Dengue Fever

Etiology:

- It is a Flavivirus caused by one of four closely related viral serotypes (DEN-1, DEN-2, DEN-3, and DEN-4).
- It is transmitted to humans by the bite of mosquitoes, most commonly Aedes aegypti.
- Dengue Fever is sometimes called "break-bone fever" because it causes severe joint and muscle pain that feels like bones are breaking.

Vector:

Dengue fever is spread by the Aedes mosquito. There are numerous species of this mosquito, but the most prevalent is Aedes aegypti, found seasonally in the southern United States. All species are found year-round in Caribbean and African nations, and as such this is where the majority of infections occur.

Clinical Manifestations:

- There is a sudden onset of high fever (103.1-106.5 *F) lasting about 1-7 days, which then fades for 1-2 days. Mild fever soon recurs with secondary red and white patchy rashes, but the illness is clinically indistinguishable from Influenza, Measles, or Rubella.
- Acute phase of illness may last for 1 week followed by a 1-2 week recovery period characterized by nausea, vomiting, weakness, malaise, anorexia, change in taste sensation, and increased sensations to touch.
- Fever is usually accompanied by headache pain, localized frontally or behind the eyes.
- Myalgias occur after the onset of fever and severely affect the legs, joints, and lumbar portion of the spine. This may last for several weeks even after the fever subsides.
- Symptoms may be milder in children compared to adults.

Treatment:

- There is no actual treatment for DF. It resolves spontaneously in about 2 weeks.
- Supportive care involves drinking fluids, getting plenty of rest, and taking antipyretics. - OTC pain medications and acetaminophen are recommended, while aspirin is not advised.
- For severe DF leading to shock or coma, aggressive emergency treatment with fluid and electrolyte supplements can be life-saving.

EBOLA VIRUS

EPIDEMIOLOGY

- In US, Ebola is not endemic and there are 4 different strains
- **Ebola-Reston(EBO-R)** monkeys, chimpanzees, gorillas; **Ebola- Zaire(EBO-Z)**-Zaire 2003 to 2004: 35 cases: 29 died (total 1,086: 883 deaths); **Ebola-Sudan(EBO-S)**- Sudan 2004: 17 cases: all pts died (total 760: 414 deaths); **Ebola-Ivory Coast(EBO-C)**
- Kikwit, DRC 1995: infection rates child < adult; child less likely to come into direct contact w/ ill pts
- No gender predilections
- **Morbidity and mortality:** very high, and vary with strain of Ebola.
    - most highly lethal Ebola subtype is **EBO-Z**; reported mortality rate 89%.

ETIOLOGY: Ebola is Filoviridae family (*ssRNA in a helical nucleocapsid*)
Virus damages microvasculature resulting in ↑vascular permeability and viral hemorrhagic syndrome.

- highly infective in aerosol form: Travel/work in Ebola-endemic area, hx of exposure to tropical African forests; occurrence in caregivers (family/medical) or in family members who have prepared dead relatives for burial w/o proper barrier protection; use/reuse of contaminated medical equipment; animal care workers who care for primates; appearance of virus in a human, at start of outbreak, not determined

RESERVOIR: no reservoir id for any Filovirus but in 1996 Zaire: bats supported Ebola virus replication w/o dying: insectivorous bats (*Tadarida pumila)* and fruit bats (*Epomophorus wahlbergi)*

- Transmission from unknown host (bats) to humans/nonhuman primates

VECTOR: unknown relationship btwn specific exposure to potential vectors(arthropod, animal, or plant) and disease but hx of exposure to tropical African forests > hx of working w/in cities in same region, in pts w/ Ebola

CLINICAL MANIFESTATIONS

- Incubation period of virus in Ebola HF is about 2 to 21 days and onset is sudden
- Severe headache, arthralgias/ myalgias, fever with or w/o chills leading to maculopapular rash (desquamates in 15% of pts during convalescence), anorexia, and asthenia occur early in the disease.
- GI symptoms: abdominal pain, N/V/D soon follow, mucous membrane involvement, conjunctivitis, odynophagia/dysphagia, and bleeding from multiple sites in GI tract.
- findings upon PE depend on stage of disease in which patients present.
    - *Early*: fever, pharyngitis, and severe constitutional signs and symptoms, bilateral conjunctival injection; *Late*: expressionless faces, bleeding from intravenous puncture sites and mucous membranes, myocarditis, pulmonary edema; *Dying pt:* tachypneic, hypotensive, anuric, in a coma.

DIAGNOSIS: Early: CBC-Leukopenia (common); Dz progression: neutrophilia, ↓ in platelets (common); ELISA-Ag detection; IgM and IgG ELISA – anti-Ebola antibodies (later in dz/recovery); PCR, Indirect immunofluorescence testing is also used but is not confirmatory; Viral Isolation

TREATMENT: No specific treatment has been effective for the Ebola virus; Supportive therapy with attention to intravascular volume, electrolytes, nutrition, and comfort may be of benefit to the patient; You must contact the CDC and isolate the patient.

COMPLICATIONS Ocular complications including pain, photophobia, increased lacrimation, decreased visual acuity, and uveitis;; Survivors have developed the following late manifestations: Myalgias, Asymmetric and migratory arthralgias, Headaches, Fatigue, Bulimia, Amenorrhea, Hearing loss, Tinnitus, Unilateral orchitis, Suppurative parotitis

PROGNOSIS: Ebola infection highly lethal, prognosis poor (life span from onset of s/s to death 7-14 days) undergo systemic multi-organ failure or die (usually from a hypovolemic shock). Pt who survived for 2 weeks will often make a slow recovery (months) ; Pt who survived can continue to shed the virus for weeks to months. Males that recovered from the infection can spread the virus via semen.

Japanese Encephalitis

Vector:

- Mosquitoes *Culex tritaeniorhynchus*

Etiology:

- A virus which is an Arbovirus

Clinical Manifestations:

- Mild symptoms or no symptoms at all.
- Symptoms usually appear 6-8 days after the bite of an infected mosquito.
- Starts as a flu-like illness, with fever, chills, tiredness, H/A, and N/V. Confusion and agitation can also occur in the early stage.

Antibiotic Treatment:

- No specific treatment Tx of symptoms and complications.
- Vaccine available. Recommended only for persons who plan to travel in areas for 4 weeks or more.
- Mannitol (Osmitrol, Resectisol) -- decrease intracranial pressure.

PLAGUE – Any epidemic disease with a high death rate *GROUP 5*

Epidemiology

- Human plague in the United States has occurred as mostly scattered cases in rural areas (an average of 5 to 15 persons each year).

Etiology

- Bacterium called *Yersinia Pestis*. Fleas are infected with this bacterium → transmits to mammals (rodents)→human beings by bites from infected fleas or rodents
- Another way of transmitting plague is through direct contact.

Vector/Resevoir

- Male *Xenopsylla cheopis* (oriental rat flea) engorged with blood. This flea is the primary vector of plague in most large plague epidemics in Asia, Africa, and South America. Both male and female fleas can transmit the infection.
- A disease of rodents.

Clinical Manifestations

- Onset 3-7 days after the infx. Ssymptoms : chills, fever, diarrhea, H/A's, swelling of infected lymph nodes, as the bacteria replicate there.
- If untreated, the rate of mortality for bubonic plague is 50%.
- In septicemic plague, there is bleeding into the skin and other organs, which creates black patches on the skin.
- Bite-like bumps on the skin, commonly red and sometimes white in the center.
- People who die from this form of plague often die on the same day symptoms first appear.
- The pneumonic plague infects the lungs, and with that infection comes the possibility of person-to-person transmission through respiratory droplets.
- The incubation period for pneumonic plague is usually between 2—4 days, but can be as little as a few hours.
- The initial symptoms, of headache, weakness, and coughing with hemoptysis, are indistinguishable from other respiratory illnesses.

Diagnosis

- Bubonic plague should be suspected when a presents w/ symptoms or has a history of possible exposure to infected rodents, rabbits, or fleas.
- If small gram-negative and/or bipolar-staining coccobacilli are seen on a smear taken from affected tissues, e.g.:
- Bubo (bubonic plague)
- Blood (septicemic plague)
- Tracheal/lung aspirate (pneumonic plague)
- If immunofluorescence stain of smear or material is positive for the presence of *Yersinia pestis* F1 antigen.
- If a culture isolated is lysed by specific bacteriophage.
- *Agglutination testing must be shown to be specific to *Y.pestis* F1 antigen by hemagglutination inhibition.

Treatment

- Should begin as soon as the disease is diagnosed.
- Streptomycin 30 mg/kg IM BID x 7 days
- Chloramphenicol 25–30 mg/kg single dose, followed by 12.5–15 mg/kg QID
- Tetracycline 2 g single dose, followed by 500 mg QID x 7–10 days (not suitable for children)
- Gentamicin 2.5 mg/kg IV or IM BID x 7 days
- Doxycycline 100 mg (adults) or 2.2 mg/kg (children) PO BID
- Vaccination available for people working in or traveling to plague-affected areas of the world.

Complications

- Gangrene of your fingers and toes
- Severe shock
- Acute respiratory distress syndrome
- Septicemia
- Death
- Meningitis
- Death

Prognosis

- Without diagnosis and treatment, the infection can be fatal in 1-6
- days; mortality in untreated cases may be as high as 95%.
- The death rate is 1-15% for those treated for bubonic plague.
- A person with primary or secondary septicemic plague (infection is active in the bloodstream and the patient has shock symptoms) has a 40% death rate, even when treated.
- Pneumonic plague has 100% death rate if not treated within the first 24 hours.

YELLOW FEVER : Yellow fever is a viral hemorrhagic fever (VHF) caused by the Flavivirus yellow fever virus.

Epidemiology: The WHO estimates the number of cases at 200,000 cases per year, with the highest proportion in sub-Saharan Africa. Yellow fever has no specific predilection; however, affected areas include 9 countries of South America, some tropical areas of Africa, and several of the Caribbean islands.

Etiology: single-stranded, RNA-enveloped Flavivirus

Vector: *Aedes aegypti mosquito*

Reservoir: Monkeys

Clinical manifestations: Acute Phase is the first phase after the incubation period of 3-6 days. In this phase clinical signs may be nonspecific. Fever, bilious vomiting, relative bradycardia, and conjunctival injection may be the only findings. In the toxic phase of illness, other findings develop, including jaundice. Hepatomegaly and right upper quadrant tenderness to palpation may be noted. Signs of bleeding dyscrasias with petechiae, ecchymoses, epistaxis, and oozing from gums and venipuncture sites may be observed. Arrhythmias are common, as are hypotension and shock that are frequently unresponsive to fluid resuscitation.

Diagnosis: Detection of yellow fever antigen by monoclonal enzyme immunoassay in serum specimens and detection of viral genome sequences in tissue or blood using PCR

Treatment: No specific treatment exists for yellow fever. Supportive care is critical.

Vaccine: Yellow fever vaccine (YF-VAX) -- This vaccine should be administered to residents of and travelers to endemic areas.

Complications: Liver failure, renal failure, pulmonary edema, myocarditis, secondary bacterial infections, hemorrhage or disseminated intravascular coagulation, encephalitis (rare), shock and death.

Prognosis: Yellow fever ranges in severity from a self-limited infection to hemorrhagic fever that carries a 50% mortality rate. Fatality rates are higher in the young. Early appearance of jaundice (day 3) indicates a poor prognosis. Up to 50% of patients who progress to the toxic phase die. Individuals who survive the toxic phase may experience renal failure. Convalescence with symptoms of weakness and fatigue may last up to 3 months.

Ehrlichiosis
This is a disease caused by a bacteria in the Rickettsiae family (same family as rocky mountain spotted fever) There are two primary forms that have been identified; Human monocytic ehrlichiosis (HME) and Human granulocytic ehrlichiosis (HGE). HME is caused by the bacteria ***Ehrlichia chaffeensis***, while HGE is caused by the bacteria ***Eherlicia Phagocytophilia***.

The epidemiology of ehrliciosis is not well documented. The disease itself has only been identified within the past 15 years and there is only about 7-8 years worth of data available from the CDC. However, in 2002, there were about 750 total cases reported nationwide with HGA>HME by about 2:1

The two bacteria mentioned above are carried by a vector. To date, the following have been identified as vectors carrying erhliciosis: the Lone star tick, the American Dog Tick, and the Deer Tick. The incubation time is upwards of 9 days before infection occurs.

Clinically, findings are non-specific and may mimic a flu virus infection. Fever, Chills, Headache, Nausea. and Muscle Aches are the most commonly reported symptoms. Occasionally, a fine petichial rash may develop at any location (not dependent upon location of bite)

Diagnosis consists of the following: Neutropenia, Thrombocytopenia, and high Transaminase on LFT. In addition, a granulocyte stain will allow visualization of ehrliciosis cells inside the WBC's. A fluorescent antibody stain may allow detection of the specific bacteria that caused the disease.

Tetracycline antibiotics are the only therapy demonstrated to treat ehrlichiosis. There is no other treatment available for children or for those pregnant.

Complications can include damage to the lungs, kidneys, or other organs. Death is extremely rare in those that are diagnosed and treated. Overall prognosis is excellent.Improvement of s/s is often seen within 24 hours and nearly all patients have full recovery after 3 weeks.

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Babeiosis

Epidemiology:

-Highest prevalance near the east coast. America reports only 5-6 cases peryear.
95% of all victims recover from the infection.
-In the U.S. Babesiosis is endemic on the east coast islands and southern Connecticut. The vectors are nymphal Ixodes ticks, and the agents are protozoan parasites of red blood cells.

Etiology:

Babesia is a rare, blood-born disease caused by *Babesia microti* and *Babesia divergens* parasites – both are members of the protozoan kingdom.

Vector:

The *B. microti* and *B. divergens* are transmitted by *Ixodes dammini* and *Ixodes ricinus*, respectively.

Reservior:

Host for *B. microti* – Rodents
Host for *B. divergens* – Cattle

Clinical Manifestations:

- An infected person generally will not show signs of the disease for about 1-4 weeks.
- After this incubation period has ended, a person will show non-specific disease signs and symptoms such as malaise, fever, headache and chills, jaundice, a slightly enlarged spleen, sweating, and weakness.
-Significantly, many of these symptoms mimic those of malaria.
-The best way to distinguish between the symptoms of the two infections is that babesia does not have the periodic fever that characterizes malaria.

Diagnosis:

-Giemsa stained thick and thin blood films

Treatment:

-Atoraquone and azithromycin

Complications:

-Renal failure in immunocompromised
-Splenectomized
-Elderly with increased mortality, Shock,ARDS, Relapse, Hemoglobinuria, Jaundice, Coma due to severe sepsis.
-Coinfection with other tick transmitted diseases such as Lyme's disease causes prolongation and severity of symptoms

Prognosis:

-In America, infections are usually not fatal - there are usually only 5-6 infections per year, but approximately 95% of infected patients recover completely. Asplenic and immunocompromised patients are at higher risk for kidney and renal failure and have a higher fatality rate, as death usually is secondary to renal failure.

Random Fact: There's a closely related infection that only dogs get that is much more deadly. It is only in dogs and it doesn't co-infect humans.