Objectives

- At the end of the presentation, the participant will be able to:
  - List the pathogens that cause neonatal sepsis
  - Discuss the epidemiology of the common pathogens of sepsis
  - Know the agents that are responsible for congenital infections
  - Discuss the clinical features of the causes of congenital infections

NEONATAL INFECTIONS

- Bacterial neonatal sepsis
- Viral infections mimicking bacterial sepsis
- In-utero infections (congenital infections)
NEONATAL SEPSIS

- Bacterial vs viral
- Early-onset vs Late-onset
- Perinatal vs Nosocomial vs Community acquisition
- Clinical syndromes similar regardless of etiology

BACTERIAL NEONATAL SEPSIS

- Overall incidence 1-5 per 1000 live births
- Early-onset sepsis
  - symptom onset within 6 days of life
  - maternal complications common
  - vertical transmission of organisms colonizing genital tract

BACTERIAL NEONATAL SEPSIS

- Late-onset sepsis
  - symptom onset after 6th day of life
  - 2 distinct groups of neonates
    - healthy newborns who have been discharged to home
    - high-risk hospitalized neonates who develop hospital-associated infection
QUESTION #1

- Of the following, the organism LEAST likely to cause early-onset neonatal sepsis is:
  - A. *Listeria monocytogenes*
  - B. *Group B Streptococci*
  - C. *Streptococcus pneumoniae*
  - D. *E. coli*
  - E. *viridans streptococci*

ETIOLOGY OF EARLY-ONSET BACTERIAL SEPSIS

- *Group B Streptococci*
  - dramatic decline since implementation of intrapartum prophylaxis
- *Escherichia coli*
  - probably most common now
- *Listeria monocytogenes*
- Less Common
  - *viridans streptococci*
  - non-typeable *Haemophilus influenzae*

QUESTION #2

- All of the following are important causes of late-onset neonatal sepsis, EXCEPT:
  - A. *Streptococcus pyogenes*
  - B. Coagulase-negative
    *Staphylococci*
  - C. *Staphylococcus aureus*
  - D. Gram-negative bacilli
  - E. *Candida albicans*
ETIOLOGY OF LATE-ONSET NEONATAL SEPSIS

- Healthy newborns previously discharged from hospital
  - Group B Streptococci
  - L. monocytogenes
  - E. coli
  - Salmonella species
- Less frequent causes:
  - H. influenzae- nontypeable
  - Neisseria meningitidis
  - Streptococcus pneumoniae

ETIOLOGY OF LATE-ONSET SEPSIS*

- High risk newborns (VLBW infants in NICU)
  - Coagulase-negative Staphylococci--48%
    - Most common cause of late-onset sepsis in hospitalized high-risk neonates
  - Gram-negative bacilli (E. coli, Klebsiella sp, Enterobacter sp, Pseudomonas sp)—20%
  - S. aureus—8%
  - Candida albicans—12%
  - Enterococci—3%


BACTERIAL NEONATAL SEPSIS: RISK FACTORS FOR VLBW INFANTS

- Invasive procedures
- Mechanical ventilation
- Indwelling intravascular catheters
- Total Parenteral Nutrition
- Widespread use of broad-spectrum antibiotics
- H2 blockers
CLINICAL MANIFESTATIONS OF NEONATAL SEPSIS

- Signs and symptoms often subtle and nonspecific
- Features of neonatal meningitis often indistinguishable from sepsis
- Noninfectious illnesses have similar features
  - RDS; Congenital heart disease; Metabolic disorders
- Most neonatal pathogens produce similar symptoms

GROUP B STREPTOCOCCI: MATERNAL COLONIZATION

- 15-40% of all pregnant women are colonized with GBS
  - Genital and GI tracts
  - Highest: African-Americans, <20yo
  - Lowest: Asian, Mexican-Americans
- Colonization
  - Constant or intermittent
  - Colonization during one pregnancy does not predict colonization in subsequent pregnancy

Mother to Infant Transmission of GBS

- GBS colonized mother
  - 50% Colonized newborn
  - 50% Non-colonized newborn
  - 98% Asymptomatic
- Colonized newborn
  - 2% Early-onset sepsis, pneumonia, meningitis

CDC Prevention Guidelines, 2010
Additional Risk Factors for Early-onset GBS Disease

- Obstetric risk factors:
  - Preterm delivery
  - Prolonged rupture of membranes
  - Infection of the placental tissues or amniotic fluid / fever during labor
  - GBS in the mother’s urine during pregnancy (marker for heavy colonization)
  - Previous infant with GBS disease
  - Low maternal levels of anti-GBS antibodies
- Demographic risk factors
  - African American
  - Young maternal age

CDC Prevention Guidelines, 2010

Question #3: Which of the following is a TRUE statement concerning early-onset GBS neonatal sepsis?

- A. Pneumonia and apnea are the most common clinical features
- B. Meningitis is present in 80% of cases
- C. The case-fatality rate is lower than with late-onset GBS infection
- D. Septic shock occurs in 75% of patients
- E. The mean age of onset is 72 hours of life

GBS Infection: Early vs Late Onset

<table>
<thead>
<tr>
<th></th>
<th>Early-onset</th>
<th>Late-onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incidence</td>
<td>0.3/1000</td>
<td>0.3/1000</td>
</tr>
<tr>
<td>Mean age at onset</td>
<td>8 hours</td>
<td>27 days</td>
</tr>
<tr>
<td>Incidence of prematurity</td>
<td>Increased</td>
<td>Not increased</td>
</tr>
<tr>
<td>Obstetric complications</td>
<td>Common</td>
<td>Unusual</td>
</tr>
<tr>
<td>Manifestations</td>
<td>Pneumonia (40%)</td>
<td>Bacteremia</td>
</tr>
<tr>
<td></td>
<td>Meningitis (5-10%)</td>
<td>Meningitis</td>
</tr>
<tr>
<td></td>
<td>Septic Shock (25%)</td>
<td>Bone/joint/skin</td>
</tr>
<tr>
<td>Mortality rate</td>
<td>10-15%</td>
<td>2-6%</td>
</tr>
</tbody>
</table>
NEONATAL SEPSIS/MENINGITIS DUE TO *E. coli* INFECTION

- Incidence approx 1 per 1000 live births
- Most cases early-onset
  - Ki capsular antigen present in 80% of cases of meningitis
- Vertical transmission major route of transmission
- Infants with Galactosemia particularly susceptible to *E. coli* infection
- CFR high (20%) and sequelae in 30-50%

NEONATAL LISTERIOSIS

<table>
<thead>
<tr>
<th></th>
<th>Early-onset</th>
<th>Late-onset</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age at onset</td>
<td>At birth</td>
<td>1-8 weeks</td>
</tr>
<tr>
<td>Birthweight</td>
<td>Often LBW</td>
<td>Usually term</td>
</tr>
<tr>
<td>Obstetric</td>
<td>Common (amnionitis, brown staining)</td>
<td>Infrequent</td>
</tr>
<tr>
<td>complications</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Source of isolate</td>
<td>Blood(75%)</td>
<td>CSF(90%)</td>
</tr>
<tr>
<td>Fatality rate</td>
<td>25%</td>
<td>5%</td>
</tr>
</tbody>
</table>

NEONATAL LISTERIOSIS

- Maternal prodromal illness common-65%
- Elevated monocyte count present in 50% of bacteremic infants
- Monocytes not typically found in CSF of infants with meningitis
- Erythematous rash with 1-2mm pale nodules on skin and pharynx occasionally seen (granulomas on path)
  - granulomatosis infantepticum
NEONATAL BACTERIAL SEPSIS: DIAGNOSIS

- Isolation of organism from blood or CSF definitive
- Latex Particle agglutination for GBS and E. coli limited by poor sensitivity and specificity
- Hematologic abnormalities often accompanying sepsis
  - elevated ratio immature to total neutrophils (>0.2)
  - neutropenia
  - elevated neutrophil count
  - thrombocytopenia

EMPIRIC THERAPY FOR NEONATAL SEPSIS

- Early-onset sepsis
  - Ampicillin + Aminoglycoside
  - Cephalosporins should not be used empirically
- Late-onset sepsis
  - VLBW infants in the NICU
    - Oxacillin or Vancomycin + Aminoglycoside or 3rd gen cephalosporin
    - “Community” acquired
      - Ampicillin + 3rd gen cephalosporin

TREATMENT OF NEONATAL LISTERIOSIS

- Ampicillin and Gentamicin initially (bactericidal), then ampicillin alone
- Cephalosporins NOT active against L. monocytogenes
- 2-3 week duration
TREATMENT OF GRAM-NEGATIVE SEPSIS AND MENINGITIS

- Ampicillin and Gentamicin
  - Initial empiric therapy
- Cefotaxime safe and effective alternative and often used in combo with aminoglycoside for meningitis
- Even with appropriate therapy, CSF cultures remain positive for at least 2-3 days after initiation of therapy
- Failure to sterilize CSF should prompt search for brain abscess or other complication
- 3-week course recommended for meningitis

BRAIN ABSCESS AND GRAM-NEGATIVE MENINGITIS

- Propensity to cause Brain Abscess
  - *Citrobacter koseri* (formerly *diversus*)
  - *Enterobacter sakazakii*
  - *Serratia marcescens*

PREVENTION OF EARLY-ONSET GBS SEPSIS

- 2002 and 2010 CDC guidelines
  - Based on data showing that the screening method was >50% more effective than the risk-factor strategy
Rate of Early- and Late-Onset GBS, 1990-2008

Key Prevention Strategies Remain Unchanged in 2010

- Universal screening of pregnant women for GBS at 35-37 weeks gestational age
- Intrapartum antibiotic prophylaxis for:
  - GBS positive screening test
  - GBS colonization status unknown with
    - Delivery <37 weeks
    - Temperature during labor > 100.4°F (>38.0°C)
    - Rupture of membranes >18 hours
  - Previous infant with GBS disease
  - GBS in the mother’s urine during current pregnancy

CDC Prevention Guidelines, 2010

Key Prevention Strategies Remain Unchanged in 2010

- Penicillin preferred drug for IAP
  - Ampicillin acceptable alternative
  - Cefazolin preferred for penicillin-allergic at low risk of anaphylaxis

CDC Prevention Guidelines, 2010
Intrapartum GBS Prophylaxis Not Indicated

• Colonization with GBS during a previous pregnancy
  • Unless another indication during the current pregnancy
• GBS bacteriuria during a previous pregnancy
  • Unless another indication during the current pregnancy
• Negative vaginal and rectal GBS screening test during the current pregnancy
  • Regardless of intrapartum risk factors
• Cesarean delivery performed before labor onset on a woman with intact amniotic membranes
  • Regardless of maternal GBS test status
  • Regardless of gestational age

CDC Prevention Guidelines, 2010

QUESTION #4

Of the following, the virus least likely to cause symptoms at birth that mimic bacterial neonatal sepsis is:
• A. Herpes simplex virus
• B. Human immunodeficiency virus (HIV)
• C. Cytomegalovirus
• D. Enterovirus
• E. Adenovirus

ETIOLOGY OF NEONATAL VIRAL SEPSIS

• Herpes simplex virus types I and II
• Enteroviruses
  • Echoviruses
  • Coxsackie
• Cytomegalovirus
• Respiratory viruses
  • RSV
  • Influenza
  • Adenovirus
Neonatal HSV Infections

- Most occur from infected maternal genital tract at delivery
- Signs of infection by 4-5 weeks of age
- Risk largely influenced by maternal antibody status
  - 50% transmission with primary infection
  - 5% transmission with recurrent infection

Neonatal HSV Infections

- High prevalence of asymptomatic maternal infection
- Thus, most infants (>75%) with neonatal HSV are born to asymptomatic mothers who have no past history of genital HSV infection or clinical findings during pregnancy

Neonatal HSV Infections

<table>
<thead>
<tr>
<th>Type of Infection</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin, Eyes, Mucus Membranes (SEM)</td>
<td>45%</td>
</tr>
<tr>
<td>Disseminated Infection</td>
<td>25%</td>
</tr>
<tr>
<td>CNS Infection</td>
<td>30%</td>
</tr>
</tbody>
</table>
NEONATAL HSV INFECTION

- Skin, eyes and/or mouth (SEM)
  - 7-14 days of life
  - Vesicles—often at sites of trauma (scalp electrode site)
  - Conjunctivitis
  - No mortality with treatment
    - 5% morbidity with treatment
    - 75% progress without treatment

Neonatal Disseminated HSV Infection

- Disseminated disease
  - 5-12 days of life
  - 56% have skin lesions
  - Lesions often absent at presentation
  - Mimics bacterial sepsis
    - DIC
    - Pneumonia
    - Hepatitis
    - CNS involvement (60-70%)
      - Seizures in 22%

- Disseminated disease
  - Rapid deterioration
  - Unremitting shock
  - Progressive liver failure
  - Bleeding
  - Respiratory failure
  - 30% mortality with treatment
Neonatal HSV Encephalitis

- Encephalitis (CNS)
  - 16-19 of life
  - 45-63% have skin lesions
  - Seizures (generalized or focal)
  - Lethargy
  - Irritability
  - Poor feeding
  - Temp instability

NEONATAL HERPES SIMPLEX INFECTIONS: DIAGNOSIS

- Diagnosis difficult in absence of vesicular lesions - need high clinical suspicion
- Viral Culture
  - cutaneous lesion
  - nasopharynx
  - CSF
  - conjunctiva
  - urine
- Direct Fluorescent Antibody
  - rapid diagnosis
  - requires vesicular lesions
- Polymerase Chain Reaction-CSF

Treatment of Neonatal HSV Infections

- Intravenous acyclovir treatment of choice
  - 20mg/kg/dose q8h IV standard dose
  - Monitor for neutropenia (20%) and nephrotoxicity (renal tubular crystallization with dehydration)
- 21-day course for disseminated or CNS infections
- 14-day course for SEM disease
Outcome of Neonatal HSV Infections

<table>
<thead>
<tr>
<th>Site of Disease</th>
<th>No Therapy</th>
<th>With Antiviral Therapy</th>
<th>Normal Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disseminated encephalitis</td>
<td>9%</td>
<td>5%</td>
<td>10%</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>50%</td>
<td>4%</td>
<td>91%</td>
</tr>
<tr>
<td>Skin, eyes, and mucous membranes</td>
<td>5%</td>
<td>0%</td>
<td>6%</td>
</tr>
<tr>
<td>Mortality</td>
<td>10%</td>
<td>4%</td>
<td>96%</td>
</tr>
</tbody>
</table>

Corey L. Wald A. NEJM 2009;361:1376

NEONATAL ENTEROVIRAL INFECTIONS

- Echoviruses 9, 11, 30 and Coxsackie B viruses most common today
- Most neonatal infections mild and non-specific, but 20% severe and life-threatening
- Diagnosis very difficult to distinguish from bacterial or HSV infection

NEONATAL ENTEROVIRAL INFECTIONS: CLINICAL FEATURES

- Macular or maculopapular rash—40%
- Hepatitis/Hepatic necrosis
- Myocarditis
- Meningoencephalitis
- Maternal history of viral illness
- Lack of obstetrical complications
- Summer and fall
- Isolation of virus from NP, throat, stool, CSF confirms Dx
IN UTERO CONGENITAL INFECTIONS

COMMON MANIFESTATIONS OF IN UTERO INFECTIONS

- General Characteristics
  - Manifestations present at birth or shortly thereafter
  - Presence of congenital defects (CHD, ocular abnormalities, calcifications, etc)

- Specific Features
  - Small for Gestational age
  - CNS: microcephaly, seizures, cerebral calcification
  - Skin: jaundice, petechiae, purpura, vesicles
  - Eye: chorioretinitis, cataracts, microphthalmia
  - Heart: PDA, PS
  - Abdomen: HSM, hepatitis
  - Lung: pneumonitis
  - Musculoskeletal: bone lesions, limb hypoplasia
QUESTION #5

Of the following, the most common agent causing congenital infection is:

- A. *Toxoplasma gondii*
- B. Cytomegalovirus
- C. Rubella
- D. Parvovirus B 19
- E. Varicella-zoster virus

Question #6: Which is a true statement concerning congenital cytomegalovirus infection?

- A. Chorioretinitis is the most common clinical manifestation
- B. 1% of all infants born have CMV infection
- C. Fetal infection occurs only after primary maternal infection
- D. Infants with asymptomatic infection have no risk of long-term sequelae
- E. Serology represents the most reliable diagnostic test

**CMV: EPIDEMIOLOGY**

- Most common congenital infection
- 1% of all newborn infants have congenital CMV infection
- Virus transmitted from both immune mothers as well as non-immune mothers
  - Severe fetal damage occurs almost exclusively w/º infection
- Infection can occur prenatally, natally, or postnatally
**SIGNS OF PRENATAL CMV INFECTION IN THE NEWBORN**

<table>
<thead>
<tr>
<th>Finding</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asymptomatic</td>
<td>90%</td>
</tr>
<tr>
<td>Symptomatic</td>
<td>10%</td>
</tr>
<tr>
<td>Petechiae</td>
<td>76%</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>77%</td>
</tr>
<tr>
<td>Jaundice</td>
<td>67%</td>
</tr>
<tr>
<td>HSM</td>
<td>60%</td>
</tr>
<tr>
<td>SGA</td>
<td>50%</td>
</tr>
<tr>
<td>CT Calcifications</td>
<td>50%</td>
</tr>
<tr>
<td>Microcephaly</td>
<td>53%</td>
</tr>
<tr>
<td>Retinitis</td>
<td>10%</td>
</tr>
<tr>
<td>Seizures</td>
<td>7%</td>
</tr>
</tbody>
</table>

**FEATURES OF CONGENITAL CMV IN SYMPTOMATIC INFANTS**

- Elevated transaminases-83%
- Thrombocytopenia (<100K)-77%
- Conjugated hyperbilirubinemia-81%
- Hemolysis-51%
- Increased CSF protein (>120mg/dL)-46%

**LONG-TERM OUTCOME OF CONGENITAL CMV INFECTION**

- Symptomatic infants
  - Death (30%)
  - Sensorineural hearing loss(58%)
  - IQ<70 (55%)
  - Microcephaly, seizures, paresis (52%)
  - Chorioretinitis (20%)
- Asymptomatic infants
  - Sensorineural hearing loss (7%)
  - IQ < 70 (4%)
  - Microcephaly (3%)
  - Chorioretinitis (2.5%)
LABORATORY DIAGNOSIS OF CONGENITAL CMV INFECTION

• Definitive
  - Isolation of virus from urine or saliva by three weeks of age
• Not Useful
  - CMV IgG and IgM antibody
    - False positives and negatives common

QUESTION #7

A newborn infant has microcephaly, is SGA, has a “blueberry muffin” rash, and bilateral cataracts. The most likely congenital heart lesion associated with this infection is:

• A. PDA
• B. ASD
• C. TGA
• D. Tricuspid atresia
• E. Coarctation of the aorta

CONGENITAL RUBELLA INFECTION

• Almost all cases due to maternal primary infection
• Overall risk to fetus 20% (70% in first trimester)
• Imported cases from Asia and Europe
• No congenital infection seen with inadvertent maternal vaccination during pregnancy
• Diagnosis
  - Viral culture from NP, urine, cataract
CONGENITAL RUBELLA: CLINICAL MANIFESTATIONS

- Most infants asymptomatic at birth
- Cataracts
- Blueberry muffin spots (dermal erythropoesis)
- CHD (PDA, PS)
- “Salt and pepper” retinopathy
- IUGR and postnatal growth restriction
- Reticuloendothelial (HSM, jaundice)

QUESTION #8

Which of the following is True regarding congenital Toxoplasma infection?

- A. The incidence is constant despite geographic location
- B. Prenatal diagnosis is not possible
- C. Treatment of infected pregnant women is not recommended
- D. Neurological and visual problems become apparent in the majority of infected asymptomatic infants

CONGENITAL TOXOPLASMOSIS

- Exposure to oocytes: cat feces and ingestion of raw beef major sources
- Fetal infection occurs only with maternal primary infection
- Incidence 1/1000-10000 live births
**CONGENITAL TOXOPLASMOSIS: FETAL INFECTION RATES**

<table>
<thead>
<tr>
<th>Trimester</th>
<th>1st</th>
<th>2nd</th>
<th>3rd</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall rate of infection</td>
<td>15%</td>
<td>30%</td>
<td>60%</td>
</tr>
<tr>
<td>Subclinical disease</td>
<td>18%</td>
<td>67%</td>
<td>90%</td>
</tr>
<tr>
<td>Mild</td>
<td>6%</td>
<td>18%</td>
<td>10%</td>
</tr>
<tr>
<td>Severe</td>
<td>41%</td>
<td>8%</td>
<td>0</td>
</tr>
<tr>
<td>Stillborn/perinatal death</td>
<td>35%</td>
<td>7%</td>
<td>0</td>
</tr>
</tbody>
</table>

**CONGENITAL TOXOPLASMOSIS: CLINICAL FEATURES**

- Majority of infected neonates are asymptomatic
- 80% Develop eye/neuro disease by adulthood
- Classic Triad
  - Chorioretinitis-86%
  - Hydrocephalus-20%
  - Cerebral Calcifications-37%
- Cerebral Calcifications
  - Distributed throughout the brain-unlike CMV
- Chorioretinitis
  - Focal necrotizing retinitis
  - Can be recurrent and progressive
  - May develop later in life without any other features of congenital infection

**CONGENITAL TOXOPLASMOSIS: DIAGNOSIS**

- **Prenatal**
  - Toxo DNA in amniotic fluid or fetal blood
  - Isolating parasite by mouse inoculation or tissue culture
  - Serial fetal US—look for increased size of lateral ventricles
- **Postnatal**
  - T. gondii DNA by PCR amniotic fluid, fetal blood, blood, CSF
  - Histopathology placenta, infected organ/tissue
  - Mouse inoculation assays of infant’s blood, placenta, umbilical cord
  - Serology- IgG, M, A, E on Mom and infant
CONGENITAL TOXOPLASMOSIS: TREATMENT

• Mother
  - Spiramycin to decrease transmission
  - Pyramethamine and sulfadiazine if fetal infection confirmed after 17 weeks gestation

• Infant
  - Decreases severity of disease and frequency of sequelae
  - Pyrimethamine and sulfadiazine
  - Therapy continued for 1 year

CONGENITAL SYPHILIS

• 30-40% of infected fetuses are stillborn
• Of infected neonates who are live-born, 70% are asymptomatic at birth and are identified by prenatal maternal screening
• Because fetus acquires infection via hematogenous route, widespread involvement (rather than primary stage) is usual

QUESTION #9

A 1-week old infant develops a copious bloody nasal discharge, lymphadenopathy, hepatomegaly and hemolytic anemia. Which of the following is the most likely additional feature in this infant?

- A. hydrocephalus
- B. periostitis
- C. limb hypoplasia
- D. seizures
- E. hydrops
UNIQUE FEATURES OF CONGENITAL SYPHILIS

- Generalized lymphadenopathy more common than other congenital infections
- Coombs-negative hemolytic anemia
- Snuffles (rhinitis)-in 25% of infants
- Exanthem
  - maculopapular rash with scaling and desquamation most common
  - vesicobullous lesions (pemphigus syphiliticus)

UNIQUE FEATURES OF CONGENITAL SYPHILIS

- Bony lesions-may be most frequently encountered manifestation
  - osteochondritis
  - osteomyelitis
  - periostitis
- CNS manifestations
  - pleocytosis; high protein, reaginic antibody
- Chorioretinitis
  - “salt & pepper”-like Rubella

| Image of an X-ray showing bone lesions, possibly related to congenital syphilis. |
INDICATIONS FOR EVALUATION OF CONGENITAL SYPHILIS

- Mother with positive non-treponemal tests confirmed by a positive treponemal test and:
  - Untreated or inadequately treated syphilis
  - Treatment in pregnancy with non-penicillin regimen
  - Lack of expected decrease in non-treponemal antibody titer after treatment
  - Treatment < 1 month before delivery
  - Treatment not documented
  - Insufficient follow-up to assess response

Evaluation for Congenital Syphilis

- PE
- Quantitative nontreponemal (RPR) and treponemal (FTA-ABS) test on infant's serum
- Antitreponemal IgM if available
- CSF for VDRL, cell count, protein
- Long-bone radiographs
- CBC, platelets
- Chest radiography, LFT's
  - as clinically indicated

Congenital Syphilis: Who needs Treatment?

- Infants with proven or probable disease
- Infants who warrant evaluation in which infection cannot be ruled out
- Infants whose follow-up cannot be assured
- Infants whose infected mothers can’t be treated, treated inadequately, or treated within 1 month of delivery
- Infants of mothers not having 4-fold decrease in titer
Treatment for Congenital Syphilis

- Aqueous Penicillin G for 10-14 days preferred therapy for proven or presumed infection
- Procaine penicillin (IM) may also be used
  - CSF concentrations may not be adequate
- Single dose benzathine Penicillin
  - recommended by some experts for:
    - asymptomatic infants with normal evaluation and whose follow-up can be assured, but whose mothers have not been treated adequately or do not have 4-fold decrease in titer

Question #10

The congenital infection most associated with hydrops fetalis is:

- A. Varicella-zoster virus
- B. HIV
- C. Human herpes virus 6 (HHV-6)
- D. Parvovirus B19
- E. Borrelia burgdorferi

EPIDEMIOLOGY OF PARVOVIRUS B19 INFECTIONS

- About 50% of women are seropositive for the virus prior to pregnancy
- Likelihood of infection after a close exposure estimated to be 30-50%
- Estimates of fetal loss following infection during pregnancy range from 2-6%
- Thus, overall risk of fetal loss due to this virus is 1-2%
CONGENITAL PARVOVIRUS B19 INFECTION

- Consequences of maternal parvovirus infections
  - Asymptomatic newborn
  - IUGR
  - Hydrops fetalis
    - severe anemia
    - high output cardiac failure
    - extramedullary hematopoiesis
  - Stillbirth
  - Isolated pleural or pericardial effusions

Answers to Questions

- 1. C
- 2. A
- 3. A
- 4. B
- 5. B
- 6. B
- 7. A
- 8. D
- 9. B
- 10. D