

Altered Immune System



NUR 143
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Anatomy and Physiology of the Immune System



- Contains lymphocytes and lymphatic tissue
- Effectiveness depends on systems ability to differentiate between “self” and “non self” properties
- Autoimmune disorders occur when immune system fails to recognize itself
- Antibody-mediated immune response
- Cell-mediated immunity
- Declines with aging

Functions of the Immune System



- Provides protection against invasion from outside the body, such as microorganisms
- Protects the body from internal threats
- Maintains the internal environment by removing dead or damaged cells
- Neutralizes, eliminates or destroy penetrating or invading organisms before invaders can multiply and overwhelm body defenses.
- The effectiveness of the immune system depends on whether it can differentiate normal body tissue (self) from foreign tissue (non-self)

Factors Affecting Immune System Functioning



- Lymphocytes redistributed, fewer in lymph nodes
 - ✦ Radiation destroys lymphocytes
- Decreased function of various organs
 - ✦ skin thinner, decreased gastric secretions
 - ✦ End result: increase in infections
- Nutrition:
 - ✦ Decreased protein leads to atrophy of lymphoid tissues and decreased antibody response
 - ✦ High fat diet if prolonged: may cause immunosuppression
- Organ damage or Involvement
 - ✦ Burns or anything impairing skin integrity compromise body's defenses
 - ✦ Serum loss decreases available antibodies
 - ✦ Stress that causes excessive cortisol levels is immunosuppressive

Immunity



- A state of responsiveness to foreign substances and tumor proteins
- Immunity involves defense, surveillance and homeostasis
- Innate immunity- born with white cells, species specific immunity to some organisms
- Acquired immunity- not born with it, results from interaction with antigens

Factors that May Cause Autoimmune Disorders



- Release of “hidden” antigens
- Chemical, physical, or biologic changes in host tissue
- An antigen whose properties closely resemble those of host tissue
- Defect in normal cellular immune functions
- Slow growing mycobacteria

Pharmacologic and Collaborative Therapies



- **Immunotherapy: desensitization**
- **Antihistamines: used in type 1 responses**
- **Epinephrine: anaphylaxis**
- **Corticosteroids: used for anti inflammatory effects**
- **Plasmapheresis: removes harmful components from the plasma**

Active Immunity



Natural active immunity

- Innate Immunity
- Present at Birth
- Includes physical and chemical barriers to invading antigens

Active Acquired Immunity

- Long term immunity
- Established when vaccinations are administered and the vaccine stimulates antibodies and memory cells.
- Occurs when, with subsequent exposure, the antibodies and memory cells specific for that antigen swiftly produce even more antibodies.
- Antibodies neutralize future invasions of the same antigen
- Length of active immunity depends on the disease or vaccination

Passive Acquired Immunity



- Temporary Immunity
- Occurs when a person is given antibodies from another source.
- Antibodies obtained from an animal or another human who has been exposed to an antigen
 - ✦ Example: antibodies passed by the mother to the fetus by way of the placenta or to a baby via breast milk
- Examples are gamma globulin or anti-serum

Humoral Immunity-



- Antibody mediated immunity, stem cells in bone marrow differentiate into B cells, daughter cells produce antibodies to bind to antigens
- Cell mediated immunity- cells from bone marrow differentiate into T cells while in thymus
 - Primary response: during first exposure to an antigen, B- cells are stimulated to become plasma cells and produce antibodies or memory cells
 - Secondary response: with re-exposure to the same antigen, the memory cells respond rapidly with antibody production
- Unlike a localized inflammatory response the immune response is systemic
 - The immune response has a memory

Humoral Immunity



- Stem cell creates various types of B cells, each type capable of responding to a certain antigen
 - Antigen presentation triggers multiple clones of a certain B cell type
 - Daughter B cells, called plasma cells, produce antibodies capable of binding certain antigens
- This leads to destruction of antigen
 - Some B cells become memory cells capable of creating clones in the future if this antigen some reappear

Immune System



- All lymphocytes are leukocytes
- Some lymphocytes are cytotoxic
- Some leukocytes are phagocytic
- Some phagocytes are granulocytes
- Monocytes can turn into macrophages and are then phagocytic but not granulocytes
- Basophils can turn into mast cells and are neither phagocytes nor granulocytes
- Monocytes and basophils are leukocytes but not lymphocytes
- Some lymphocytes make antibodies, but antibodies are not cells
- Now what's a lonely Eosinophils to do?

Cellular Players



- **Neutrophils- first phagocytes to arrive at injury**
 - 90 min to 10 hours
- **Monocytes-blood**
 - Macrophages-tissue- second phagocyte
 - 5 hours to arrive but long lived
- **Basophils: blood/Mast cells-tissue-**
 - granulocytes full of histamine & serotonin
- **Eosinophils- enzymes toxic to parasites**
 - degrade vasoactive amines, involved in allergic responses

Cell Mediated Immunity



- **T Cytotoxic cells-** most attack antigens in a specific fashion using cytolytic substances, others serve as memory cells
- **Natural Killer Cells-** granulocytic lymphocytes attacking viruses, tumor cells, and grafts
- **T Helper Cells (CD4)-** bind to macrophage/antigen
- **T Suppressor Cells (CD8)-** prevent too much CD4

T-Cells



- **Helper T Cells (CD4 + Cells)**
 - Initiate the immune system response, including allergic responses
- **Suppressor T Cells**
 - Turn off the immune response
 - Are important in autoimmune disorders
 - Prevent the body from self-destruction
- **Cytotoxic T Cells**
 - Kill tumor, viral, and foreign cells

Inflammation



- Reaction of vascularized tissue to local injury
- Inflammation= 'itis'
- Manifestations-
 - redness, warmth, pain, swelling, loss of function
- Process & Purpose-
 - begins at time of injury
 - prepares wound for healing
 - blood vessels constrict then dilate
 - increased capillary permeability
 - blood cells and substances move into interstitial area

Aspects of Inflammation



- **Activation-** basophils and mast cells degranulate to release histamine and serotonin, this causes vasodilation- increased vascular permeability- smooth muscle contraction
- **Vascular response-** immediate vasoconstriction followed by vasodilation, increase in extravascular fluid

Vasoactive Substances



- **Histamine-** vasodilation, increased vascular permeability, smooth muscle contraction
- **Serotonin-** similar actions to histamine
- **Bradykinin-** similar to histamine plus produces pain
- **Leukotrienes-** similar to histamine plus attract Eosinophils and neutrophils to area
- **1st two found in cells, 2nd two are circulating plasma proteins**

Antibodies



- Are large protein structures
- They bind to antigens creating an antibody-antigen complex
- Makes it more vulnerable to attack
- Antigen is eliminated by phagocytes and by membrane attack complex (in conjunction with complement)

Antibody Types



- **IgM-** responsible for primary response
- **IgG-** responsible for secondary response, almost 80% of total, crosses placenta, activates complement
- **IgA-** in mucus membranes & skin
- **IgD-** assists in B cell differentiation
- **IgE-** fixes to mast cells and basophils, part of allergic reactions, defends against parasites

Immunoglobulin Characteristics and Functions



TABLE 11-2

Immunoglobulin Characteristics and Functions

CLASS	PERCENTAGE OF TOTAL	CHARACTERISTICS AND FUNCTION
IgG	75%	Found In Intravascular and Interstitial compartments. Most abundant Ig; also known as gamma globulin. Active against bacteria, bacterial toxins, and viruses. Crosses placenta providing Immune protection to neonate.
IgA	10–15%	Found In saliva, sweat, tears, mucus, bile, colostrum, and vaginal secretions. Provides local protection to prevent entry of bacteria and viruses especially through the respiratory and gastrointestinal tracts.
IgM	5–10%	High concentrations In the blood; less In the lymph. First antibody formed and lasts about 1 week. Reacts effectively against bloodborne bacteria and viruses.
IgD	1%	Found on surface of B cells. Exact function unknown; may bind antigens to B-cell surface.
IgE	<1%	Found on mast cells and basophils. Stimulates release of chemical mediators such as histamine and bradykinin that cause allergic reactions and hypersensitivity responses such as anaphylaxis. Acts In preventing helminth (worm) infections.

Results of Aging



- **Immune system**
 - Increased risk of infection
 - Increased risk of cancers
 - Altered response to antigens
 - Possible reactivation of TB

Hypersensitivity Reactions



- Hypersensitivity reactions occur as the body becomes confused identifying self from non-self
- An allergy is an abnormal, individual response to certain substances that normally do not trigger such an exaggerated reaction.
- In all types of allergies, a reaction occurs only on second and subsequent contacts with the allergen.
- Skin testing may be done to determine the allergen.



- **Type I: IgE-Mediated Hypersensitivity**
 - Allergic asthma
 - Allergic rhinitis (hay fever)
 - Anaphylactic shock
 - **5-10% of children have Type I type of response, usually a family history**
 - **Triggered by hypersensitivity reaction to airborne allergens**
- **Type II: Cytotoxic Hypersensitivity**
 - Blood transfusion reaction

Hypersensitivity Reactions



- **Type III- Immune Complex reactions- IgG & IgM antibodies form antibody-antigen complexes which are large and precipitate out of solution, this clogs up small vessels and nephrons, chemotactic substances may destroy adjacent tissue**
 - 6-12 hours, e.g.. Lupus
- **Type IV- Cell Mediated-Delayed reactions.**
 - T Lymphocytes involved, increased vascular permeability & fibrin deposition, 1-3 days,
 - e.g. TB test, poison ivy, transplant rejection

Type IV Delayed Hypersensitivity



Hypersensitivity to Latex



- The chemicals used in processing the latex cause the reaction.
- Client can have a Level I reaction (within minutes) or a Level IV reaction
 - ✦ delayed hypersensitivity
- Symptoms may be as mild as redness, hives, and itching.
- Symptoms may be severe, including local symptoms and systemic signs
 - ✦ laryngospasm, wheezing, cardiac arrest, or anaphylaxis

Treatment of Hypersensitivity Reactions



- When medicating the client with hypersensitivity, be attentive to the possibility of an allergic response.
- Report symptoms of redness, hives, or respiratory distress immediately to the physician.
- A second exposure increases the severity of a reaction. Treatment is implemented promptly.
- An anaphylaxis kit may be prescribed for the client to use when needed.
- Discharge instruction must be reinforced with the client and family.

Treatment of Allergies



- Advise clients to identify offending allergens and try to avoid contact with them
- Some medications can offer relief
 - ✦ Antihistamines block histamine receptors, alleviating runny nose (rhinitis), itchy eyes, hives, and other symptoms
 - ✦ Decongestants help open air passages
 - ✦ Overuse of decongestant nasal sprays can result in rebound congestion
 - ✦ Corticosteroids are used systemically as well as topically for allergic responses

Anaphylaxis



- A serious and dramatic allergic reaction with the release of histamine from the damaged cells
- Can cause shock and death if not treated immediately
- Any medication has the potential: penicillin, insulin, muscle relaxants,
- Causes: X-ray dyes
 - Bees, wasps, hornets and snake bites
 - foods (shellfish, eggs, meats, berries, peanuts) blood transfusions and vaccines
 - It can occur in individuals who are extremely sensitive to an allergen.
- Symptoms develop suddenly and can progress to severe levels within minutes.

Early Clinical Manifestations



- **Initial S/S: occurring within seconds to minutes after exposure to allergen**
 - Generalized feeling of warmth, apprehension, uneasiness
 - Weakness, feeling of impending doom
 - Generalized pruritus and urticaria, Itching of palms, soles, eyes, ears
 - Hoarseness
 - Constrictions of throat or feels like a lump in the throat
 - May experience stridor (crowing sound) or may wheeze
 - Hypotensive, rapid, weak, irregular pulse – due to vasodilatation an increased capillary permeability and leakage of intravascular fluids
 - Dysrhythmias, shock and cardiac arrest can occur within minutes as intravascular volume is lost
 - Death is usually result from respiratory failure from bronchospasm, laryngeal edema and circulatory collapse

Treatment



- **Maintain airway – may need to intubate, CPR is necessary**
- **Give sq epinephrine 1:1000 – up to .5 ml**
- **May give IM Benadryl or IV**
- **Start IV if not in place**
- **Oxygen**
- **Have ET and Trach ready**
- **May need to repeat epinephrine every 10-20 minutes**
- **Identification of allergies**
- **Instruct the client to wear a**
 - **Medic-Alert bracelet.**
 - **In the use of prescribed medication for immediate treatment of a reaction.**

Autoimmunity



- Inappropriate T Cell or B Cell response to self-proteins
- Probably genetic predisposition
- Predisposition triggered by infection, hormones, drugs
- Autoimmune destruction of tissues leads to signs & symptoms
 - i.e.- SLE, R. Arthritis, DM-1, MS etc.
 - Tx- Remove the auto-antibodies (Plasmapheresis)

Histocompatibility



- A series of genes which determine
 - (among other things) cell membrane characteristics
- Histocompatibility: matching is key is matching donor and recipient for tissue transplant
 - These genes may also be associated with development of specific diseases
- Human leukocyte antigens (HLA)
 - A persons HLA characteristics are coded within a large cluster of genes known as major histocompatibility (MHC)
 - The possibility of two persons having the same HLA are rare except in identical twins
 - Some siblings may have similar HLA
 - In organ transplants matching the HLA as closely as possible tends to decrease rejection

Immunodeficiency Disorders



- Impairment of one or more of the components of the immune system
- Primary- improper development of part of immune system
- Primary includes graft vs. Host & many you never heard of, rare & serious
 - Secondary- immune deficiency caused by some other illness
 - Secondary includes HIV, Diabetes, Age, Stress, and many induced by medical treatment

Rheumatoid Arthritis



- Chronic systemic inflammatory disease; the cause is unknown
- Leads to destruction of connective tissue and synovial membrane within joints
- Weakens and leads to dislocation of the joint and permanent deformity
- Formation of pannus occurs at the junction of synovial tissue and articular cartilage projecting into the joint cavity and causing necrosis

Clinical Manifestations



- Inflammation, tenderness, and stiffness of the joints
- Moderate to severe pain and morning stiffness lasting longer than 30 minutes
- Joint deformities, muscle atrophy, and decreased range of motion
- Spongy, soft feeling in joints
- Low-grade temperature, fatigue, and weakness
- Anorexia, weight loss, and anemia
- Elevated sedimentation rate and positive rheumatoid factor

Rheumatoid Arthritis



- **Systemic Manifestations**

- Fatigue
- Anorexia
- Weight loss
- Nonspecific aching and stiffness
- Fever
- Rheumatoid nodules

- **Joint Manifestations**

- Swelling
- Stiffness
- Warmth
- Tenderness
- Pain
- “Boggy” feel
- Limitation of range of motion
- Bony deformities

Late Clinical Manifestations



- Joint deformities (swan neck or ulnar deviation)
- Mod. To severe pain and morning stiffness
- Osteoporosis
- Severe fatigue
- Anemia
- Weight loss
- Subcutaneous nodules
- Peripheral neuropathy
- Vasculitis – if blood vessels involved, the organ supplied by that vessel can be affected, have malfunction and eventual failure
- Pericarditis
- Fibrotic lung disease
- Sjogren's syndrome
- Renal disease

Rheumatoid Arthritis



- **Laboratory Tests**
 - Rheumatoid factors (RFs)
 - ESR
 - Antibodies to cyclic citrullinated peptide (CCP)
- **Nursing Diagnoses**
 - Chronic pain
 - Fatigue
 - Disturbed body image
 - Ineffective role performance

Rheumatoid Arthritis



- **Goals of Care**
 - Reduce pain and inflammation
 - Preserve function
 - Prevent deformity
- **Treatment**
 - Balanced program of rest and exercise
 - Physical and occupational therapy referral
 - Heat and cold application
 - Use of assistive devices
 - Plasmapheresis
 - Total lymphoid irradiation

Rheumatoid Arthritis

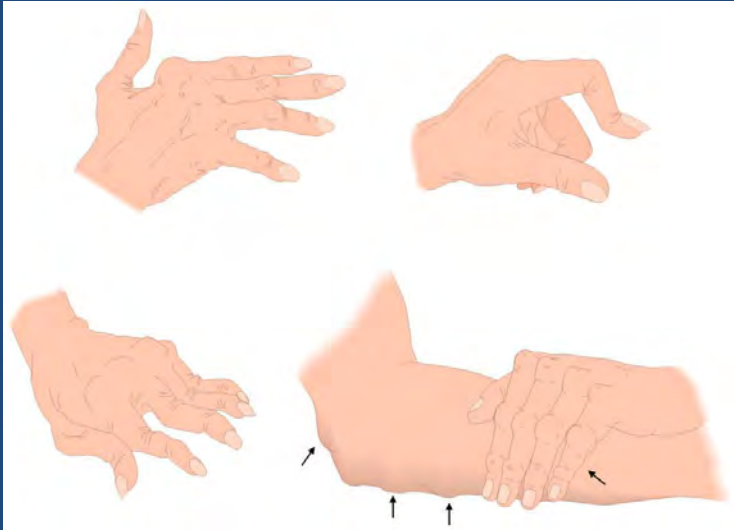


Figure 21-6B

Common joint deformities



Medical Management



- **Surgery**
 - Done when drug and non drug measures are not able to manage pain
 - Most common is synovectomy for repair of damaged tendons of hand to release nerve entrapments
- **Nonsurgical**
 - Drug therapy
 - Rest
 - Positioning
 - Ice
 - Heat
 - Alternate therapies

Juvenile Rheumatoid Arthritis (JRA)



- **An autoimmune inflammatory disease affecting the joints**
- **The cause is unknown**
- **Juvenile onset is diagnosed before 16 years of age**
- **A leading cause of disability in children**
- **Treatment is supportive and directed toward preserving joint function, controlling inflammation, minimizing deformity, and reducing the impact that the disease may have on the development of the child**

JA - Types



- **Systemic**
 - Presence of recurrent intermittent high fever for 3-4 weeks
 - Rash – pale red, non pruritic, trunk and extremities
 - Joint manifestations eventually in 5 or more joints
- **Polyarticular**
 - Presence of arthritis in more than 4 joints
 - Fever, anemia, fatigue, anorexia
 - Rheumatoid factor is positive
 - Resembles adult RA and more likely to become chronic
- **Pauciarticular JRA – most common form**
 - Presence of arthritis in fewer than 5 joints – knees most common
 - Best prognosis, except may develop uveitis
 - ✧ (iris inflammation and eyesight problems)

Therapeutic Management



- **Preserving joint function**
 - maintain mobility with activities that don't strain inflamed joints
 - Heat or cold warm bath in am or warm water soaks for 20-30 int. before activity - decreases pain and inflammation
- **Controlling inflammatory process**
 - Drug therapy basically the same as for adults
 - Teach to manage medication therapy
 - Watch for side effects
 - Reduce fatigue
- **Minimizing deformity**
 - Splints maintain position of function – should not be worn past period of inflammation or may cause contracture
- **Reducing impact of the disease on child's development**
 - Rest – school day may need to be shorten
 - Support groups

Lupus Erythematosus



- Lupus can be categorized into three groups:
 - discoid lupus erythematosus
 - systemic lupus erythematosus
 - drug-induced systemic lupus erythematosus
- Systemic lupus erythematosus (SLE, or lupus)
 - A chronic progressive systemic inflammatory disease that can cause major organs and systems to fail.
 - Connective tissue and fibrin deposits in blood vessels on collagen fibers and on organs.
 - Leads to necrosis and/or inflammation in blood vessels, lymph nodes, GI tract, and pleura.
 - There is no cure for the disease.

Causes



- The cause is unknown and it is thought to be due to a defect in the immunological mechanisms or from genetic origin.
- Precipitating factors include medications, stress, genetic factors, sunlight or ultraviolet light, and pregnancy.
- The Lupus Foundation of America estimates that approximately 1,400,000 Americans have a form of lupus.
- Although lupus can strike men and women of all ages, 90% of individuals diagnosed with the disease are women, and 80% of those afflicted with systemic lupus develop it between the ages of 15 and 45.
- Lupus is two to three times more prevalent among people of color, including African-Americans, Hispanics, Asians, and Native-Americans.

The Butterfly Rash of Systemic Lupus Erythematosus



Symptoms and Occurrence



- **Achy joints 95%**
- **Fever more than**
 - 100 degrees F
 - 38 degrees C 90%
- **Arthritis (swollen joints) 90%**
- **Pain in the chest on deep breathing (pleurisy) 45%**
- **Prolonged or extreme fatigue 81%**
- **Skin Rashes 74%**
- **Anemia 71%**
- **Kidney Involvement 50%**

ACR Criteria for Diagnosing SLE



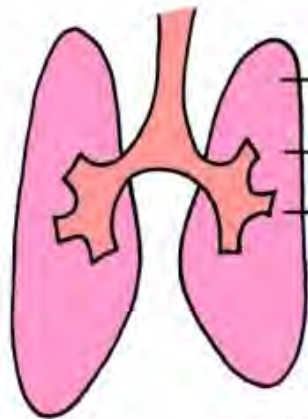
- Malar rash
- Discoid rash
- Photosensitivity
- Oral ulcers
- Arthritis
- Pleuritis or Pericarditis)
- Renal disorder (persistent Proteinuria or cellular casts)
- Neurological disorder (seizures or psychosis)
- If at least 4 of the 11 criteria develop at one time or individually over any period of observation, then the patient is likely to have SLE.

SLE Treatment



- **ASA and NSAIDs**
- **Antimalarial drugs**
- **High dose corticosteroid therapy**
- **Immunosuppressive agents**

SYSTEMIC LUPUS ERYTHEMATOSUS (SLE)



Tachypnea

Cough

Pleural Inflammation/
Effusion

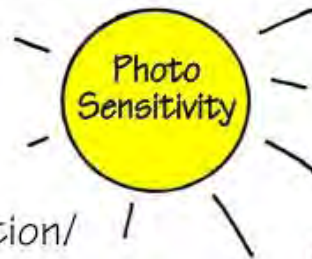
- Weight Loss
- Fatigue
- Fever ↑ Infection
- Arthritis
- Emotional Lability
- Hematologic Disorders
- Neurologic Disorders



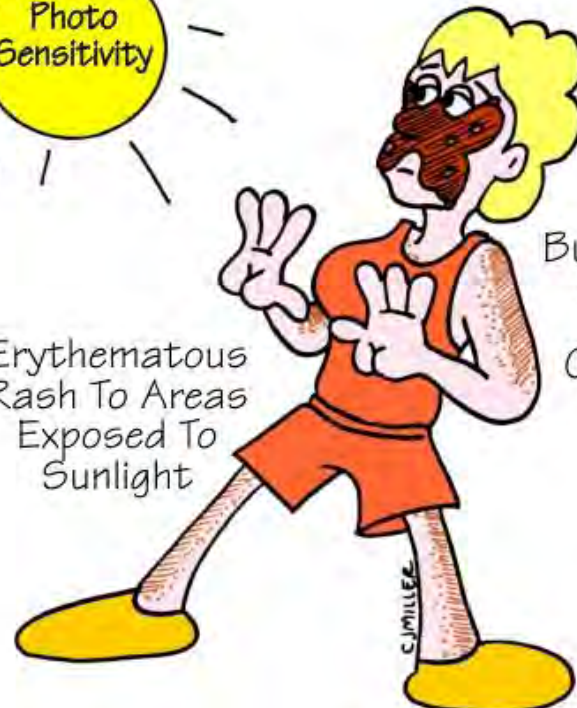
Raynaud's
Phenomenon

Pericarditis

Vascular
Inflammation



Erythematous
Rash To Areas
Exposed To
Sunlight



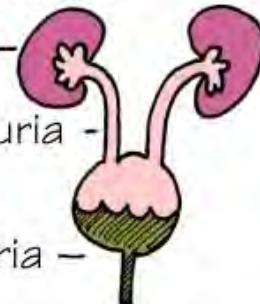
Butterfly
Rash
Over
Cheeks

C.J. MILLER

Lupus Nephritis -

Proteinuria -

Hematuria -



Nursing Interventions



- **Monitor skin integrity and provide frequent oral care.**
- **Instruct the client to clean skin with a mild soap, avoiding harsh and perfumed substances.**
- **Assist with the use of ointments and creams for the rash as prescribed.**
- **Identify factors contributing to fatigue.**
- **Administer iron, folic acid, or vitamin supplements as prescribed if anemia occurs.**

Nursing Diagnosis



- **Skin Integrity Impaired r/t presence of rash and skin lesions and or oral lesions.**
- **Knowledge deficit r/t adapting life style and treatment and prevention of complications**
- **Administer topical or systemic corticosteroids, salicylates and NSAIDs as prescribed for pain and inflammation**
- **Monitor for bruising , bleeding, and injury.**
- **Provide emotional support and encourage the client to verbalize feelings.**
- **Provide information regarding support groups and encourage use of community resources.**

HIV as a Type of Immunodeficiency



- HIV is an RNA retrovirus – must replicate inside a human cell
- HIV gains access to the inside of the cell by binding to CD4 receptors
- CD4 receptors are found on lymphocytes, monocytes/macrophages,

Etiology & Epidemiology



- First recognized as clinical syndrome in 1981, but studies of a blood sample saved from a man who died in the Congo in 1959 were positive for HIV—making him first confirmed case
- HIV probably present in U.S. in '60s if not earlier
- HIV--a human specific retrovirus
 - (has only RNA as genetic material)
- Two subtypes: HIV-1 in western hemisphere, Europe, and Asia;
 - HIV-2 is endemic to west Africa

Background



- **1981- Public health officials document a new immune deficiency disease**
- **1985- The viral cause of AIDS is identified**
- **1985- Antibody tests to detect HIV are developed**
- **1987- First anti-viral agent (Retrovir)**
- **1994- Begin to measure viral load**
- **2007- Combination drug therapy versus increasing HIV drug resistance**
- **2007- Worldwide the epidemic continues . . .**

Epidemiology



- By 2001 over 816,000 cases of AIDS diagnosed in N. American and over 467,000 deaths
- 45,000 new infections yearly
- Women & teens increasingly infected
- At risk- poverty, rural, people of color, people who live with violence, those marginalized
- Worldwide- 42 cases of HIV infection; 3.2 million children

Percent of population
ages 15–49 with
HIV/AIDS, 2005/2006

WORLD	0.9
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More developed regions	0.5
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Less developed regions	1.1
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Africa	4.5
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North America	0.6
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Latin America/Caribbean	0.5
--------------------------------	------------

Europe	0.5
--------	-----

Oceania	0.4
---------	-----

Asia	0.2
------	-----

The HIV Virus



- A retrovirus = replicates in a backwards fashion i.e. from RNA to DNA
- Access to host's DNA requires entry thru CD4 receptors on cell surface
- Reverse Transcriptase produces a single strand DNA, this is copied into a double strand
- Integrase allows the DNA to be spliced into host's genome (this means all daughter cells infected & cell can manufacture viral RNA)

HIV as Immunodeficiency



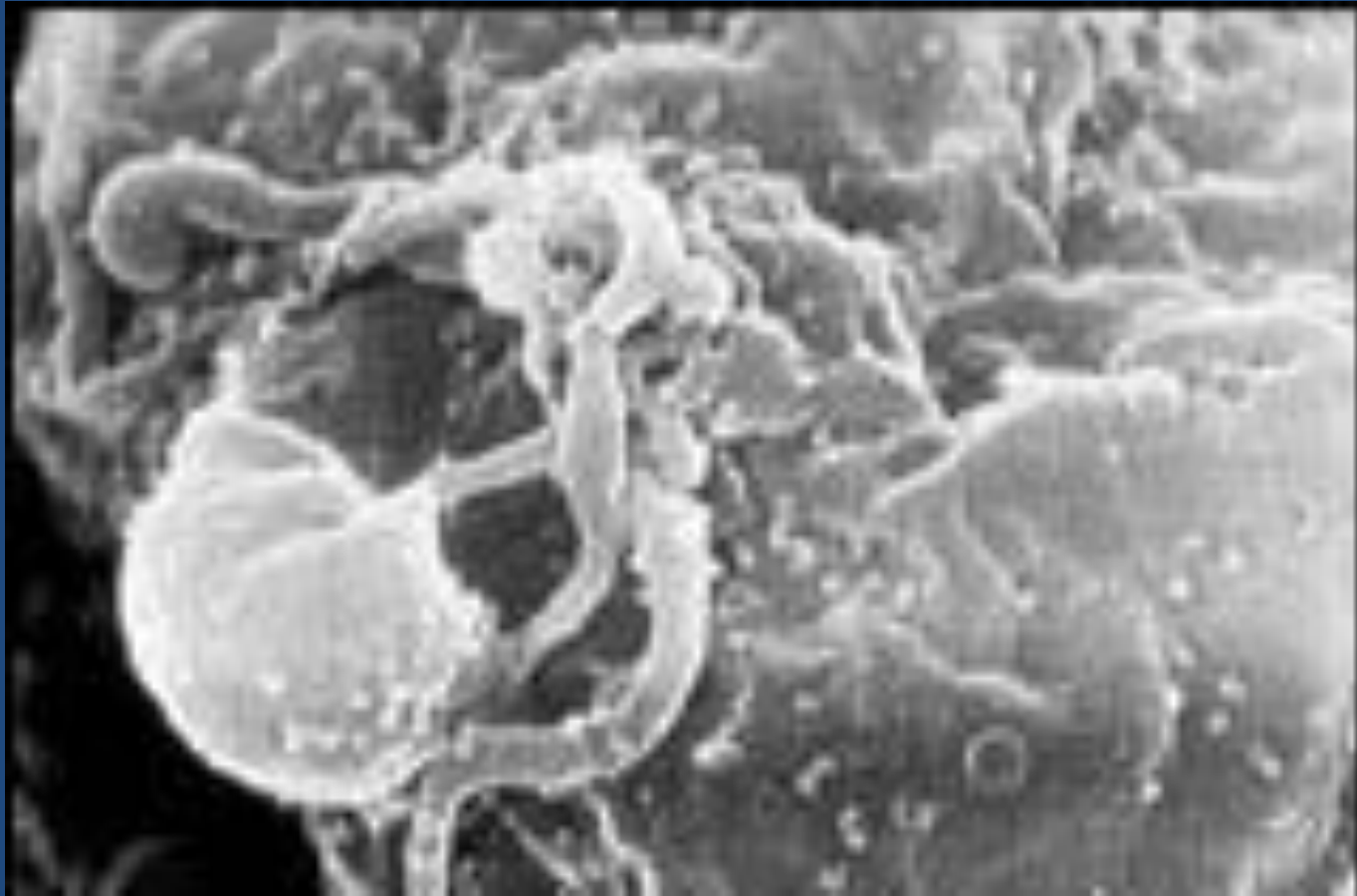
- Initial infection- requires sufficient quantity of virus, repeated exposure, or susceptible individual
- Viremia- initial large quantity of virus in blood followed by ?10 years of low quantities
 - but virus replicating by the billions in cells/lymph nodes
- CD4 Cells are particularly affected as they have so many CD4 receptors
 - can destroy a billion CD4 cells/day
- The body races to make more CD4 cells but eventually destruction exceeds creation

HIV as Immunodeficiency

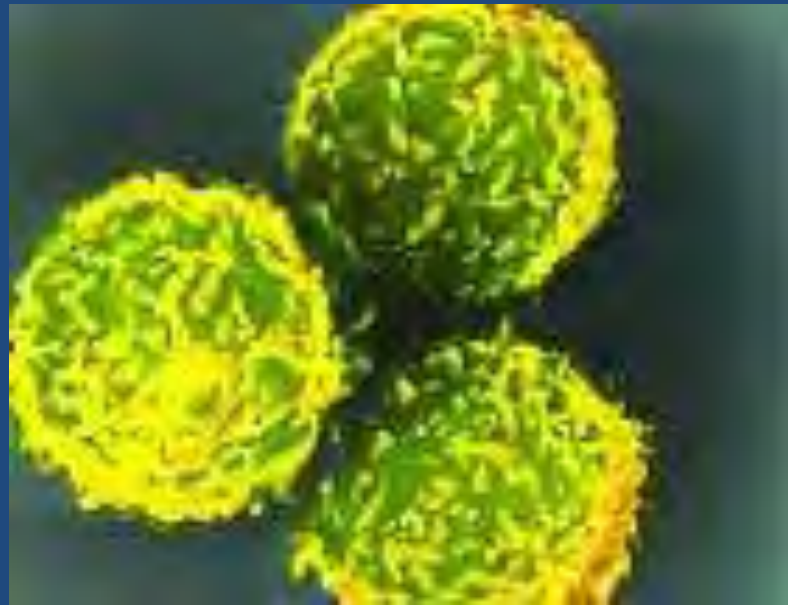


- As HIV replicates mutations occur
 - the virus spills from cells and lymph into blood (levels rise)
- HIV kills CD4 Cells by putting holes in cell membrane, by fusing many cells together, bodies own immune system attacks infected cells
- HIV also kills phagocytes and uses macrophages to spread itself
- Although HIV infects a variety of cells, its main target is the T4-lymphocyte (also called the "T-helper cell"),
 - a kind of white blood cell that has lots of CD4 receptors.
 - The T4-cell is responsible for warning your immune system that there are invaders in the system.
- Replication: Once HIV binds to a cell, it hides HIV DNA inside the cell's DNA: this turns the cell into a sort of HIV factory.

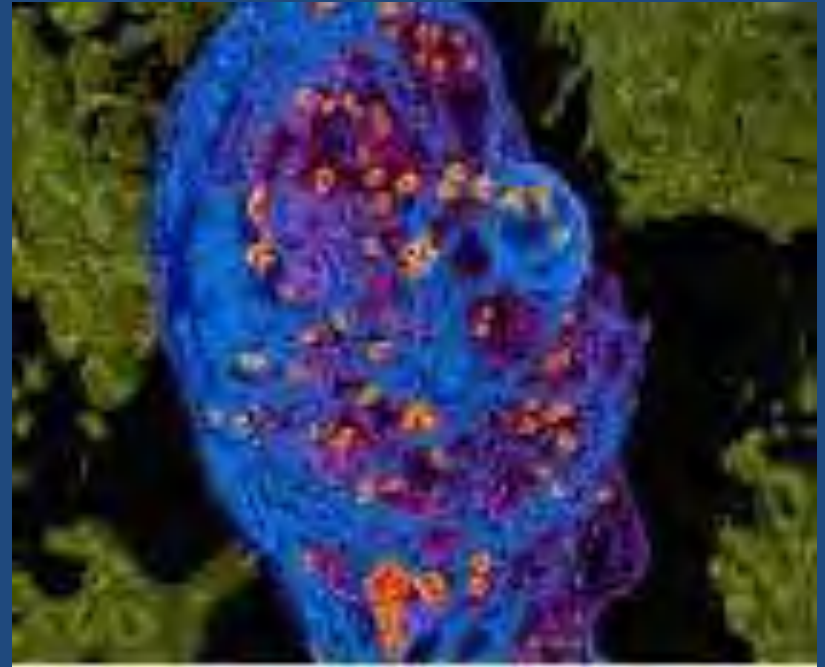
What is a Retrovirus?



<http://www.cnn.com/2002/HEALTH/conditions/07/10/aids.costs/index.html>

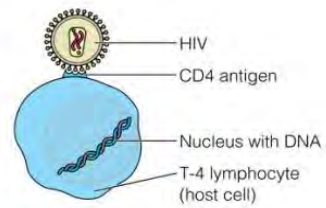


T Lymphocyte



AIDS virus attacking T Lymphocyte

Virus invades cell with CD4 antigen



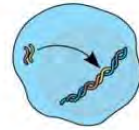
Virus sheds protein coat



Viral RNA converted with reverse transcriptase to viral DNA



Viral DNA integrates with host cell DNA



Virus remains latent



Virus infects daughter cells during host replication



Virus actively replicates, causing lysis of host cell as virus seeks to invade other cells



HIV Transmission

- Transmission possible within a few days of infection
- Ability to transmit is lifelong
- General factors- sufficient quantity, duration of exposure, susceptibility of host, virulence of organism (subtypes)
- Easiest transmission is during early infection (first 2-6 months) or late stage of illness
 - times of high viral load

HIV Transmission

- Casual contact with HIV infected individual does not lead to transmission- hugging, dry kissing, hand shake, toilet seat, coughing, sharing work/school space
- Not transmitted- tears, saliva, urine, emesis, sweat, feces, sputum
- Sexual transmission- semen, vaginal secretions, blood, receptive partner, menses, presence of genital lesions

AIDS

“How You Don’t Catch It”



AIDS: PREVENTION OF TRANSMISSION IN THE HEALTH CARE SETTING

* Maintain Standard Precautions:

- Hand Washing/Hygiene
- Protective Barriers
 - Gloves
 - Mask
 - Eye Shield
 - Gown

* Do NOT Recap Needles & Syringes.

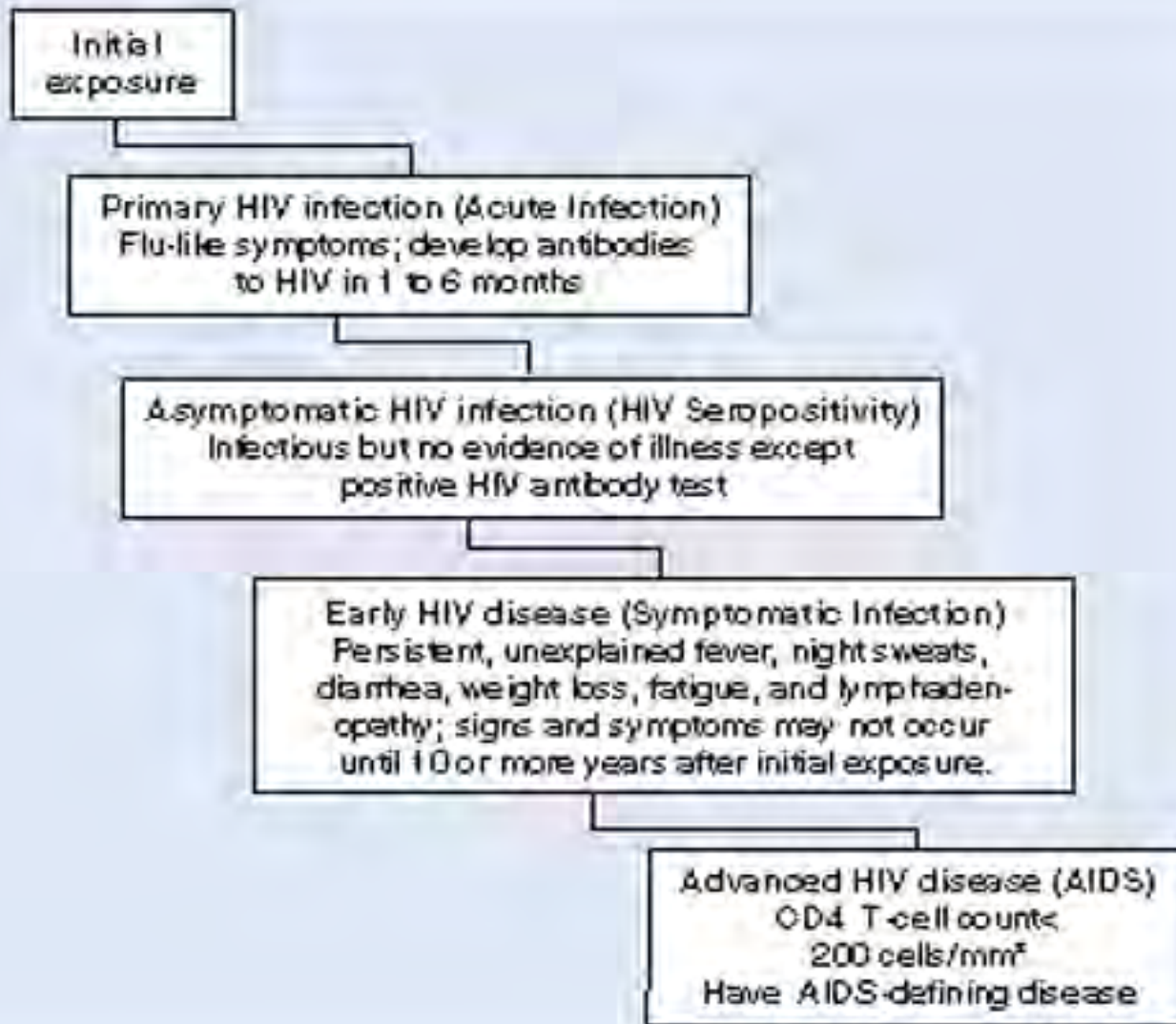
* Clean Up Spills of Blood & Body Fluids Immediately Using Germicidal Solution.

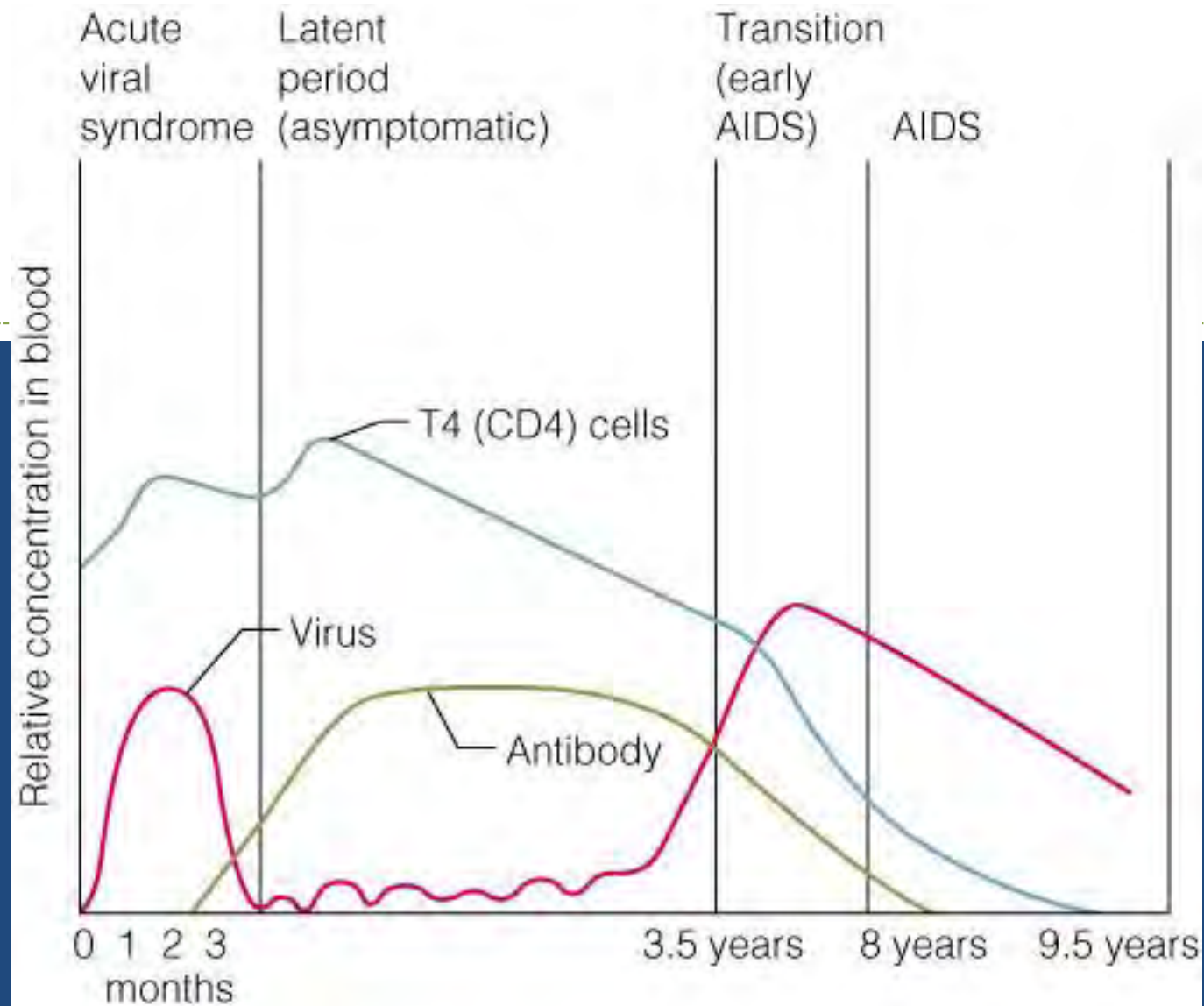


* Consider ALL Body Fluids to be Contaminated.

* Avoid Contaminating the Outside of Specimen Containers During Collection.

* Cleanse Work Surface Areas with Appropriate Germicide.
(1:10 Concentration of Household Bleach)
is Effective





HIV as Immunodeficiency



- **Acute infection-** flu-like syndrome 1-3 weeks after infection
- **Early Chronic Infection-** low viral load, normal CD4 count, more vague flu symptoms, ?10 years
- **Intermediate Chronic-** CD4 count drops, viral load rises, symptoms increase, opportunistic infections
- **Late Chronic (AIDS)-** very low CD4 count, opportunistic diseases, malignancies, dementia

Progression from HIV to AIDS



- **Ranges from months to years**
 - ✦ More rapid for people who are infected by transfusion
 - ✦ Less rapid for those infected from single sexual encounter
 - ✦ More rapid for those frequently re-exposed to HIV
 - ✦ More rapid for those with other sexually transmitted illnesses
- **Progression**
 - **Individual infected: Seropositive**
 - develops antibodies in 21 days and has acute (flu-like) symptoms that resolve in 2-3 weeks
 - **“Window” : infection to detectable antibodies**
 - is usually 3 weeks to 3 months
 - 99% seroconvert within 6 months
 - **Asymptomatic for months to years**
 - length influenced by frequency of re-exposure to HIV, presence of other STDs, nutrition, pregnancy, stress

When is HIV a case of AIDS?



- +HIV infection, and
- CD4 count below 200
- Or, opportunistic infection
- Or, opportunistic cancer
- Or, wasting disease (loss of 10% or more body mass)
- Or, dementia

Diagnosis



- Screening- test for HIV specific AB (enzyme immunoassay- EIA), delay before + 2 months
- If EIA + then repeat the EIA
- If still positive do an immunofluorescence assay- IFA (Western Blot)
- Neonates of HIV + mothers will have mother's HIV AB for 18 M. so need to do DNA PCR or viral culture

HIV Risk Assessment



- Blood transfusion prior to 1985
- Sexual contact – just about any
- Shared needles
- + STD HX

Manifestations of HIV/AIDS



Opportunistic Infections



- Occurrence of OIs and conditions based on decreasing CD4+ count.
- CD4+ count of >500:
 - Recurrent vaginal candidiasis, lymphadenopathy.
- CD4+ count of 200-500:
 - Herpes zoster, oral candidiasis, anemia, Kaposi sarcoma, non-Hodgkin's lymphoma.
- CD4+ count of 100-200:
 - *Pneumocystis Carinii* pneumonia, AIDS dementia complex, AIDS related wasting.
- CD4+ count of <50:
 - CMV retinitis, MAC, Cryptosporidiosis, primary multifocal encephalopathy, primary CNS lymphoma.

Manifestations of HIV/AIDS



- **Early Clinical Manifestations of HIV/AIDS**

- Flu and mononucleosis type symptoms
- Persistent generalized lymphadenopathy
- General malaise, fever, fatigue, night sweats, involuntary weight loss

- **Later Clinical Manifestations of HIV/AIDS**

- Dementia and neurologic effects
- Opportunistic infections
- Pneumocystis carinii pneumonia
- Tuberculosis
- Candidiasis
- Wasting syndrome
- Kaposi's sarcoma
- Lymphoma

CLINICAL MANIFESTATION OF AIDS

IMMUNOLOGIC:

- Low white cell counts
CDT₄ count < 200/mm³
- Opportunistic Infections
- Lymphadenopathy
- Fatigue

INTEGUMENTARY:

- Poor Wound Healing
- Skin Lesions
- Night Sweats

RESPIRATORY:

- Cough
- SOB

GASTROINTESTINAL:

- Diarrhea
- Weight Loss
- Nausea/Vomiting

CENTRAL NERVOUS SYSTEM:

- Confusion
- Dementia
- Headache
- Visual Changes
- Personality Changes
- Pain
- Seizures



OPPORTUNISTIC INFECTIONS:

Protozoal Infections

- Pneumocystis Carinii
Pneumonia
- Toxoplasmosis (Encephalitis)
- Cryptosporidiosis (GI)

Fungal Infections

- Candidiasis - Stomatitis
Esophagitis
Vaginal

Bacterial Infections

- Mycobacterium Complex
- Tuberculosis

Viral Infections

- Cytomegalovirus
- Herpes Simplex Virus
- Varicella-Zoster Virus

MALIGNANCIES:

- Kaposi's Sarcoma
- Non-Hodgkin's Lymphoma
- Hodgkin's Lymphoma
- Invasive Cervical
Carcinoma

AIDS DEMENTIA COMPLEX:

Cognitive, Motor and
Behavioral Impairments
in 70% AIDS Clients

Treatment of HIV



- **Prevention, public education, condoms & safe sex, accessible screening, prevention of transmission**
- **Disease monitoring**
- **Prevention of opportunistic diseases**
- **Symptom management**
- **Psychosocial, financial support**
- **Drug therapy**

HIV Drug Therapy



- Goals- decrease HIV levels to <50 / ul, maintain or increase CD4 counts, prevent symptoms & opportunistic diseases
- Antiretroviral drugs classified in terms of which part of the replication cycle they work at
- Combination therapy is used to decrease resistance and decrease likelihood the virus will recover

Types of Drugs



- **Reverse Transcriptase Inhibitors- several subtypes (eg. AZT, Viramune)**
- **Protease Inhibitors (Viracept)**
- **Fusion Inhibitors (Fuzeon)**
- **Problems: resistance, S/E, drug-drug interactions, expensive, don't always work**

Nursing Process



- **Goal to prevent opportunistic infections**
- **Assess coping mechanisms**
- **Monitor nutritional status**
- **Assess family and community support**