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% Non-colonized newborn
- 50% Colonized newborn
- 98% Asymptomatic
- 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

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 (1-4/1000 before IAP)</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 complications</td>
<td>Common (amnionitis, brown staining)</td>
<td>Infrequent</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 infantisepticum
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
  - 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

<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
  - 58% have skin lesions
    - Lesions often absent at presentation
  - Mimics bacterial sepsis
    - DIC
    - Pneumonia
    - Hepatitis
    - CNS involvement (60-70%)
      - Seizures in 22%
Neonatal Disseminated HSV Infection

- **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 crystalization 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>w/ Antiviral Therapy</th>
<th>Normal Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disseminated</td>
<td>85</td>
<td>51</td>
<td>Rare</td>
</tr>
<tr>
<td>Central nervous system</td>
<td>32</td>
<td>6</td>
<td>Rare</td>
</tr>
<tr>
<td>Skin, eyes, meninges</td>
<td>5</td>
<td>6</td>
<td>Rare</td>
</tr>
</tbody>
</table>

* Data on patients who did not receive therapy per 83 and data on patients who received intravenous glycemic therapy per 3.

A normal outcome is defined as the achievement of developmental milestones within 24 months after infection. Skin, eye, and meningeal infections will progress to meningitis or disseminated disease in the absence of antiviral therapy in a high proportion of infants.

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: icterus, 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/1º infection
• Infection can occur prenatally, nataly, or postnatally

SIGNS OF PRENATAL CMV INFECTION IN THE NEWBORN

<table>
<thead>
<tr>
<th>Finding</th>
<th>Frequency</th>
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</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 (29%)
- 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 erythropoiesis)
- CHD (PDA, PS)
- “Salt and pepper” retinopathy
- IUGR and postnatal growth restriction
- Reticuloendothelial (HSM, jaundice)

CDC Public Health Image Library
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/nerve 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

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
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

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
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
- Qualitative 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

Of the following, 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

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