بسم الله الرحمن الرحيم
CONGENITAL INFECTIONS

Prepared by:

MOHAMMED KAREM

6TH stage

College of medicine

Diyala University

Supervised by Dr. Dawood Alazawi

6/5/2012
CONGENITAL INFECTIONS

OVERVIEW:-

- Definition
- Epidemiology and pathophysiology
- Manifestations of congenital infections
- Diagnosis of congenital infections
- Prevention and treatment
Congenital infections are infections that are passed from a pregnant woman to her fetus. That can lead to severe fetal anomalies or even fetal loss. They are a group of viral, bacterial, and protozoan infections that gain access to the fetal bloodstream transplacentally via the chorionic villi. Hematogenous transmission may occur at any time during gestation or occasionally at the time of delivery via maternal-to-fetal transfusion.
Congenital infections are include the following infections:-

- **1-TORCH.**
  - **T** – *Toxoplasmosis* / *Toxoplasma gondii*.
  - **O** – Other infections(*Coxsackievirus*, *Syphilis*, *Varicella-Zoster Virus*, *HIV*, and *Parvovirus B19*).
  - **R** – *Rubella*.
  - **C** – *Cytomegalovirus*.
  - **H** – *Herpes simplex virus*.
STARCH

(S) Syphilis, (T) toxoplasmosis, (A) acquired immunodeficiency syndrome, (R) Rubella, (C) Cytomegalovirus, (H) Herpes.....

CHARTS

Cytomegalovirus, Herpes viruses, AIDS viruses

Syphilis, Toxoplasmosis, Rubella.
**Syphilis**

- **Syphilis** is a **sexually transmitted infection** caused by the **spirochete** bacterium *Treponema pallidum* subspecies *pallidum*.

- that can cause, in later stages, damage to the muscles, **brain** and other major internal organs.

- Untreated maternal syphilis can result in:-
  - stillbirth/perinatal death.
  - premature delivery.
  - about half of survivors have long-term neurological sequelae.
CONGENITAL SYPHILIS

Mainly result from:-
- transplacental infection.
- Contact with a chancre at birth.
- Haematogenous infection through out pregnancy.

2/3 of affected live-born infants are asymptomatic at birth.

Clinical symptoms split into early or late (2 years is cutoff).

3 major classifications:
  i. Fetal effects
  ii. Early effects
  iii. Late effects
CLINICAL MANIFESTATIONS

- Fetal:
  - Stillbirth
  - Neonatal death
  - Hydrops fetalis
  - Intrauterine death in 25%.
  - Perinatal mortality in 25-30% if untreated.
Early congenital (typically 1st 5 weeks):

- Cutaneous lesions (palms/soles).
- maculo-papular eruption over buttocks and lower torso, palms and soles.
- bullous eruptions.

- generalised lymphadenopathy and HSM.
- Jaundice.
- haemolytic anaemia/thrombocytopenia/pancytopenia.
- *Periostitis* and metaphysial dystrophy.
- Funisitis (umbilical cord vasculitis).
- eye involvement glaucoma, Uveitis.

- mucosal features rhinitis (snuffles) develops at 1 week and worsens. Initially clear then progressively purulent and blood stained.
- mucous 'patches' seen on palate and lips.
Periostitis of long bones seen in neonatal syphilis
Late congenital: many years after birth

- Frontal bossing.
- Short maxilla.
- High palatal arch.
- **Hutchinson's triad**, a set of symptoms consisting of deafness, **Hutchinson's teeth** (centrally notched, widely-spaced peg-shaped upper central incisors), and interstitial keratitis (IK), an inflammation of the cornea which can lead to corneal scarring and potentially blindness. 8th nerve deafness.
- Saddle nose.
- Perioral fissures.
13-DAY-OLD FEMALE CHILD WITH CLEFT LIP ((SYPHILIS))

Congenital Syphilis - Mucous Patches

Source: CDC/ NCHSTP/ Division of STD Prevention, STD Clinical Slides
CONGENITAL SYPHILIS IN A 2-WEEK-OLD INFANT BOY WITH MARKED HEPATOSPLENOMEGALY. THE INFANT KEPT HIS UPPER EXTREMITIES IN A FLAIL-LIKE POSITION BECAUSE OF PAINFUL PERIOSTITIS.
Hutchinson teeth – late result of congenital syphilis

Saddle nose
If the disorder is suspected at the time of birth, the placenta will be examined for signs of syphilis. A physical examination of the infant may show signs of liver and spleen swelling and bone inflammation.

A routine blood test for syphilis is done during pregnancy. The mother may receive the following blood tests:

- Fluorescent treponemal antibody absorbed test (FTA-ABS)
- Rapid plasma reagin (RPR)
- Venereal disease research laboratory test (VDRL)

An infant or child may have the following tests:

- Bone x-ray
- Eye examination
- Lumbar puncture
- Dark-field examination to better detect syphilis-related bacteria under a microscope
- Liver function tests
- Urinalysis for proteinuria
Confirmed if T. pallidum identified in skin lesions, placenta, umbilical cord, or at autopsy.

Presumptive diagnosis if any of:
- Physical exam findings.
- CSF findings (positive VDRL).
- Osteitis on long bone x-rays.
- Funisitis (“barber shop pole” umbilical cord).
- RPR/VDRL >4 times maternal test.
- Positive IgM antibody.
Treatment

- Treatment of choice parenteral penicillin G for all stages of syphilis.

- Infants with suspected or confirmed infection should be treated with:
  - Penicillin for at least 10 days.
  - Benzyl penicillin, 30mg/kg per dose 12 hourly, IM or IV or
  - Procaine penicillin, 30mg/kg daily, IM

- Maternal treatment during pregnancy very effective (overall 98% success)

- Treat newborn if:
  - They meet diagnostic criteria
  - Mom was treated <4wks before delivery
  - Maternal titers do not show adequate response (less than 4-fold decline)
Follow up

Babies should be evaluated at 3-monthly intervals over the first year of life, with serological tests performed at each visit. In cases of neurosyphilis, ongoing serum and CSF analysis should be undertaken 6-monthly for the first 5 years of life. Re-treatment is needed if titres do not fall, or clinical signs of disease persist or develop.

Prevention

Prevention relies upon adequate antenatal services and screening facilities.
Toxoplasma gondii is a parasitic organism. The domestic cat is the primary host.

- Infection can be contracted by ingesting oocysts present in faecal material of infected hosts.
- Eating pseudocysts present in undercooked meat.
- Risk of fetal infection is lowest in early pregnancy but most fetuses infected early have severe consequences.

<table>
<thead>
<tr>
<th></th>
<th>Risk of fetal infection</th>
<th>Severe consequences of infection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1st trimester</td>
<td>10%</td>
<td>70%</td>
</tr>
<tr>
<td>3rd trimester</td>
<td>60%</td>
<td>&gt; 1%</td>
</tr>
</tbody>
</table>
Infants congenitally infected with Toxoplasmosis can be completely **asymptomatic**

**Neurological manifestations** include:

- convulsions
- hydrocephalus with bulging fontanelle
- microcephaly
- other evidence of CNS involvement (including calcification)

**Haemopoietic manifestations** include:

- anaemia
- thrombocytopenia purpura
- blueberry muffin appearance (seen in 25% of generalised infection)
- lymphadenopathy
- HSM
- Rash (tiny red spots or bruising)
Generalised features include

- Lethargy and malaise
- Poor feeding
- Vomiting
- Diarrhoea
- Temperature instability
- Jaundice
- Eye damage
- Hearing loss
- Low birth weight (intrauterine growth restriction)
- Vision problems
Intracranial calcifications in congenital toxoplasmosis. (A) Posterior-anterior and (B) lateral views of the skull showing scattered bilateral calcific flecks, nodules and linear streaks in frontal and parietal lobes of an infected infant.
Congenital toxoplasmosis with intracranial hydrocephalus. With dilated lateral ventricles

Toxoplasmosis Chorioretinitis
Investigations

- **Antenatal diagnosis** can be performed using *fetal blood* sent for:
  - PCR
  - IgM assay
  - Culture

- **Postnatal investigations** include:
  - Specific IgM or IgA in cord or baby's blood
  - CBC (anaemia/thrombocytopenia)
  - Liver function tests
  - Culture by inoculation of blood/placenta in mice
  - Cranial US (hydrocephalus and calcification)
  - CT scan (more sensitive than ultrasound at identifying calcification)
  - Ophthalmological review
If infant diagnosed **prenatally**, treat mom with
- Spiramycin, pyrimethamine (anti-malarial, dihydrofolate reductase inhib), and sulfadiazine (sulfa antibiotic)

**SYMPTOMATIC INFANT:**
- Treatment consists of prolonged therapy for the **first year of life** with:
  - **pyrimethamine** (1mg/kg orally daily)
  - **sulphadiazine** (50mg/kg orally, twice daily)
  - **folinic acid** (1ml/kg, orally twice weekly) is added
- The addition of **steroids** in severe infection has been suggested, but no evidence exists for this practice.
- Ongoing opthalmological and developmental follow up is mandatory.
Outcome
Infants symptomatic at birth have high incidence of long term difficulties: chorioretinitis (over 90%), developmental delay (50%), *seizures* (40%), microcephaly (20%), deafness, hydrocephalus.

Prevention
Prevention of Toxoplasmosis is aimed at preventing ingestion of infected material. Pregnant women should be warned to avoid foods/products that may be contaminated with the oocytes.
Human immunodeficiency virus (HIV) is a lentivirus (a member of the retrovirus family) that causes acquired immunodeficiency syndrome (AIDS), (ssRNA-RT viruses)

Women with HIV can infect their babies while they’re pregnant or during delivery. HIV can also be passed from mother to baby through breast milk.

However, if mothers receive treatment for HIV during their pregnancy, the risk of passing the virus to their babies can be significantly reduced.

Is congenital AIDS/HIV common?

Between 6,000 and 7,000 children are born to HIV-infected mothers each year in the United States.
Symptoms

- Even though there are no symptoms of HIV, the virus is actively infecting and killing cells of the immune system. As the immune system deteriorates, complications begin to develop.

Symptoms vary child-to-child depending on age, but may include:

- Lymph nodes that remain enlarged for more than three months.
- Weight loss.
- Frequent and long-lasting fevers and sweats.
- Short-term memory loss and delayed physical and developmental growth.
- HSM.
- Intermittent diarrhea (diarrhea that may come and go).
- Constant or recurring ear infections.
Recurrent **bacterial infections**, particularly invasive infections like **meningitis**, **septicaemia** and **pneumonia**.

Recurrent frequent common childhood infections such as **otitis media**, chest infection, urinary tract infections, **sinusitis**.

Unusual infections such as **Mycobacterium avium** complex (MAC), **Pneumocystis jirovecii** pneumonia.

Persistent oral **candidiasis** that fails to respond to standard therapy.

Recurrent or severe viral infections, eg **herpes simplex**, **herpes varicella-zoster** infection as **shingles**, **cytomegalovirus** (CMV).
Q: HOW IS HIV DIAGNOSED?

- **Early diagnosis** of HIV infection should occur rapidly postnatally where mothers are known to be HIV-positive, as this allows for prophylaxis against, and early detection and treatment of opportunistic infection in neonates.

- Standard ELISA tests are unreliable for the first 18 months because of the transmission of maternal antibodies which persist for some time in the baby.

- HIV RNA PCR.

- CD4 count.

- HLA B5701.
Additional tests

Additional tests are performed at diagnosis to assess concurrent infection and risk of different opportunistic infection and can include:

- Serology for hepatitis A, hepatitis B, hepatitis C, Epstein-Barr virus, CMV, herpes simplex virus, mumps, measles, rubella, VDRL and toxoplasmosis.
- Malaria film.
- Mantoux' test/TB cultures.
- Baseline CXR.
- Brain ultrasound/MRI (where there are neurological signs).

Monitoring

- CD4 counts.
- Viral load.
- Screening - audiology, dental, neurodevelopmental, ophthalmology, TB.
A combination of zidovudine and lamivudine given to mothers in the antenatal, intrapartum and postpartum periods and to babies for a week after delivery, or a single dose of nevirapine given to mothers in labour and babies immediately after birth may be most effective.

Other strategies include the use of elective Caesarean section and avoidance of breastfeeding where possible.
CONGENITAL RUBELLA

- Togavirus, ss RNA enveloped.
- The most critical time is the first trimester (the first 3 months of a pregnancy). After the fourth month, the mother's rubella infection is less likely to harm the developing fetus.
- Risk factors for congenital rubella include:
  - Not getting the recommended rubella immunization
  - Contact with a person who has rubella (also called the 3-day measles)
Maternal symptoms of infection

- About 50% asymptomatic
- Maculopapular rash
- Fever
- Lymphadenopathy
- Fatigue
- Sore throat
Fetal symptoms

- Malaise
- Post-auricular adenopathy
- Conjunctivitis
- NON-PRURITIC, ERYTHEMATOUS, MACULOPAPULAR RASH
- Fever
- Bruises
- Body Ache
- Bruises Easily
- Feels Hot to Touch
- Perianal Rash
Congenital rubella syndrome is a severe, disabling condition featuring:
- Eye disorders (cloudy cornea/cataracts, salt and pepper chorioretinitis, microphthalmia)
- Sensorineural deafness
- Cardiovascular (pulmonary stenosis and PDA)
- Microcephaly
- Growth restriction
- Haemopoietic disorders
  - HSM
  - Lymphadenopathy
  - Thrombocytopenia anaemia
  - Extramedullary haemopoiesis (blueberry muffin skin appearance)
- Pneumonitis with associated respiratory signs
- Renal tract abnormalities
Eye anomalies may include cataracts, glaucoma, strabismus, nystagmus, microphthalmia, and iris dysplasia.
MANIFESTATIONS OF CONGENITAL RUBELLA

Deafness
Eye
CNS
Cardiac

6/5/2012
Infantile Glaucoma in a patient with congenital rubella syndrome....

Cataracts due to Congenital Rubella Syndrome
Blueberry muffin spots representing extramedullary hematopoiesis

Rash
**Investigations**

- **Diagnosis** is usually demonstrated by evidence of maternal seroconversion or rising IgG titres. This occurs some 10 days after contact. IgM assay is useful where exact 'contact time' is not known. IgM persists for around 2 months after primary infection. Re-infection can be identified by seeing a four-fold or more rise in IgG titres.

- **Fetal diagnosis** is possible from
  - cord blood IgM.
  - rubella PCR of amniotic fluid.

- **Postnatal diagnosis** is by
  - IgM.
  - isolation of rubella virus (possible form many sites including NPA, eye, throat, CSF, stool and urine for up to 12 months).
OTHER TESTS INCLUDE

- CBC
- renal function and electrolytes
- liver function tests
- cranial ultrasound (looking for discrete calcification)
- echocardiography (looking particularly for Pulmonary Stenosis and PDA)
- renal ultrasound
- CXR (indicated if the baby has respiratory symptoms)
- long Bone Xrays
- hearing assessment is mandatory, even in babies with no overt disease at birth.
- Ophthalmological assessment
- Endocrine problems can occur in the long term including diabetes mellitus and hypothyroidism.
Management
There is no specific treatment. Management is supportive and aimed at addressing specific problems present (developmental/sensory/endocrine/cardiac).

Prevention
- Rubella immunisation is offered to all children in combination with measles and mumps vaccination at 1 year of age.
- All women should be screened at first antenatal clinic appointment, and if found to be rubella susceptible, offered immunisation in the post-partum period.
Children with congenital rubella syndrome are likely to have severe developmental issues.

Ongoing hearing and vision assessments are essential in babies whose mothers contracted rubella after 12 weeks gestation.

Rarely, a form of subacute sclerosing panencephalitis with demonstration of raised rubella antibodies in CSF, has been documented.
CONGENITAL CMV

- DNA virus of Herpesvirus family
- Most common congenital viral infection
- Horizontal transmission
- Vertical transmission
  - In utero
  - During delivery
  - Breast feeding
Infection in Pregnant Women

- Reactivation of latent infection or primary infection.
- Foetal infection by reactivation less likely.
CLINICAL MANIFESTATION

- 90% of congenital CMV – asymptomatic at birth.
- 0.5 – 15% of these are at risk for psychomotor hearing, neurologic, ocular, or dental abnormalities within first few years of life.
- 5-10% of cases may have sensorineural hearing loss.
<table>
<thead>
<tr>
<th>System</th>
<th>Feature</th>
<th>Frequency (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin</td>
<td>Petechiae</td>
<td>75</td>
</tr>
<tr>
<td></td>
<td>Purpura, ecchymoses</td>
<td>10</td>
</tr>
<tr>
<td></td>
<td>Jaundice</td>
<td>67</td>
</tr>
<tr>
<td>Hepatobiliary</td>
<td>Direct bilirubin &gt;2 mg/dL (34.2 mcmol/L)</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>Elevated ALT (&gt; 80 IU/mL)</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>Hepatomegaly</td>
<td>60</td>
</tr>
<tr>
<td>Hematopoietic</td>
<td>Thrombocytopenia (&lt;100 × 10³/mcL [&lt;100 × 10⁹/L])</td>
<td>77</td>
</tr>
<tr>
<td></td>
<td>Anemia</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>Splenomegaly</td>
<td>60</td>
</tr>
<tr>
<td>CNS</td>
<td>Microcephaly</td>
<td>53</td>
</tr>
<tr>
<td></td>
<td>Intracranial calcifications (computed tomography)</td>
<td>54</td>
</tr>
<tr>
<td></td>
<td>Poor feeding, lethargy</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>Seizures</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td>Increased CSF protein (&gt; 120 mg/dL)</td>
<td>47</td>
</tr>
<tr>
<td>Auditory</td>
<td>Sensorineural hearing loss</td>
<td>50</td>
</tr>
<tr>
<td>Visual</td>
<td>Chorioretinitis</td>
<td>10</td>
</tr>
</tbody>
</table>
calcifications within the periventricular regions

petechial rash
DIAGNOSIS

Maternal:
- Serology IgG & IgM. CMV specific IgM can persist unto 18/12 & is present in 10% recurrent cases.
- Presence of both IgG & IgM are presumptive evidence of primary infection. Paired specimen needed if seroconversion from -ve to +ve not documented.

Antenatal:
- Amniotic fluid: Culture, PCR for viral DNA.
- Fetal blood: CMV specific IgM, culture, LFT’s, PCR
- Ultrasound: Ascites and hydrops, microcephaly, IUGR, ventriculomegaly, intracerebral calcification.
DIAGNOSIS OF CONGENITAL CMV

- **Postnatal**: by isolating virus in first 3 wks of life.
- Placenta: Histology, PCR.
- Serology of blood or Urine. IgM persist for 8/12.
- PCR.
Supportive care as always is vitally important with attention to general care.

Antiviral therapy: - Ganciclovir (6mg/kg dose every 12 hrs IV FOR FIRST 6 WEEKS OF LIFE (prevent hearing deterioration). The most common adverse effect in neonates is neutropenia, which occurs in as many as 60% of recipients.
Throughout the pregnancy, practice good personal hygiene, especially handwashing with soap and water, after contact with diapers or oral secretions (particularly with a child who is in day care).

Laboratory testing for antibody to CMV can be performed to determine if a woman has already had CMV infection.

Avoid contact with saliva when kissing a child. Clean toys, countertops, and other surfaces that come into contact with children’s urine or saliva.
HERPES SIMPLEX VIRUS

- HSV type 1 & 2.
- DNA virus of herpes virus family
- Neonatal infection is usually the result of HSV 2 as this is the main virus associated with genital infection.
- Spread by sexual contact or oral transmission.
- The baby is usually infected perinatally during passage through the birth canal.
- Premature rupturing of the membranes is a well recognized risk factor.

- The baby may also be infected from other sources such as oral lesions from the mother or a herpetic whitlow in a nurse.
- Babies born to mothers with a primary genital infection at the time of delivery have a 50% risk of developing infection, compared with <5% in cases of recurrent infection present at the time of delivery.
Mother with active herpes infection (although active infection may not be apparent)

Blisters due to congenital herpes
Neonatal HSV usually presents within 2 weeks of birth

- 90% are acquired during passage through the birth canal or through ascending infection
- 5% have 'congenital' HSV infection
- 5% have post-natally acquired infection
- Usual clinical presentations are
- skin/eye/mouth (SEM) localised disease
  - untreated, >70% will progress to disseminated disease
  - 25% will have virus in CSF at initial presentation
  - isolated vesicles or 'crops'
  - occasionally other skin reactions can be present, including zoster-like eruptions
  - keratoconjunctivitis with dendritic ulcers
  - choriodoretinitis
- oral vesicular lesions can be present
- non-specific presentation with
  - lethargy
  - poor feeding
  - fever
  - convulsion
  - apnoea
  - respiratory distress
  - hepatomegaly
  - jaundice
  - DIC
Pneumonitis: tends to occur day 4 to 7.

- respiratory distress and can develop into haemorrhagic pneumonitis
- CXR shows diffuse pneumonic change
- meningo-encephalitis isolated or part of disseminated disease presents with:
  - encephalopathy (mean 11 days of age)
  - seizures are common and often intractable
  - absent gag-reflex is a particular feature
- Later calcification and cerebral atrophy can develop.
The unwell baby should be examined for vesicles.

Specimens from lesions, throat and eye swabs should be performed.

The use of immunoflourescence can provide rapid evidence of infection. Viral culture can take 5 days to demonstrate typical cytopathic changes.

Lumbar puncture is mandatory, with CSF sent for PCR and culture. However, negative PCR testing on CSF does not completely rule out HSV infection.

EEG and brain imaging are useful adjuncts in cases where diagnosis of CNS infection is in doubt.

Serological studies are of little value early on as IgM may take 2 weeks to appear and IgG titres may not rise in babies and may reflect maternal antibody status.
Specific early treatment is with Acyclovir 10mg/kg IV three times daily for a total of 14 to 21 days.

Vidarabine 15mg/kg 12 hourly, IV is also effective but is more cumbersome.

Supportive care as always is vitally important with attention to general care.

Eye lesions require topical treatment (eg. Idoxuridine) and ophthalmological referral is essential.
Pregnant women who have visible ulcers from genital herpes at the time of delivery usually are encouraged to have a **Caesarean section** to prevent HSV from spreading to the newborn.

Women known to have herpes before delivery may be counseled to take antivirals for the last few weeks of their pregnancy.
Congenital Parvovirus Infection:-

- DNA virus, Causes erythema infectiosum,
- Responsible for transient aplastic crisis in sickle cell anaemia.

CONGENITAL INFECTION
- More pronounced during 2nd rather than 1st trimester.
- Severe and often fatal in foetus.
- ~9% foetal loss 4-6 weeks after development of rash in mother.
- Hydrops foetalis most common manifestation and Profound anaemia.
DIAGNOSIS AND TREATMENT

- **Diagnosis :-**
  - Pregnant mother
    - Specific IgM.
    - IgG seroconversion.
    - Elevated serum α-feto-protein.

- **Foetus**
  - Ultrasound for symptoms associated with hydrops and body cavities.
  - PCR

- **Treatment:**
  - Intrauterine transfusions.
  - Digitalis based drugs
VARICELLA-ZOSTER VIRUS

- 90% of pregnant women already immune, therefore primary infection is rare during pregnancy.
- Primary infection during pregnancy carries a greater risk of severe disease, particularly pneumonia.

First 20 weeks of Pregnancy:

- up to 3% chance of transmission to the fetus.
- recognised congenital varicella syndrome.
- Scarring of skin.
- Hypoplasia of limbs.
- CNS and eye defects.
- Death in infancy normal.
VZV can cross the placenta in the late stages of pregnancy to infect the fetus congenitally.

Neonatal varicella may vary from a mild disease to a fatal disseminated infection.

If rash in mother occurs more than 1 week before delivery, then sufficient immunity would have been transferred to the fetus.

Zoster immunoglobulin should be given to susceptible pregnant women who had contact with suspected cases of varicella.

Zoster immunoglobulin should also be given to infants whose mothers develop varicella during the last 7 days of pregnancy or the first 14 days after delivery.
Known as *Streptococcus agalactiae*, is the causative agent of postpartum infection and as the most common cause of neonatal sepsis.

Group B streptococci colonize the vaginal and gastrointestinal tracts in healthy women, with carriage rates 15%-45%.

Neonates can acquire the organism vertically in utero or during delivery.

Only 1-2% of colonized neonates develop invasive disease.
GROUP B STREPTOCOCCUS

Pathogenesis:
- Disease is divided into early and late disease.
- Early sepsis often presents within 24 hours of delivery but can become apparent up to 7 days postpartum.
- Late sepsis between one week postpartum and age 3 months.

Treatment:
- Intrapartum antimicrobial prophylaxis.
- Penicillin or ampicillin is the initial approach.
- Clindamycin and erythromycin are standard in individuals with penicillin allergy.
THANK YOU