presence of spirochetes in the patient’s blood.*
Appearance of *Borrelia recurrentis* in blood. Giemsa stain. x850 - This may be an epidemic louse-borne human disease caused by *Borrelia recurrentis*. During the febrile phase the organisms are present in the blood, either as tightly coiled helical spirochetes, as shown here or loosely coiled forms.
Vector-Transmitted Diseases

3. Lyme Disease – Borrelia burgdorferi
   - Lyme disease is caused by Borrelia burgdorferi and is transmitted by a tick (Ixodes).
   - Lyme disease is prevalent on the U.S. Atlantic Coast.
   - Ixodes pacificus – Pacific coast
   - Ixodes scapularis – Rest of US
   - Get rash and flu-like symptoms. A skin lesion spreads from the site of the bite, clearing in the center (bull’s eye rash). Later complications are arthritis, and occasionally heart and neurological abnormalities. Similar to late stage syphilis.
   - Field mice provide the animal reservoir.
   - Diagnosis is based on serological tests and clinical symptoms.
   - Can treat with antibiotics.
Lyme disease in the US reported by county for 2000.
CDC, 2002
Life cycle of the tick of Lyme disease, usually involving deer and mice. Humans become accidental hosts.
31 BC.

*Ixodes dammini* - Tick associated with Lyme disease
The annular lesion associated with Lyme Borreliosis. Erythema chronicum migrans. Bull’s Eye Rash
The common bull’s-eye rash of Lyme disease, that appears at the site of the bite.
II. Bacterial Diseases of the Cardiovascular and Lymphatic Systems

• I. Vector-Transmitted Diseases


    • a. Epidemic Typhus

      – The human body louse *Pediculus humanus corporis* transmits *Rickettsia prowazekii* in its feces, which are deposited while the louse is feeding.

      – Epidemic typhus is prevalent in crowded and unsanitary living conditions that allow the proliferation of lice.

      – *Rubbed into wound when host scratches bite*

      – The signs of typhus are rash, prolonged high fever, and stupor.

      – Tetracyclines and chloramphenicol are used in treatment.

    • b. Endemic Murine Typhus

      – Endemic murine typhus is a less severe disease caused by *Rickettsia typhi* and transmitted from rodents to humans by the rat flea.

      – *Actually occurs sporadically rather than in epidemics.*

    • c. Spotted Fevers

      – *Rickettsia rickettsii* is a parasite of ticks (*Dermacentor* species) in the southeastern U.S., Appalachia, and the Rocky Mountain states.

      – The rickettsia may be transmitted to humans, in whom it causes tickborne typhus fever.

      – Chloramphenicol and tetracyclines effectively treat Rocky Mountain spotted fever, or tickborne typhus.

      – Serological tests are used for laboratory diagnosis.

      – In the east, dog ticks are mainly responsible, in Rocky Mountains, wood ticks. The rickettsiae are passed among ticks by transovarian passage, infecting tick eggs as they are produced.
Close-up of wrist and hand of child with Rocky Mountain Spotted Fever.
Infectious Diseases - Rocky Mountain spotted fever (most common) (Rickettsia rickettsii). The rash appears after several days of fever, starting peripherally and spreading centrally to the trunk. It is initially macular but later becomes petechial and purpuric. Sometimes large ecchymoses develop.
Life cycle of the tick vector (Dermacentor spp.) of Rocky Mountain spotted fever. Humans are accidental hosts.
The rash caused by Rocky Mountain spotted fever. Rash can be mistaken for measles. Rash may appear on palms and soles whereas viral rashes do not.
III. VIRAL DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

A. Infectious Mononucleosis - Epstein-Barr virus (EBV).
   - Infectious mononucleosis is caused by the EB virus. Fever, sore throat, swollen lymph glands in neck, general weakness.
   - The virus multiplies in the parotid glands and is present in saliva (kissing disease). It causes the proliferation of atypical lymphocytes in the blood.
   - The disease is transmitted by the ingestion of saliva from infected individuals.
   - Diagnosis is made by an indirect fluorescent-antibody technique.
   - Have to watch out for ruptured spleen as a complication.
A case of Burkitt’s lymphoma, a cancerous tumor caused by Epstein-Barr virus (EBV).
Infectious Mononucleosis - The tonsils are swollen and covered with uniform white exudate. The uvula looks swollen and the patient’s speech is nasal.
Infectious Mononucleosis - Groups of palatal petechiae as seen in this picture are common in infectious mononucleosis, but are not specific for this diagnosis, nor are they always seen even in severe forms of this illness.
Infectious Mononucleosis - Blood film showing atypical lymphocytes. These are larger than normal lymphocytes with a higher ratio of cytoplasm to nucleus. The cytoplasm is basophilic and the nucleus indented or lobulated.
• **Cytomegalovirus (CMV) Inclusion Disease - herpes virus- HHV-5**
  
  – CMV (a herpesvirus- HHV-5) causes intranuclear inclusion bodies and cytomegaly of host cells.
  – CMV is transmitted by saliva, urine, semen, cervical secretions, and human milk.
  – CMV inclusion disease can be asymptomatic, a mild disease, or progressive and fatal. Immunosuppressed patients may develop pneumonia.
  – If the virus crosses the placenta, it can cause congenital infection of the fetus, resulting in impaired mental development, neurological damage, and stillbirth.
  – Babies get CMV – negative blood. Means ab negative. Would be antibody pos for life if have had disease. May be problem with macrophages and T cells. Organism lives inside.
  – Diagnosis is based on isolation of the virus or detection of IgG and IgM antibodies.
Microscopic appearance of distinctive inclusion bodies caused by cells infected by CMV, useful in diagnosis.
Figure 23.20

Typical US prevalence of antibodies against EBV, CMV, and *Toxoplasma gondii* (TOXO) by age.
III. VIRAL DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

c. Classic Viral Hemorrhagic Fevers

(Most hemorrhagic fevers are zoonotic diseases. If medically familiar they are “classic” hemorrhagic fevers.)

1. Yellow fever is caused by an arbovirus (yellow fever virus). The vector is the mosquito *Aedes aegypti*.
   - Endemic in Africa and tropical countries in Central and South America. Monkeys are reservoirs of virus.
   - Signs and symptoms include fever, chills, headache, nausea, and jaundice.
   - Mortality is high. Liver damage.
   - Diagnosis is based on the presence of virus-neutralizing antibodies in the host.
   - No treatment is available, but there is an attenuated, live viral vaccine.
c. Classic Viral Hemorrhagic Fevers

- Dengue is caused by an arbovirus (dengue fever virus) and is transmitted by the mosquito *Aedes aegypti*. There are 4 distinct serotypes of dengue fever. > 100 million cases/yr worldwide.
  - Signs are fever, muscle and joint pain, and rash.
  - Seldom fatal but are painful, giving disease name of “breakbone fever”.
  - Mosquito abatement is necessary to control the disease.
  - Dengue hemorrhagic fever (DHF) occurs when a person is reinfected with a second serotype of dengue virus. This may be rapidly fatal, especially in SE Asian children. Survival of the first type confers immunity to that type but creates a dangerous situation when infected by an other of the 4 types.
D. Emerging Viral Hemorrhagic Fevers

1. Human diseases caused by Marburg, Ebola, and Lassa fever viruses were first noticed in the late 1960s.

   - Marburg and Ebola are the filoviruses. Lassa is an Arenavirus. “Hot Zone” a popular book on these viruses.
   - Marburg virus is found in nonhuman primates; Lassa fever viruses are found in rodents.
   - Rodents are the reservoirs for Argentine and Bolivian hemorrhagic fevers.
Ebola hemorrhagic fever virus. Note the filoform (filamentous) virus appearance.
D. Emerging Viral Hemorrhagic Fevers

2. *Hantavirus* pulmonary syndrome is caused by *Hantavirus*.

- The virus is contracted by inhalation of dried rodent urine, especially in out buildings.
- *Southwest US*. Fatal pulmonary infection causing lungs to fill with fluid. Elsewhere in world are known as hemorrhagic fever with renal syndrome.
IV. PROTOZOAN DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

• **A.** American Trypanosomiasis (Chagas’ Disease) - *Trypanosoma cruzi*
  
  – *Trypanosoma cruzi* causes Chagas’ disease. The reservoir includes many wild animals. The vector is a reduviid, the ‘kissing bug.’ Lives in thatch roofs and cracks of buildings.
  
  – Look for trypanosomes in the intestinal tract of the reduviid bug after feeding on patient’s arm, which confirms the diagnosis.
  
  – *Occurs in southern US and throughout Mexico, Central America, and South America.*
  
  – *Disease is transmitted to humans when insect bites are contaminated by the insect’s feces.*
  
  – *Most damage is caused by inflammatory reactions after transport by blood to the liver, spleen, heart, and so on.*
    
    • One symptom is loss of involuntary muscular contractions in the esophagus and GI tract due to nerve damage. These organs become grossly enlarged: megaesophagus and megacolon.
    
    • The disease is most dangerous to children, in whom it damages the heart.
Life Cycle of *T. cruzii*
Trypanosoma sp. Trypomastigote in a blood smear.
IV. PROTOZOAN DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

B. Toxoplasmosis - *Toxoplasma gondii*

- Toxoplasmosis is caused by the sporozoan *Toxoplasma gondii*.
- *T. gondii* undergoes sexual reproduction in the intestinal tract of domestic cats, and oocysts are eliminated in cat feces.
- Oocysts can be ingested by cattle and other animals.
- Sporozoites hatch from an oocyst and invade host cells.
- In the host cell, sporozoites reproduce to form either tissue-invading tachyzoites or bradyzoites.
- Bradyzoites reproduce in tissue cysts.
- May become chronic as the immune system becomes increasingly effective.
- Host cell develops a wall to form the tissue cyst. Bradyzoites reproduce very slowly. Brady means slow. Can persist for years.
- Humans contract the infection by ingesting tachyzoites or tissue cysts in undercooked meat from an infected animal or contact with cat feces.
- Subclinical infections are probably common because the disease symptoms are rather mild. 40% of population is positive for ab.
- Congenital infections can occur. Signs and symptoms include severe brain damage or vision problems.
- Toxoplasmosis can be identified by serological tests, but interpretation of the results is uncertain.
Life cycle of *Toxoplasma gondii*. Note the cat is the definitive host where the protozoa reproduces sexually.
75 BC

Life cycle of *T. gondii*
51 BC

*Toxoplasma gondii* tachyzoites - Section of Brain
Malaria - Anopheles mosquito - *Plasmodium falciparum* – *P. vivax* – *P. malaria & ovale*

- The signs and symptoms of malaria are chills, fever, vomiting, and headache, which occur at intervals of 2-3 days. 300-500 million infected. 2-4 million killed/yr.
- Malaria is transmitted by *Anopheles* mosquitoes. The causative agent is any one of four species of *Plasmodium*.
- *Plasmodium falciparum* – Most dangerous and geographically widespread. About 20% of cases.
- *P. vivax* – Also widespread. About 80% of cases.
- *P. malaria & ovale* – Lower infection rate, geographically restricted, and milder disease.
- Sporozoites reproduce in the liver and release merozoites into the bloodstream, where they infect red blood cells and produce more merozoites.
- Laboratory diagnosis is based on microscopic observation of merozoites in red blood cells.
- New drugs are being developed as the protozoa develop resistance to drugs such as chloroquine. A vaccine is being developed?
Malaria in the US when cases were commonly endemic as recently as 1912.
Malaria. Left, RBCs lysing and releasing merozoites that will infect other RBCs. Right, Stained blood smear showing early ring stage (vacuole with nucleus) of parasite feeding on RBC contents.
Life cycle of stages of malaria
Plasmodium falciparum ring form trophozoites
Plasmodium falciparum – gametocyte
IV. PROTOZOAN DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

D. Leishmaniasis
   – Visceral, cutaneous, and mucocutaneous clinical forms.
   – About 20 species of protozoans that are transmitted by sandflies (very small).
   – Many cases are being dx in veterans from Mediterranean Gulf Wars.
Cutaneous leishmaniasis lesion on the back of a hand.
V. HELMINTHIC DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

A. Schistosomiasis - *Schistosoma mansoni*

- Species of the blood fluke *Schistosoma* cause schistosomiasis.
- Eggs eliminated with feces hatch into larvae that infect the intermediate host, a snail. Free-swimming cercariae are released from the snail and penetrate the skin of a human.
- The adult flukes live in the veins of the liver or urinary bladder in humans.
- Adult flukes reproduce, and eggs are excreted or remain in the host.
V. HELMINTHIC DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

• A. Schistosomiasis - *Schistosoma mansoni*
  – Granulomas are from the host’s defense to eggs that remain in the body.
  – Causes damage such as abscesses and ulcers to the liver and other organs such as the lungs or urinary system.
  – Observation of eggs or flukes in feces, skin tests, or indirect serological tests may be used for diagnosis.
  – Chemotherapy is used to treat the disease; sanitation and snail eradication are used to prevent it.
Schistosomiasis life cycle, with snail as intermediate host and human as definitive host.
Schistosome adult worms as they appear in the circulation. Note the appearance of the female adult worm inside the curled body of the adult male.

(a) Male and female schistosomes. The female lives in a groove on the ventral surface of the male schistosome ("split-body"), is continuously fertilized, and continuously lays eggs. The sucker is used by the male to attach to the host.

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A granuloma from a patient with schistosomes. Eggs laid in tissue lead to an inflammatory response by the host forming a granuloma.
V. HELMINTHIC DISEASES OF THE CARDIOVASCULAR AND LYMPHATIC SYSTEMS

• B. Swimmer’s Itch
  – Swimmer’s itch is a cutaneous allergic reaction to cercariae that penetrate the skin. The definitive host for this fluke is wildfowl.
  – Larvae does not mature in humans.
Swimmer’s Itch – arm