Infectious Disease
and
HIV/AIDS

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INFECTION DISEASE

Sepsis

I. DEFINITIONS

A. Sepsis: clinical scenario characterized by systemic inflammation and “dysregulation” leading to organ dysfunction, organ failure and possibly death

B. Bacteremia: presence of bacteria in blood

C. SIRS: (Systemic Inflammatory Response Syndrome)—host response
   1. Temp > 38ºC or <36ºC
   2. Heart rate > 90 min
   3. Respiratory rate > 20 min or PaCO2 < 32 mmHg (respiratory alkalosis may be first sign of sepsis)
   4. WBC > 12,000 cells, < 4000 or > 10% band forms

D. Sepsis: systemic response to infection—SIRS + documented infection

E. Septic shock—vasodilatory or distributive shock/hypoperfusion
   1. Sepsis with hypotension
   2. Oliguria
   3. Lactic acidosis
   4. Mental status changes

II. EPIDEMIOLOGY

A. Gram negative: more lethal, 50% of all sepsis cases endotoxin mediated

B. Gram positive: increased incidence of pneumonia/central lines/FQ use exotoxins, “super antigens”

C. High risk populations
   1. Extremes of age
   2. Comorbidities that cause immune depression
   3. Invasive devices including ventilators
   4. Prior drug therapy
   5. Miscellaneous—childbirth, trauma, burns, intestinal catastrophe
III. COMPLICATIONS

A. Acute renal failure / acute tubular necrosis

B. Adult respiratory distress syndrome

C. Disseminated intravascular coagulation

D. Heart failure—high output failure

E. Gastrointestinal bleeding

F. Liver failure

G. Death

IV. TREATMENT

A. Aggressive supportive care

B. Stabilization—“Goal Directed Therapy”

1. CVP 8-12 cm
2. ScvO2 > 70%
3. Norepinephrine or dobutamine to maintain MAP > 65 mmHg
4. Maintenance of Hematocrit > 30%
5. Intubation, sedation, mechanical ventilation
6. Renal replacement therapy

C. Blood sterilization

1. Unless organism has been identified, multi-drug combinations should be used
2. MRSA prevalence / empiric use of vancomycin specific antibiotics vary and are institution dependent

D. Underlying infection

E. Other therapies:

1. Xigris--recombinant activated Protein C
   a. Hold in situations where bleeding is a concern
   b. “High risk of death” patients only
   c. Activated protein C depleted
2. Steroids
3. Insulin
Soft Tissue Infections

I. IMPETIGO
   A. Superficial skin infection
      1. Nonbullous form (impetigo contagiosa)
         a. Honey crusted lesions
         b. GA B-hemolytic strep
      2. Bullous impetigo
         a. Staph aureus (Ca-MRSA v. MSSA)
   B. Treatment
      1. Topical: mupirocin
      2. Systemic: anti-staph agents

II. FOLLICULITIS
   A. Bacterial infection of hair follicles
   B. Most common organism--*Staph aureus*
   C. Hot tub dermatitis
      1. Pseudomonas folliculitis
      2. 1-4 days after bathing in a hot tub
   D. Generally self limited, local care, may occasionally need systemic antibiotics

III. CUTANEOUS ABSCESS
   A. Regardless of causative agent, drainage of the abscess remains the most important aspect of management
   B. Antibiotics indicated for cellulitis
   C. Perineal area—*Bacteroides fragilis*
   D. Staph, Strep (Ca-MRSA, MSSA)
      1. TMP/SMX, clindamycin, levaquin, vancomycin

IV. CELLULITIS
   A. Cutaneous infection involving the dermis and subcutaneous tissues
1. Alpha-hemolytic strep / MSSA most common etiologies
2. Induration, tenderness and erythema most common finding; systematic symptoms are rare
3. Multiple areas suggest bacterial seeding from hematogenous location

B. Specific syndromes:

1. Erysipelas
   a. Alpha hemolytic strep
   b. Sharply demarcated, indurated lesion
   c. Generally with systemic symptoms
2. Facial cellulitis
   a. Venous drainage may lead to intracranial infections or cavernous sinus thrombosis
3. Periorbital cellulitis
   a. Violaceous infection liked to Hemophilus influenza
4. Associated with bacteremia and patients can be quite ill
5. In-patient therapy generally indicated

C. Treatment

1. Penicillinase stable agents—1st generation cephalosporin, penicillinase penicillins

V. NECROTIZING FASCIITIS

A. Nomenclature

1. Confusion arising from historical derivations (pathogens, site, circumstances, eponyms, etc.) - Fournier's gangrene, Meleney's synergistic
2. Preferred is "necrotizing fasciitis"

B. Presentation

1. Can affect any body part - most common on extremities, abdominal wall, perianal/groin, post-op wounds
2. Initially erythematous, swollen, exquisitely tender and painful
3. Rapid progression from red-purple to blue-gray with bullae
4. Anesthetic due to thrombosis of small vessels and superficial nerve destruction
5. Subcutaneous gas
6. Systemic toxicity with fever of 102-105°F
7. Bedside exploration - look for undermining of skin and "dishwater" fluid
8. Mortality 20-47% overall
C. Management

1. Fluids
2. Monitoring
3. Antibiotics
   a. Triple coverage (such as ampicillin/gentamicin/Clindamycin or metronidazole)
4. Role of hyperbaric oxygen - in compromised host
5. Surgical drainage/debridement

VI. GAS GANGRENE

A. Etiology

1. Gas gangrene is synonymous with clostridial myonecrosis, which is an acute, rapidly progressive infection manifesting as gas production in the soft tissues, muscle necrosis, and systemic toxicity. Clostridium perfringens is the most common organism, but several other clostridial species have been the cause. Infection is known to follow trauma, surgery, and burns, and has been associated with malignancies.

B. Clinical course

1. After inoculation, two day incubation precedes pressure or heaviness followed rapidly by pain and tachycardia. The wound may drain dark, serosanguineous fluid followed by the development of subcutaneous emphysema. If no treatment is begun within 48 hours of the onset of systemic symptoms, mortality is 100%.

C. Treatment

1. Appropriate management of the ABCs and fluid resuscitation should begin immediately. A three-component therapy must be instituted simultaneously based on practical availabilities:
   a. Antibiotics (penicillin G or chloramphenicol) have been proven beneficial.
   b. Surgical debridement is also helpful, but the timing and extent of surgery is controversial - if a compartment syndrome is suspected, immediate fasciotomy is necessary.
   c. Hyperbaric oxygen therapy may help stop proliferation of the clostridia, limit tissue necrosis, and better delineate healthy from non-viable tissue. Tetanus prophylaxis should be administered.
VILBITE WOUNDS

A. Dog and cat bites

1. Infection rates
   a. Dog 1.6 to 30%
   b. Cat 15.6 to 50%

2. Bacteriology
   a. 33 different organisms have been isolated from dog bite wounds; 11 from cat bite wounds
   b. Common organisms
      i. Dogs: staph, strep, Pasteurella multocida, Enterobacteriaceae, Pseudomonas
      ii. Cats: Pasteurella multocida

3. Management
   a. Usual wound management
   b. Drain and cleanse (irrigate) wound
      i. Complete inspection
      ii. Foreign bodies (especially teeth)
      iii. Underlying injuries B tendons, joints
         • Assess and update tetanus status
   c. Antibiotics based on known pathogen [ideal]
   d. Empiric therapy based on local information and time course of infection
   e. Immunocompetent host with local cellulitis can be managed as outpatients
   f. If signs of infection are present within the first 24 hours after being bitten, P. multocida is likely penicillin V or amoxicillin if penicillin allergic: 1st generation cephalosporin or erythromycin. If infection develops beyond 24 hours, staph or strep are likely: antistaphylococcal penicillin or 1st generation cephalosporin
   g. Inpatient therapy for lymphangitis, tenosynovitis, septic arthritis, or systemic signs
      i. Without sepsis penicillin G and nafcillin, add an aminoglycoside if gram-negatives suspected
      ii. If septic, obtain aerobic/anaerobic blood and wound cultures, imipenem/cilastatin or ampicillin/subactam

B. Human bites

1. Frequently involve the hand (violent bite or closed fist injury), but any body part may be bitten
2. If seen before 18-24 hours after bite, usually few signs of infection
3. After 24 hours, patients usually present due to complications (infection)
4. Organisms: staph, strep and *Eikenella corrodens*
5. Antibiotics
   a. Inpatient: penicillin G and antistaphylococcal penicillin or 1st
generation cephalosporin
   b. Outpatient: amoxicillin/clavulanic acid, Penicillin allergic:
      clindamycin and (+/-) quinolone

C. Rabies
   1. Epidemiology
      a. Raccoon epizootic has spread from the Northeast US to Ohio
      b. In 1994 there were six rabies-associated human deaths
   2. Disease
      a. Recognizing the clinical syndrome of rabies is too late
      b. Encephalitis due to rabies virus is a fatal disease acquired
         through contact with an infected animal
   3. Post-exposure prophylaxis

Tick-borne Illness

I. LYME DISEASE
   A. Spirochete: *Borellia burgdorferi*
   B. Stages of Infection
      1. Stage 1—ECM: Erythema chronicum migrans
      2. Stage 2—Disseminated phase
      3. Stage 3—Late disease
   C. 1975, Lyme Connecticut—several unexplained arthritis cases
   D. New York/coastal and wooded Middle Atlantic States
   E. Wisconsin, Michigan, Minnesota
   F. Coastal and wooded California and Oregon
   G. Agents of Infection
   H. US—Ixodes dammini / Ixodes scapularis
   I. Preferred hosts of infection
      1. White-tailed deer
      2. White-footed mouse
J. Transmission requires greater than 48-72 hours of tick attachment

1. Stage 1—Erythema chronicum migrans
   a. Only 20% of patients recall tick bite
   b. Seen within 3 days to 3 weeks of bite
   c. Large annular rash
      i. < 20 cm circumference
      ii. “Burning” type pain
   d. Associated symptoms
      i. Fevers, chills
      ii. Headache
      iii. Stiff neck
   e. Often self-limited
      i. Arthralgias—80%
      ii. Neuro/CNS—15%
      iii. Cardiac—5%

2. Stage 2
   a. Lyme carditis
      i. 3 days to 3 months after bite
      ii. Generally skin lesions still present
      iii. Varying degrees of atrio-ventricular block
         • 1st Deg AVB
         • Wenkebach
         • Complete heart block
      iv. May shift from one rhythm to another
   b. Cranial nerve palsies
      i. bilateral facial nerve
   c. 50% all CNS
   d. Peripheral neuropathies/neuritis
   e. Motor or sensory
   f. 1 month duration

K. Encephalitis

1. Chronic
2. Disruptions to mood, memory and sleep
3. Cerebral vasculitis
4. Pregnancy—no known effect on mother or fetus

L. Diagnosis
1. IgG / IgM Ab via ELISA
2. Generally overused, of little utility in ED

M. Treatment
1. Stage 1
   a. Oral doxycycline, amoxicillin, cefuroxime
2. Stage 2/3
a. Ceftriaxone  
b. IV Penicillins  
c. Treatment length varies  
d. Prednisone controversial  

II. ROCKY MOUNTAIN SPOTTED FEVER  

A. Etiology—R. rickettsii  
   1. Dog tick—Dermacentor variabilis  
   2. Wood tick—Dermacentor andersoni  

B. Clinical  
   1. Infection requires the tick to be attached for several hours  
   2. Incubation of 3-12 days  
   3. Severe headaches/rigors/fever  
   4. Abdominal pain—involvement of abdominal musculature  
   5. Classic triad—fever, rash, headache  

C. Rash  
   1. Begins as erythematous macules on wrists / ankles  
   2. Over 24 hours—trunk and face with maculopapular rash  
   3. ++lesions on palms and soles  

D. Diagnosis  
   1. Thromobcytopenia  
   2. Antibody assays  

E. Treatment  
   1. Doxycycline / Tetracycline—even in children  
   2. Chloramphenicol if allergic  

III. Q FEVER  

A. Acute systemic infection of the lung  

B. Vector is the tick—via inhalation of aerosolized particles infected with C. burnetii  

C. Clinical  
   1. Presents as atypical pneumonia  
   2. 10-40 day incubation period  
   3. NO rash
4. Granulomatous hepatitis
5. Hepatosplenomegaly
6. Jaundice, abnormal liver function tests

IV. CHRONIC Q FEVER—ASSOCIATED WITH ENDOCARDITIS

A. Diagnosis—antibody titers, IgM
   1. Treatment
      a. Doxycycline / Tetracycline / Fluorquinolone
      b. +/- Rifampin

B. Ehrlichiosis
   1. Etiology—E. chaffeensis

C. Parasite
   1. 25% of all infections co-infected with B. burgdorferi
   2. Requires 24-48 hours of tick attachment
   3. Clinical
      a. 10 days
         i. Fevers, HA, myalgias
         ii. Mental status changes
   4. Diagnosis

D. Leukopenia

E. Elevated transaminases

F. Treatment—Doxycycline

Influenza

I. EPIDEMIOLOGY

A. Influenza A and B virus—yearly worldwide epidemics with origins in SE Asia

II. COMPLICATIONS

A. Influenza Pneumonia—viral infection in lung, generally severe

B. Bacterial Pneumonia—superinfection
   1. Streptococcus pneumoniae: 50%
   2. Due to influenza infection and abnormalities with
tracheobronchial tree/impaired secretion clearance
3. Clinical—rapid/abrupt decline after improvement

C. Myositis—markedly elevated CK, prominent in legs

D. Reye Syndrome
   1. ?Influenza “B” v. VZV
   2. Profound encephalopathy
   3. Elevations in LFTs with near normal bilirubin
   4. Hepatomegaly
   5. Elevated serum ammonia
   6. Associated with aspirin use

III. TREATMENT
   A. Aggressive supportive care – immunization
   B. Neuraminidase inhibitors—effective for tx or prophylaxis
   C. Amantadine / Rimantadine
   D. Ribavirin

SARS (Severe Acute Respiratory Syndrome)

I. EPIDEMIOLOGY
   A. Worldwide outbreak 2003
   B. China, Hong Kong, Vietnam, Singapore and Canada
   C. Approximately 8,500 cases with >900 death
      1. Fatality rate > age 60 was 43%
      2. Few cases in age < 15 yrs

II. PATHOPHYSIOLOGY
   A. SARS Coronavirus—possible relation to civet coronavirus
   B. Spread by direct droplet spread (suggested by clustering of infection)

III. DIAGNOSIS—WHO CRITERIA
   A. Fever > 38°C
B. Cough or shortness of breath

C. Close contact with known infection or known symptoms 10 days after travel to endemic area

D. Radiographic findings
   1. Normal to diffuse interstitial infiltrates (ARDS)
   2. Pneumothorax/pneumomediastinum in advanced cases
   3. Small parenchymal cysts seen on CT scan

E. No current clinically useful tests exist (involve reference testing/CDC)

IV. TREATMENT

A. Aggressive supportive care
B. Rivavirin—no effect against SARS corona virus
C. Prevention and early quarantine of future outbreaks
D. Vaccine development

West Nile Virus

I. EPIDEMIOLOGY

A. Widely distributed flavivirus, not seen in North America until 1999
B. Bird-Mosquito-Bird Cycle
   1. Humans/other animals are incidental hosts
   2. Human transmission from mosquito or from infected blood exposure

II. PATHOPHYSIOLOGY

A. Virus-laden saliva injected into host
B. Viremia leads to seeding of CNS, resulting in encephalitis
C. 2-14 day incubation period
D. Peak incidence in late summer/early fall
III. CLINICAL

A. Acute Febrile Illness (West Nile Fever)
   1. Muscle weakness 60%
   2. Difficulty concentrating 50%
   3. Rash—maculopapular
   4. Neuroinvasive meningoencephalitis
      a. More common in elderly
      b. Persistent cognitive and neurological impairment
      c. Severity of presentation not indicative of long-term prognosis
   5. Often confused with Guillain-Barre
      a. Focal neurologic findings
      b. Brachial plexopathy
      c. Weakness
   6. Ocular—vitritis or chorioretinitis
   7. Hepatitis or pancreatitis
   8. Central diabetes insipidus

B. Serologic
   1. IgM Ab to virus in serum or CSF
   2. IgM timing may be delayed
   3. IgM may persists for up to 6 months
   4. Cross reactivity with other flaviviruses or vaccines

IV. TREATMENT

A. Aggressive supportive care

B. Ribavirin studies suggest possible detrimental effect

C. Prevention
   1. Vaccination—2 currently exist
   2. Personal protection measures—DEET
   3. Donor blood screening

Herpes Virus Infection

I. EIGHT VIRUSES

   A. HSV-1, HSV-2

   B. VZV
C. CMV

D. EBV

E. HHV-6, 7

F. HHV-8, KSHV (Kaposi Sarcoma associated herpes virus)

II. CHARACTERISTICS OF HERPES VIRUS INFECTION

A. Latency and reactivation

B. Viral DNA persists but complete replication and destruction of infected cells does not

C. HSV/VZV – neural ganglion cells

D. EBV– B cells

E. CMV– Multiple

F. HHV – 6, 7 - ?, salivary glands

III. HERPES SIMPLEX VIRUS 1, 2

A. Epidemiology

B. Humans only known host

1. HSV1 – primary infection occurs mainly in childhood
2. HSV2— infection occurs predominantly in sexually active adolescents and young adult
3. Direct contact with infected secretions/sores is the principal mode of transmission
4. HSV1—primarily oral
5. HSV2—primarily genital
   a. More likely male to female
   b. Occurs even in the absence of lesions
   c. Recurrences and severity tend to decrease over time

C. Clinical syndromes

1. Oral-Labial
   a. Generally asymptomatic
   b. Gingivostomatitis or pharyngitis
      i. Small vesicles on oral mucosa and pharynx
      ii. Pain, halitosis, cervical adenopathy
      iii. May persist for up to 3 weeks
c. Recurrent herpes labials
   i. Shorter, milder
   ii. Heralded by local pain, tingling
   iii. Vesicles at vermilion border
      • Crust in less < 3 days
      • Precipitated by wind/sun, local trauma, fever, menstruation, emotional stress

2. Ocular herpes
   a. HSV-1, unilateral follicular conjunctivitis, blepharitis or corneal epithelial opacities
   b. Reactivation–keratitis, blepharitis, keratoconjunctivitis
   c. Branching dendritic ulcers seen on fluorescein
   d. Diminished visual acuity

3. Genital herpes
   a. Incubation of 2-7 days, followed by fever, malaise and inguinal
   b. Adenopathy
   c. Urethral involvement sometimes results in dysuria or urinary retentions

4. Aseptic meningitis
   a. Recurrence with prodrome of tenderness, itching or tingling
   b. Healing occurs in 6-10 days

5. Perianal and anal herpes
   a. Pain, itching, tenesmus, discharge, fever, chills, headache, sacral paresthesias
   b. Difficulty in urination may occur
   c. Vesicles and ulcerations leading to erythematous cryptitis with inguinal adenopathy
   d. Herpetic Whitlow
      i. Generally involves one digit—neighboring finger may be involved
      ii. Intense itching or pain followed by formation of deep vesicles that may coalesce

6. Medical—HSV-1
7. General public—HSV-2
   a. Lesions resolve in 2-3 weeks
   b. Do not incise

8. Bell's Palsy
9. HSV Encephalitis

D. Neonatal infection

1. HSV-2—either ascending or deliver through infected genital tract
   a. Risk
      i. Premature
      ii. Prolonged membrane rupture
   b. Fetal scalp electrodes
2. Clinical presentation
   a. Days to weeks after delivery
   b. Vesicles, conjunctivitis
   c. Sepsis
   d. Seizures, CNS, lethargy
   e. Disseminated infection may involve the liver, lungs or adrenal glands
   f. Untreated--->70% fatal
   g. Localized disease is generally self limited

3. Treatment
   a. Cesarean section: indicated to prevent perinatal infection
   b. Acyclovir/Valcyclovir

4. Genital—suppressive of severe and recurrent infection
   a. 10mg/kg TID of acyclovir for herpes encephalitis

Varicella-Zoster Virus

I. CAUSATIVE AGENT OF CHICKEN-POX AND SHINGLES (VARICELLA AND HERPES ZOSTER)

A. Primary infection: Chicken Pox

B. Spread by respiratory droplets or by direct contact with active lesions
   1. Most frequently in winter/spring
   2. Incubation 10-20 days

C. Clinical
   1. Headache, fever and malaise
   2. Vesicles generally appear initially on the face or trunk
   3. Most cases are uncomplicated and resolve in 7 to 10 days without
   4. Scarring

D. Complications
   1. Pneumonia
   2. Encephalopathy
   3. Reye Syndrome—encephalopathy and fatty liver nephritis,
      orchitis, carditis congenital varicella syndrome—primary
      infection during first 20 weeks of pregnancy
   4. Limb hypoplasia
   5. Skin scarring
   6. Ocular defects
   7. Disseminated disease
   8. Leukemia, lymphoma, Hodgkin’s Disease
Herpes Zoster-- Reactivation of VZV Infections

I. VARICELLA IN ONE PATIENT CANNOT PRODUCE HERPES ZOSTER IN ANOTHER

A. Persons exposed to patient with herpes zoster can contract varicella

B. Associated with depressed T-cell function

C. Virus persisted in sensory ganglia neurons

D. Clinical features

1. Pain, several days before lesions appear
2. Unilateral, appears on the thorax
3. Limited from 1-3 dermatomes, although a few isolated skin lesions distant from area are not uncommon
4. Vesicles with erythematous base
5. Superinfections are common
6. Complications
   a. Postherpetic neuralgia
   b. More severe, particularly in elderly
   c. May persist for months/years
   d. Segmental Myelitis
   e. Guillain-Barre
   f. Ramsay-Hunt
7. Infection of geniculate ganglion of the seventh cranial nerve facial paralysis vesicles on eardrum and side of the tongue

E. VZV encephalitis

1. Treatment
2. Isolation of patients
3. Immunocompromised patients who are exposed should receive prophylaxis with VZV immune globulin (up to 4 days after exposure)
4. Vaccinations
   a. Single dose in children
   b. Booster in adults
      i. IV acyclovir
         • May shorten the course and reduce severity
      ii. Steroid use controversial—may be justified in persons older than 50
Cytomegalovirus

I. UBIQUITOUS VIRUS

A. Transmission occurs by close contact including genital in utero primary infection

B. Primary infection may result in CMV inclusion disease

C. Numerous congenital abnormalities

II. PRIMARY INFECTION

A. CMV Mononucleosis--Normal Host

1. Vigorous host T-cell response
2. Fever, malaise, fatigue and myalgia
3. Headache, splenomegaly
4. Mild liver enzyme abnormalities
5. Atypical lymphocytoses
6. NO heterophile antibodies
7. May have positive RF and ANA
8. Complications
   a. Hemolytic anemia
   b. Guillain-Barre
9. Resolves in 2-4 weeks

Epstein-Barr

I. INFECTS HUMAN B CELLS, EPITHELIAL CELLS OF NASOPHARYNX AND UTERINE CERVIX

A. Associated with Non-Hodgkin’s, Hodgkin’s and Burkitts lymphoma

B. Heterophile antibody-positive infectious mononucleosis

1. Transmission requires close contact, generally oral-oral
   a. Infected B-cells—produce a variety of antibodies heterophile antibody
   b. Immortalized--capable of continuous proliferation
2. Clinical features
   a. Young children
      i. Asymptomatic or trivial infections—generally do not produce heterophile antibodies
   b. Elderly
      i. Persistent febrile syndrome--generally do not produce
heterophile antibodies

C. Infectious mononucleosis

1. Clinical
   a. Fever
   b. Exudative pharyngitis
   c. Lymphadenopathy
   d. Hepatosplenomegaly
   e. Palatal enanthema
   f. Periorbital edema
   g. Jaundice
2. Maculopapular diffuse rash associated with ampicillin
   a. Resolves in 1-3 weeks
   b. Malaise and fatigue may persist for weeks to months
   c. Splenic rupture: rare, abdominal pain during the second and third weeks of illness
3. Testing
   a. Heterophile Ab—may persist > 1 year
   b. EBV antibodies
   c. Presence of IgM antibodies to viral capsid antigen suggests acute infection

D. Treatment—supportive

Other Human Herpesviruses

I. HHV-6
   A. 80% adults are positive

   B. Roseola
      1. Exanthema subitum
      2. Fever followed by a rash
         a. Often associated with febrile seizures
      3. Suggested may play a role in MS

II. HHV-7

   A. Human T-cell infection

   B. 90% are seropositive

   C. Association with CFS (HHV-6)
III. HHV-8
   A. Viral sequences with KS and body cavity based lymphoma in patient with AIDS
   B. Suggested relationship with sarcoidosis and multiple myeloma

Malaria

I. EPIDEMIOLOGY
   A. Four species: P. falciparum, P. vivax, P. ovale, P. malariae
   B. 500 million cases a year; 1-2 million deaths/year; Anopheles mosquito

II. LIFE CYCLE
   A. Sporozoites injected from salivary gland of Anopheles
   B. Infect liver and divide (may be subacute process)
   C. Travel via bloodstream
   D. Infect RBCs
   E. Merozoites are released further infected RBCs

III. FALCIPARUM
   A. Location: Tropical Africa, SE Asia, Republic of Haiti, Amazon, Dominican Republic
   B. Can infect up to 50% of all RBCs
   C. Mortality can exceed 30%
   D. 10-14 day incubation period
   E. 48 hour fever spike

III. PREVENTION
   A. Chloroquine
      1. Effective against all 4 species
2. Widely prevalent chloroquine resistance among Falciparum sp.

B. Mefloquine / Atovaquone-proguanil / Tetracycline are primary choice for current travelers

Cystercercosis

I. OVERVIEW

A. Taenia solium—pork tapeworm
   1. Undercooked pork/sausage—type of disease depends on which stage of lifecycle ingested
   2. Eggs—CNS
   3. Cystercerci—GI
   4. Clinical
   5. New-onset seizures
   6. CT—encysted / calcified lesions

B. Conventional anticonvulsants

C. Neurosurgery

D. Tapeworm

E. Rx—Mebendazole for acute infection
INFECTIOUS DISEASE

PEARLS

1. Sepsis is a syndrome characterized by systemic inflammation and dysregulation leading to organ dysfunction and possibly death.

2. SIRS is characterized by hyper- or hypothermia, tachycardia, tachypnea.

3. Respiratory alkalosis is often the first sign of SIRS.

4. Treatment of underlying disorder/infection is paramount in the appropriate management of sepsis.

5. Lyme disease, left untreated, will often progress through three characteristic stages including rash, disseminated and late disease.

6. Cardiac involvement of Lyme disease is manifest by varying degrees of atrioventricular block.

7. Rocky Mountain Spotted Fever exhibits the classic triad of fever, rash and headache associated with tick bite. Treatment includes tetracycline, even in children.

8. Influenza infection has been associated with Reye syndrome. Bacterial superinfection is not uncommon.

9. Herpesvirus infection is characterized by infection, latency and reinfection.

10. Bell’s palsy is associated with HSV infections.

11. Malaria infections are characterized by cyclic fevers associated with lysis of infected RBC cells.

12. Impetigo is caused by group A beta hemolytic strep. or S. aureus. Bullous forms almost always caused by S. aureus. Treat topically with mupirocin or systemically with anti-Staph agent.

13. Cellulitis is an infection of the deeper dermis and sub-Q fat.

14. Erysipelas is group A strep infection that is sharply demarcated and accompanied by fever and chills.

15. Facial cellulitis can spread intracranially via venous channels.
REFERENCES


HIV/AIDS

I. DEFINITIONS

A. HIV-1
   1. Human retrovirus responsible for majority of AIDS cases; many different strains.
   2. Mutated SIV from chimpanzees.

II. STATUS OF HIV TESTING AND DISEASE MARKERS

A. EIA screening test—cost: $60; sensitivity > 99.9%
B. Western blot confirmatory test—cost: $75; tests 2 major viral proteins
C. CD4 Lymphocyte count—: $50-100; target cell for HIV; predictor of disease progression measured at time of diagnosis and every 3-6 months
D. Viral load test—cost $100-200; most important predictor of disease progression; measures HIV RNA in blood; high numbers = more active disease
E. Rapid tests—SUDS 10-15 min/test
F. Saliva test—cost $25; HIV-Ab screen and confirmatory Western Blot
G. Absolute Lymphocyte Count
   1. ALC > 2000 suggestive of CD4 > 200
   2. ALC < 1000 suggestive of CD4 < 200

III. PRIMARY HIV INFECTION: ACUTE RETROVIRAL SYNDROME

A. Frequency—seen in 50-90% patients
B. Time of onset: 2-4 weeks post-exposure as a mono-like syndrome lasting 1-2 weeks, but may last for up to 6 weeks
C. Clinical: fever (95%), fatigue, lymphadenopathy (74%), non-exudative pharyngitis (70%), weight loss, myalgias (54%), diarrhea (32%), headache (32%), and measles-like rash (70%)
D. Differential diagnosis: EBV mono, primary CMV or HSV, viral hepatitis, rubella, toxoplasmosis, secondary syphilis, measles, disseminated GC, drug reaction
E. DX: Lymphopenia is a common lab finding; confirmation by finding a positive plasma HIV RNA obtained on same day as a negative Western blot

IV. CURRENT TREATMENT CONCEPTS

A. HIV RNA levels - viral load; useful in determining prognosis, estimate the risk of disease progression and aid in making antiretroviral therapy decisions

B. Goal: viral load undetectable = < 50 RNA copies/mL

C. Treatment goals:
   1. After initiation of treatment: undetectable; < 400 copies/mL.
   2. Antiretroviral activity: > 0.5 log decrease.
   3. Drug treatment failure: rise in viral load; failure to achieve desired reduction.
   4. HAART (highly active antiretroviral therapy) treatment standard regimen consisting of triple therapy with two NA’s plus a PI or a NNRTI can reduce the viral load to < 50 RNA copies/mL in treatment of naive patients.
   5. Monotherapy creates resistance.
   6. It is the “all or none” concept - take all three drugs or stop them all then restart a new regimen.

V. COMMON SIDE EFFECTS OF ANTI-HIV DRUGS

A. Nucleoside Analogues
   1. Zidovudine (AZT)
      a. Hematological: macrocytic anemia
      b. GI intolerance

B. Lamivudine (3TC)
   1. Rash
   2. Well-tolerated

C. Stavudine (d4T)
   1. Neuropathy (30%)
   2. Diarrhea
   3. Anemia

D. Didanosine (ddI)
1. Neuropathy (15%)  
   a. Pancreatitis (10%)  
2. Diarrhea

E. Zalcitabine (ddC)  
   1. Neuropathy (30%)  
   2. Pancreatitis, stomatitis

F. Abacavir  
   1. Hypersensitivity reaction (GI, rash, respiratory) can be life-threatening reaction if re-challenged (3%)

G. Protease inhibitors  
   1. Saquinavir—well tolerated  
   2. Retonavir  
   3. Indinavir  
      a. Kidney stones (8-13%)  
      4. Nelfinavir—GI intolerance

H. Non-Nucleoside Reverse Transcriptase Inhibitor  
   1. Delavirdine—Rash  
   2. Lopinavir  
      a. Abdominal pain  
      b. Hyperglycemia, rash, peeling skin

VI. OPPORTUNISTIC INFECTIONS—OVERVIEW / MOST COMMONS  

A. Opportunistic Infection → PCP  
B. Systemic Opportunistic Infection → MAC  
C. CNS Fungal Infection → Cryptococcus  
D. Serious Viral Infection → CMV  
E. Gastrointestinal Infection → Candida  
F. Cause of Retinitis → CMV  
G. Cause of focal encephalitis → Toxoplasma  
H. Complaint → diarrhea
VII. RESPIRATORY INFECTIONS

A. Pneumocystis jiroveci (carini) pneumonia (PCP)

1. Most common life-threatening OI; declining incidence from 50% in 1987 to 25% currently

2. Clinical
   a. Gradual onset over weeks
      i. Fever, SOB, DOE, non-productive cough; physical findings minimal: tachypnea, +/- rales

3. PCP suspected
   a. Increased LDH, decreased PaO₂, increased WBC

4. CXR
   a. diffuse, bilateral infiltrates
   b. mild disease—normal
   c. atypical upper lobe disease with aerosolized pentamidine
   d. Spontaneous PTX and pneumatoceles are common.
   e. High Resolution CT—ground glass appearance

5. PCP Classification
   a. Mild: PaO₂ > 70, A-a gradient < 35
      i. po meds +/- output therapy - if tolerating po meds and not clinically ill can discharge home
   b. Severe acute ill
      i. PaO₂ < 70, A-a gradient >35
      ii. IV antibiotics, steroids, admit

6. Treatment
   a. all treatment—21 days
      i. Trimethoprim/sulfamethoxazole (TMP/SMX)
      ii. 5 mg/kg/dose IV or 2 DS tablets Q 8 hrs
      iii. 25% develop rash +/- fever
      iv. Alternatives: pentamidine IV (if Bactrim allergic), TMP/dapsone, clindamycin plus primaquine (good po outpatient regimen), or atovaquone
   b. Steroids reduce risk of respiratory failure and death by 50%
   c. Attenuate inflammatory response due to organism/tissue destruction
      i. Recommended if PaO₂ < 70 mmHg, A-a gradient >35, or pulse ox < 90% on room air; contraindicated for suspected TB or disseminated fungal infection
      ii. Dose: Prednisone 40 mg po bid X 5 d, then taper for remainder of treatment (11 days)
      iii. Methylprednisolone 125 mg IVP - Give 15-30 mins prior to antibiotic

7. PCP prophylaxis
   a. CD4 less than 200
   b. Previous PCP
   c. Oral TMP/SMX 1 double strength tab QD or 3 times a week
B. Mycobacterium tuberculosis

2. Occurring with increased frequency and severity.
3. Risk factors: social- prison, shelters, IVDA, inner city, and staff of such institutions.
4. Respiratory isolation and mask with any febrile coughing HIV patient.
5. Atypical late in disease: SOB > cough, lower lobe infiltrate, +/- cavitation, systemic symptoms.
6. Typically CD4 <400 may be atypical or miliary with advanced disease.
7. Diagnosis
   a. Sputum AFB smear and cultures (50%); collect under controlled conditions.
   b. PPD
      i. PPD + in early HIV
      ii. PPD often negative in late HIV (30-50%)
      iii. Anergy is common; ANY HIV patient with PPD > 5 mm is considered to have clinical infection
   c. CXR: often atypical lower lobe, diffuse infiltrates, or marked adenopathy
8. Extrapulmonary involvement common: lymph nodes, liver, blood, bone marrow, meninges; common presentation for multi-drug resistant TB.
9. Treatment
   a. Minimum of triple drug therapy; need four-drug therapy in resistant areas
   b. INH, rifampin, pyrazinamide, ethambutol (or streptomycin) daily for 2 months followed by 18 weeks of INH/RIF 2-3 times a week; if INH or RIF not used, treat at least 18 months after negative culture
   c. Ethambutol if extrapulmonary TB or INH resistance is suspected
   d. Respiratory isolation may be discontinued after 2 weeks of chemotherapy or 3 negative sputum samples

C. Mycobacterium Avium Complex: no need for isolation; MAI/MAC is everywhere

1. Persistent bacteremia with widespread systemic symptoms: fever, night sweats, fatigue, N/V/D, abdominal pain, generalized lymphadenopathy, anemia, jaundice, hepatosplenomegaly, respiratory symptoms.
2. Diagnosis Presumptive MAI.
3. AFB in stool smears and cultures (98%); anemia and leukopenia, elevated alkaline phosphatase.
4. Disseminated MAI: usually CD4 < 50
   a. Most common SYSTEMIC opportunistic bacterial infection
5. Treatment
   a. clarithromycin + ethambutol or rifabutin
6. Prophylaxis—clarithromycin or azithromycin +/- rifabutin.

D. Bacterial pneumonia

1. Occurs frequently in HIV; recurrent bacterial pneumonia is AIDS case definition.
3. Clinical
   a. As per normal host—fever, productive cough, pleuritic chest pain, signs of consolidation
   b. symptoms generally abrupt and present for less than one week
4. Diagnosis
   a. Sputum gram stain and culture and blood culture; bacteremia more common
5. CXR: segmental or lobar infiltrates
6. Treatment—same as in normal host
   a. All macrolides can produce drug interactions with AZT, ddI
   b. HIV patients should receive pneumococcal vaccine

B. Other causes of respiratory symptoms

1. Kaposi’s sarcoma: commonly seen with cutaneous involvement
   a. SOB, dry cough, hemoptysis, fever, CP
   b. CXR: spreading linear densities, nodular infiltrates
2. Spontaneous pneumothorax
3. Pericardial tamponade—Complication of aerosolized pentamidine, PCP pneumonia or TB
4. Cardiomyopathy
5. Sinusitis: very common; S. pneumonia, H. influenza

VIII. CENTRAL NERVOUS SYSTEM INFECTIONS

A. CT scan with contrast and/or MRI—perform if risk factors for HIV with unexplained neurologic complaints; 50% have cerebral atrophy, focal lesions, mass effect

1. LP (if no mass effect)
   a. Smear for AFB
Infectious Disease and HIV/AIDS

b. VDRL
c. Fungal/AFB culture
d. CMV-PCR
e. Cryptococcal AG

B. Cryptococcus neoformans meningitis: Most common FUNGAL CNS infection

1. CD4 count usually < 100
2. May be disseminated or isolated or initial manifestation
3. Clinical
   a. May be subtle—HA (75%), fever variable
   b. Changes in personality or mentation occur slowly; photophobia and neck stiffness are rare; cranial nerves often involved
   c. Seizures are uncommon (10%), no focal deficits
4. Diagnosis
   a. CT scan: atrophy, hydrocephalus, non-diagnostic; may see cryptococcomas
   b. CSF examination and culture
   c. No pleocytosis; glucose and TP normal
5. India ink (CFS) smear positive 60%-80%
   a. Opening pressure > 200 mm in 70%.
6. Cryptococcal antigen: serum 95% - indicates systemic disease
7. CSF: 99% positive in cryptococcal meningitis

C. Treatment

1. Amphotericin B +/- flucytosine for 2 weeks then fluconazole or itraconazole po X 8-10 weeks; daily LP removing 30 ml at a time may be needed to reduce ICP
2. Prophylaxis—life-time with fluconazole

D. Toxoplasmosis: most common cause of FOCAL encephalitis

1. Focal
   a. Reactivation of Toxoplasma gondii obligate intracellular parasite; oocytes transmitted by cats, flies, cockroaches, and undercooked meat
   b. Encephalitis seen with CD4 count < 100
2. Clinical
   a. fever, HA, confusion, lethargy; seizures 30%; focal neuro deficit in 75%
3. Diagnosis
   a. Contrast CT scan, MRI: single or multiple ring or nodular-enhancing lesions with surrounding hypodensity (edema) usually found in basal ganglia
   b. TB, lymphoma, fungus, hemorrhage can appear as similar
lesions
  c. CSF: nonspecific and may be normal; brain biopsy definitive
4. Toxoplasmosis titers
  a. Brain biopsy – definitive
5. Treatment
  a. Treat for 8 weeks or until neg. CT
  b. Pyrimethamine plus folinic acid plus sulfadiazine or
     trisulfapyrimidines
  c. Steroids are controversial; may be required to decrease edema
  d. Lifetime prophylaxis with sulfadiazine, pyrimethamine and
     folinic acid
E. CMV encephalitis: CD4 count < 100
   1. Clinical
      a. Fever, +/- delirium, rapidly progressive confusion, apathy,
         malaise and HA no focal neuro deficits
   2. Diagnosis
      a. CT: diffuse low attenuation areas; MRI: high signal areas;
         CSF: CMV-PCR
   3. Treatment
      a. Ganciclovir, foscarnet
F. HIV meningitis: aseptic encephalitis
   1. Direct consequence of HIV; may be self-limited
      a. Clinical— HA, meningismus
      b. Diagnosis: exclude opportunistic infection;
         i. CSF: pleocytosis, increased protein, p24 Ag, HIV
            culture, PCR
      c. Treatment— supportive with high-dose AZT
G. Bacterial meningitis—uncommon in HIV; consider Listeria if
   suspicious
H. Infiltrative CNS Disorders
   1. Primary CNS lymphoma
      a. Clinical
      b. HA, seizures, focal deficits, encephalopathy, CN
         polyneuropathy, fever
      c. Diagnosis
         i. Requires brain bx to confirm
         ii. CT: hypodense solitary lesions with peripheral
             enhancement; may be primary or metastatic disease;
             Thallium CT brain positive
      d. CSF: variable; normal to increased protein, decreased glucose
      e. Treatment
i. Radiation or multiagent chemotherapy; if edema, consider Decadron/mannitol

f. Often mistaken for toxoplasmosis and then identified after no improvement in 7-10 days of antibiotic therapy; prognosis poor

2. Progressive Multifocal Leukoencephalopathy (PML)
   a. Rare progressive, demyelinating disease caused by the papovavirus JC
   b. Clinical—diffuse encephalopathy, behavioral changes, mental impairment, dementia, focal deficits; progresses rapidly over several months
   c. Diagnosis—CD4 count < 100
      i. EEG: diffusely slow
      ii. CSF: positive PCR for JC
      iii. CT: multiple non-enhancing lesions of the white matter; MRI: demyelination.
   d. Treatment
      i. Poor prognosis; antiretroviral therapy may be helpful

3. Treatment—TCAs, anticonvulsants, neurontin, acupuncture

IX. GI MANIFESTATIONS OF HIV

A. Diarrhea: most common symptom in HIV—50-60% AIDS patients

1. Common causes of diarrhea: a specific agent is not found in 30-50% of patients; medications are a frequent cause
   a. Enteropathic bacteria: Salmonella, Shigella, Campylobacter, C. difficile if previously on antibiotics
   b. Mycobacterium avium complex
      i. Clinical—abdominal pain, anemia, weight loss, fevers, night sweats, lymphadenopathy
      ii. Diagnosis—AFB in stool smears and culture
         ● Barium: nonspecific mucosal abnormalities
         ● Biopsy of small or large bowel, liver, BM, lymph nodes, blood cultures
         ● CT: large retroperitoneal lymph nodes
      iii. Treatment—clarithromycin + ethambutol OR rifabutin + ethambutol
   c. CMV
      i. Clinical—diarrhea, bleeding or perforation
      ii. Diagnosis—barium: abnormal colonoscopy: normal mucosa, erythema, submucosal bleeding or discrete ulcers; biopsy: CMV IgG antibody-usually present
      iii. Treatment—Ganciclovir IV or po, Foscarnet po
   iv. All patients should have eye exam to exclude retinitis
   v. Parasites—collect multiple stool samples
      ● Entamoeba histolytica-Clinical-Proctocolitis: diarrhea, tenesmus, abdominal pain, rectal discharge
• Giardia lamblia—Clinical-nausea, severe watery diarrhea, cramps, bloating, malabsorption
• Diagnosis—If neg stool exam for trophozoites, perform string test
• Isospora belli—acid-fast protozoa; watery diarrhea
• Cryptosporidium—coccidian protozoa seen with AFB stain; Sx: cholera-like diarrhea, N/V, cramping, weight loss, malabsorption, acalculous cholecystitis common
• Microsporidia

B. Proctitis

1. Causes—Herpes simplex, gonorrhea, candida, syphilis, Chlamydia trachomatis, HPV, Enterobius vermicularis, Phthirus pubis, Sarcoptes scabiei, trauma
2. Clinical—tenesmus, mucoid, suppurative discharge
3. Treatment—as per cause; stool softeners prn; for HSV: oral acyclovir

C. Oral candidiasis

1. Most common infection of the GI tract of AIDS
2. Usually asymptomatic but may cause odynophagia

D. Esophagitis—intraoral lesions may or may not be seen

1. Infectious—Candida, HSV, CMV, hyperacidity; Kaposi’s sarcoma
2. Clinical-dysphagia (commonly Candida); odynophagia (usually HSV, CMV, aphthous ulcers), low-grade fever, weight loss
3. Diagnosis—barium swallow or EGD
4. Treatment-ketoconazole; remember gastric acid is needed for ketoconazole – don’t mix with H2 blockers; fluconazole + nystatin swish and swallow if Candida

E. Bowel obstruction: May be secondary to lymphadenopathy with TB, CMV, HIV, lymphoma

F. AIDS cholangiopathy

1. Causes: Cryptosporidium, Microsporidia, CMV
2. Clinical-RUQ abdominal pain without diarrhea, fever, elevated alkaline phosphatase, normal bilirubin; acalculous cholecystitis; may be result of portal lymphadenopathy from opportunistic infections and neoplasm
G. Pancreatitis: very common

1. Causes: direct viral injury, secondary to gall bladder disease, extrinsic compression due to retroperitoneal nodes or neoplasm or side effect of medications: ddI, ddC, pentamidine IV

X. DERMATOLOGIC MANIFESTATIONS IN HIV PATIENTS

A. Skin findings may be the earliest sign of AIDS

1. Herpes zoster in young person, or increased severity of disease
2. Over 90% have skin manifestations acute exanthem occurs with primary infection; non-pruritic macular-papular on face and trunk; important to exclude possibility of a drug reaction, e.g., sulfa.

B. Mucocutaneous HSV

1. Clinical—painful grouped vesicles or non-healing ulcers around nose, mouth, perineum
2. Diagnosis—viral culture
3. Treatment—acyclovir po; if ocular involvement, may need IV

C. Mucocutaneous Candida (see GI section)

D. Varicella zoster

1. Clinical—may precede the diagnosis of AIDS; diffuse, bilateral varicella hospitalization needed
2. Acyclovir—consider Varicella Immune Globulin

E. Bacillary angiomatosis—Bartonella hensalae and Bartonella Quintana

1. Tender friable vascular papules and subcutaneous nodules bx to distinguish from KS
2. Treatment—erythromycin or doxycycline X 8 wks

F. Kaposi’s sarcoma (KS)—uncommon in heterosexual HIV patient

1. Clinical—painless, large violaceous, malignant lesions; skin, oral/mucosal and visceral
2. Associated with HHV-8; anti-retroviral therapy, radiation, alpha-interferon, chemotherapy

G. Hairy leukoplakia—probable reactivation of EBV
1. Clinical— whitish-gray plaque adherent to lateral edges of tongue
2. Treatment—acyclovir

XI. OPHTHALMOLOGIC DISEASE IN HIV PATIENTS

A. Cotton wool spots are one of the earliest signs

B. CMV retinitis; 20-40% in patients with CD4 < 50
   1. Clinical—blurred vision or floaters initially unilateral, central or peripheral, painless blindness occurs despite therapy
   2. Cottage cheese and ketchup lesions
   3. Treatment— intraocular ganciclovir release devices Q 6 months plus ganciclovir po; foscarnet IV or cidofovir IV plus probenecid

C. Toxoplasma chorioretinitis

D. Other ophthalmic HIV diseases—conjunctivitis (Reiter’s), optic neuritis, MAI, histoplasmosis, cryptococcosis, candidiasis, pneumocystosis.

E. Obtain ophthalmologist consultation to guide therapy and disposition

XII. HIV AND WOMEN AND CHILDREN

A. Incidence
   1. Fastest growing group is young women
   2. Nearly all children with HIV acquire it perinatally 15% total AIDS cases, 20% new cases are women; 50% acquired HIV heterosexually; 3rd leading cause of death of young females; 1st for young African American females

B. Frequent complications
   1. HPV and Cervical Neoplasia: high incidence of abnormal Pap (obtain Pap Q 6 mos)
   2. Severe PID
   3. Breast CA
   4. Recurrent or persistent vaginal fungal infections: e.g., Candida
   5. HIV transmission to infant
   6. AZT given during pregnancy, labor, delivery and administration to newborn for first 6 weeks of life reduces transmission to 8% compared with 25% in untreated patients
   7. Breast-feeding increases risk of HIV from 10% to 19% and
C. Occupational / Post-Exposure Prophylaxis

1. 2/3 of all patients have unknown status; use Universal Precautions. Standard Precautions
2. Assess risk of HIV, HBV, HCV; determine HBV and HIV status of both victim and source
3. All wounds should be washed with soap, water

XIII. HIV

A. HIV Seroprevalence rate of 0.1-14.6% in all patients presenting to an ED (inner city > suburban). Recent report cited 30 yr risk for ED physician at 0.1-1.4% depending upon demographics

1. Risk of seroconversion following exposure to HIV positive source
   a. Needlestick or cut - 0.3%
   b. Eye, nose, mouth - 0.1%
   c. Intact skin exposure < 0.1%
   d. Unprotected anal intercourse - 0.008-0.032 infections/exposure
   e. Receptive vaginal intercourse - 0.0005-0.0015 infections/exposure
   f. HIV Post-exposure prophylaxis (PEP) - reduces risk of seroconversion by 79%

2. Determine risk of exposure:
   a. Highest risk: BOTH larger volume of blood (e.g. deep injury, large-diameter needle previously in course patient’s vein or artery) AND high titer of HIV (e.g. source patient with acute retroviral illness or end-stage AIDS)
   b. Increased risk: EITHER larger volume of blood OR high titer of HIV
   c. No increased risk: No larger volume of blood, No high titer of HIV, (e.g. injury with solid suture needle from source patient with asymptomatic HIV)

3. Percutaneous exposure:
   a. Blood— high risk
      i. Recommend ZDV + 3TC + IDV
   b. Blood—increased risk
      i. Recommend ZDV + 3TC + IDV
   c. Blood— no increased risk
      i. ZDV + 3TC
   d. Fluid containing visible blood, other potentially infectious fluid or tissue
      i. Offer ZDV + 3TC

4. Other body fluid (urine)
a. Don’t offer
   i. Mucous Membrane—Blood
b. Offer ZDV + 3TC + IDV
   i. Skin-Increased Risk (eg., exposure to high titer of HIV, prolonged contact, extensive area involved, or skin is visibly compromised)—Blood
c. Offer ZDV + 3TC + IDV
   i. Initiate treatment promptly: within 1-2 hrs post exposure and continue for 4 weeks.
d. Increase fluid intake and do not ingest with fatty foods
5. Offer following sexual assault
a. Recommended baseline lab testing: HIV, RPR, anti-HBs, HbsAg. At two weeks: B CBC with diff, BUN, Cr, Liver Function tests.
b. Hepatitis B—more virulent than HIV
c. Risk of seroconversion
d. HBV vaccinated: 0%
e. HBV unvaccinated: 6-30% depending upon HbeAg status of source
f. Hepatitis C—risk of seroconversion 1.8%
g. Current Status of Blood Transfusion Supply and HIV
h. Donor history and blood tests using hepatitis profile, HIV 1-p24 Ag, anti-HIV

XIV. RISK OF DISEASE TRANSMISSION
A. HIV: 1/600,000-1/2,000,000 transfusions

B. HBV: 1/66,000 transfusions

C. HCV: 1/500,000 transfusions
HIV/AIDS

PEARLS

1. AIDS is defined by having an AIDS indicator condition or a CD4 count <200/m$^3$.

2. Primary HIV occurs 2-4 wks after exposure and presents as a mono like syndrome: rash, fever, HA, lymphadenopathy, organomegaly, arthralgia, myalgia, sore throat, leukopenia.

3. PCP is the most common life-threatening opportunistic infection in AIDS patient. ED evaluation: pulse ox, ABG, CBC, LDH, CXR (bilateral infiltrates, usual). Rx: TMP/SMX, add steroids if PaO$_2$ <70, A-a gradient > 35.

4. Increased incidence of TB in AIDS. PPD may be negative. Three or four drug therapy - INH, rifampin, pyrazinamide, ethambutol.

5. Disseminated MAI: most common systemic opportunistic bacterial infection. Abdominal cramping, diarrhea, lymphadenopathy, fever. CD4 < 50/_m$^3$. Rx: clarithromycin plus ethambutol or rifabutin.

6. Bacterial pneumonia occurs more frequently in AIDS. Organisms same as in normal host. Rx: same as for normal host.

7. Do CT scan and LP (if no mass effect) on all HIV patients with CNS symptoms.

8. Cryptococcal meningitis: most common fungal CNS infection. India ink positive, cryptococcal Ag positive, Rx: amphotericin B.

9. Toxoplasmosis: most common cause of focal encephalitis. May cause focal neurologic deficits, seizures. CT: ring enhancing lesion; Rx: clindamycin plus pyrimethamine and sulfadiazine.

10. Diarrhea is the most common complaint; found in 50-60% of AIDS patients. Causes: bacterial, MAI, viral, parasitic.

11. Acalculous cholecystitis can be caused by Cryptosporidium, Microsporidia, CMV.

12. Pancreatitis can be caused by direct viral attack, lymphadenopathy, gall bladder disease or drugs such as ddl, ddC, pentamidine.

13. Oral candida: the most common infection of the GI tract in the AIDS patient.
14. Herpes zoster may be seen heralding the onset of AIDS. Rx: Acyclovir.

15. CMV retinitis presents with blurred vision - blindness inevitable. Rx: ganciclovir.

16. Risk of seroconversion following a needlestick from an HIV positive individual - 0.3%.

17. Risk of HIV from blood transfusion - 1/600,000 - 2,000,000.

8/10
REFERENCES


