Congenital Infection

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Rout of Transmission

Transmission	Types
Intra-uterine	Transplacental
	Ascending infection
Intra-partum	Contact with infected material during delivery, secretion , blood faeces
Post-partum	Breast feeding
	Blood transfusion
	Nosocomial

Important Information for any congenital infection

- Background prevalence
- Incidence of infection in pregnancy
- Risk of mother to child transmission
- Timing of mother to child transmission
- Risk factors for maternal and perinatal infections
- Consequences of both congenital/perinatal infection short and long term

Terminology

1. Congenital
2. Perinatal
3. Neonatal

- What is TORCH
    1. **T**oxoplasmosis,
    2. **O**ther (syphilis ,parvovirus &VZV),
    3. **R**ubella,
    4. **C**MV,
    5. **H**erpes(Hepatitis &HIV),

Risk and features of congenital infection

- Risk of congenital infection:

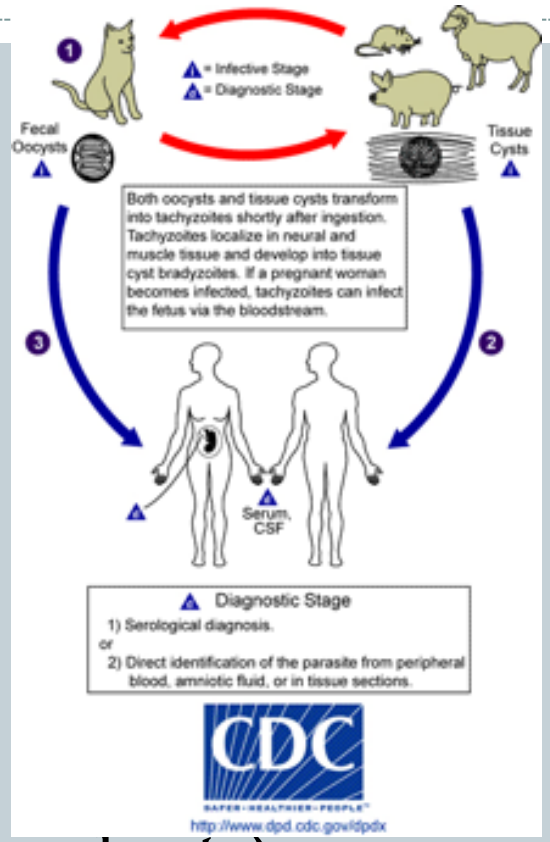
1. Organism (Teratogenicity)
2. Type of maternal infection(Primary ,recurrent)
3. Time during pregnancy (1^{st},2^{nd},3^{rd} Trimester)

- features of congenital infection :

1. Intrauterine growth retardation(IUGR)
2. Fever
3. Skin rash, joundice
4. Microcephaly
5. Hepatosplenomegaly(HSM)
6. Generalized lymphadenopathy
7. Thrombocytopenia
8. IgM, Persistent IgG

Toxoplasmosis

- Toxoplasma gondii
- Definitive host is the domestic cat
- Contact with oocysts in feces



- Ingestion of cysts (meats, garden products)
- Can be transmitted from the mother to the baby

Epidemiolology

- European countries (ie France, Greece)
- Usually asymptomatic
- Primary maternal infection in pregnancy
- Infection (Transmission) rate higher with infection in 3rd trimester
- Fetal death higher with infection in 1st trimester

Clinical presentation

- Mostly asymptomatic
- Classic triad of symptoms:
  1. *Chorioretinitis*
  2. *Hydrocephalus*
  3. *Intracranial calcifications*

- Other symptoms include fever, rash, HSM, microcephaly, seizures, jaundice, thrombocytopenia, lymphadenopathy

Diagnosis , treatment and prevention

- Diagnosis

1. Maternal serology IgM/IgA , IgG,
2. Fetal ultrasound/ tissue culture and PCR.
3. Newborn
    1. Serology
    2. Culture
    3. PCR

- Treatment

1. Spiramycin
2. Pyrimethamine and sulfadiazine

- Prevention

1. Avoid exposure to contaminated food or water and undercooked meat
2. Hand washing

Syphilis

- Treponema pallidum (spirochete)
- Transmitted via sexual contact
- Mother with primary or secondary syphilis
- Typically occurs during second half of pregnancy

Clinical features

- Intrauterine death in 25%
- 3 major classifications

Congenital Syphilis

- Late abortion or stillbirth
- Infantile
  - Rash and Funisitis (umbilical cord vasculitis)
  - Osteochondritis
  - Periostitis
  - Liver & lung fibrosis
- Childhood
  - Interstitial keratitis
  - Hutchinson teeth
  - Eighth nerve deafness
  - **Frontal bossing, Short maxilla, High palatal arch, Saddle nose , Perioral fissures**

Diagnosis and Treatment

Diagnosis

1. **RPR/VDRL**: non-treponemal test
2. **MHA-TP/FTA-ABS**: specific treponemal test
3. Confirmed if T. pallidum identified in skin lesions, placenta, umbilical cord, or at autopsy

Treatment

Penicillin G

Prevention

- RPR/VDRL screen in ALL pregnant women early in pregnancy and at time of birth

Parvovirus P 19

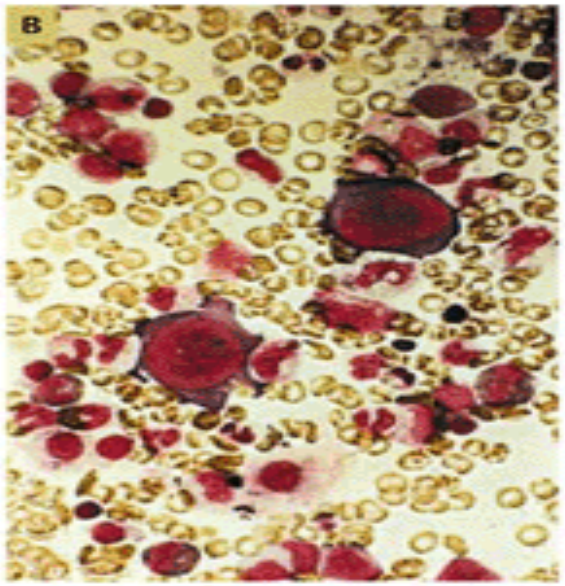
- Parvovirus P 19
- Causative agent of Fifth disease (erythema infectiosum)
- Spread by the respiratory route, blood and transplacental

Epidemiology

- Most of the population is eventually infected.
- Half of women of childbearing age are susceptible to infection.
- Risk of fetal death highest when infection occurs during the second trimester of pregnancy (1st 20 wks of pregnancy (12%).
- Minimal risk to the fetus if infection occurred during the third trimesters of pregnancy.

Clinical Faeture

- Known to cause fetal loss through hydrops fetalis; severe anaemia, congestive heart failure, generalized oedema and fetal death
- No evidence of teratogenecity



Diagnosis, and Treatment

- Diagnosis
  - ultrasound
  - Serology IgM, persistant IgG
  - PCR
- Traetment
  - intrauterine transfusions and administration of digoxin to the fetus.

Neonatal Varicella

- 90% of pregnant women already immune
- Primary infection during pregnancy carries a greater risk of severe disease

Clinical Features

- 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

Diagnosis

Test		Pregnant mother and Fetus	Neonate
Direct form the vesicles	Culture	+	+
	DFA	+	+
	PCR	+ Fetal blood and amniotic fluid	+
Serology	IgM Rising IgG	+ +	+
US and MRI		+	

Treatment and Prevention

- *Acyclovir* at first signs of varicella pneumonia
- Pre-expoure;live-attenuated vaccines before or after pregnancy but not during pregnancy.
- Postexposure Zoster immunoglobulin to susceptible pregnant women and infants whose mothers develop varicella during the last **5 days** of pregnancy or the first **2 days** after delivery and premature baby <28 wks of gestation

rubella

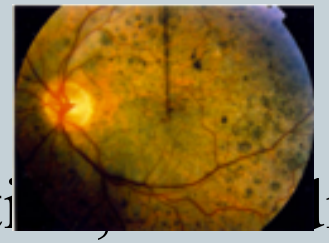
- R=rubella
- RNA enveloped virus, member of the togaviridae family
- Spread by respiratory droplets and transplacentally

Epidemiology

- Vaccine-preventable disease
- No longer considered endemic.
- Mild, self-limiting illness
- Infection earlier in pregnancy has a higher probability of affected infant (first 12 wks 70% and 13-16 wks 20% and rare >16 wks of pregnancy)

Clinical Features

- Cataracts, glaucoma
- “Salt and Pepper” retinopathy
- Cardiac malformations
- Neurologic (less common)
- Others to include growth retardati isease, HSM, thrombocytopenia, *“blueberry muffin” lesions*



Diagnosis

- Maternal IgG is useless!
- Viral isolation virus from nasal secretions, throat, blood, urine, CSF.
- Serologic testing. IgM = recent postnatal or congenital infection.
- Rising monthly IgG titers suggest congenital infection.

Treatment Prevention

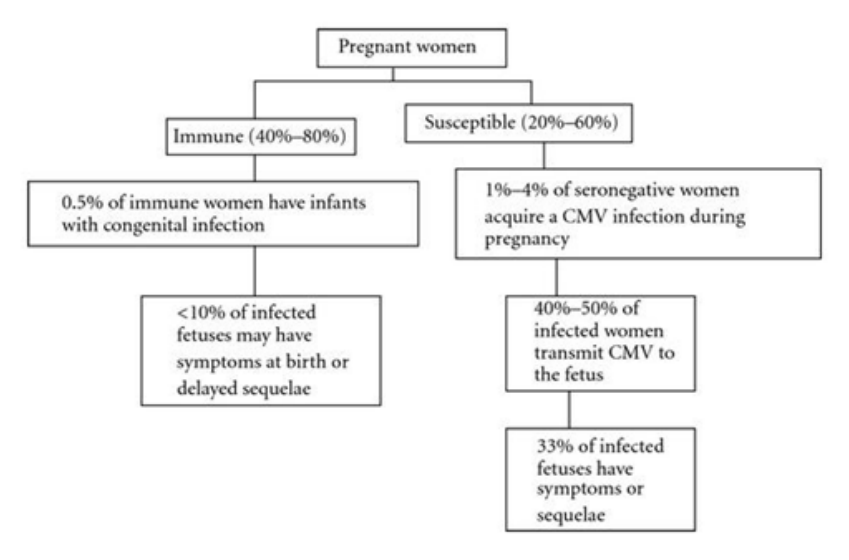
- Supportive care only with parent education
- Prevention by immunization
- Maternal screening
- Vaccinate if not immune (avoid pregnancy for three months)

cytomegalovirus

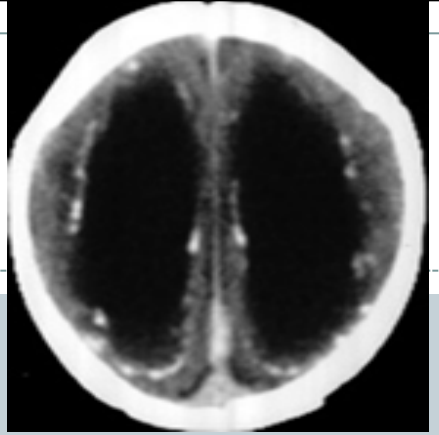
- C=cytomegalovirus
- Most common congenital viral infection~40,000 infants per year.
- Mild, self limiting illness

Epidemiology

- Transmission can occur with primary infection or reactivation of virus but 40% risk of transmission in primary infection
- Increased risk of transmission later in pregnancy but more severe sequalae associated with earlier acquisition



Clinical presentation



- 90% are asymptomatic at birth
- Up to 15% develop symptoms later
- Microcephaly, periventricular calcifications, neurological deficits, HSM, petechiae, jaundice, chorioretinitis
- >80% develop long term complications: Sensorineural hearing loss (most common)
- , vision impairment, developmental delay

Diagnosis

- Maternal IgG shows only past infection
- **Viral isolation from urine or saliva in 1st 3 weeks of life**
- Viral load and DNA copies can be assessed by PCR
- Detection of Cytomegalic Inclusion bodies in affected tissue
- Serologies not helpful given high antibody in population

Treatment Prevention

- Gancyclovir x6wks in symptomatic infants

herpes simplex

- H=herpes simplex (HSV)
- HSV1 or HSV2

Epidemiology

- Primarily transmitted through infected maternal genital tract
- Primary infection with greater transmission risk than reactivation
- Rationale for C-section delivery prior to membrane rupture

Clinical presentation

- Most are asymptomatic at birth
- 3 patterns of equal frequency with symptoms between birth and 4wks:Skin, eyes, mouth , CNS disease, Disseminated disease (present earliest)
- Initial manifestations very nonspecific with skin lesions NOT necessarily present

Diagnosis and treatment

Diagnosis

- Culture of maternal lesions if present at delivery
- Cultures in infant
- CSF PCR
- Serologies is useless

Treatment

- High dose of acyclovir