

Lecture 1

Congenital Infection

- Additional Notes
- Important
- Explanation
- Examples

Introduction

- Infections acquired in utero or during the birth process are a significant cause of fetal and neonatal mortality and an important contributor to early and later childhood morbidity. The original concept of the TORCH perinatal infections was to group five infections with similar presentations, including rash and ocular findings. These five infections are: "TORCH"
 - ✓ Toxoplasmosis "aggressive"
 - ✓ Other (syphilis ,parvovirus (p19) &VZV),
 - ✓ Rubella "aggressive"
 - ✓ Cytomegalovirus (CMV)
 - ✓ Herpes simplex virus (HSV)
- Risk of congenital infection:
 - ✓ Organism (Teratogenicity)
 - ✓ Type of maternal infection(Primary "transmit to the fetus", recurrent)
 - ✓ Time during pregnancy (1st "most sever",2nd,3rd Trimester)
- Features of congenital infection:
 - Intrauterine growth retardation(IUGR)
 - ✓ Skin rash, joundice
 - Microcephaly, hydrocephaly
- Hepatosplenomegaly (HSM)

- ✓ Fever
- Generalized lymphadenopathy
- ✓ Thrombocytopenia
- ✓ IgM, Persistent IgG

Congenital Toxoplasmosis

- Toxoplasma gondii, definitive host is the domestic cat.
- Contact with oocysts in feces.
- Infection (Transmission) rate higher with infection in 3rd trimester
- Fetal death higher with infection in 1st trimester.
- Mostly asymptomatic, however the classic triad of symptoms:
 - ✓ Chorioretinitis "nflammation of the choroid (thin pigmented vascular coat of the eye) and retina of the eye"
 - √ Hydrocephalus
 - ✓ Intracranial calcifications
- Diagnosis:
 - ✓ Serology "increase IgM, IgG, serial IgG → acute infection in baby"
 - ✓ Culture "rarely done"
 - ✓ PCR
- Treatment
 - ✓ Spiramycin
 - Pyrimethamine and sulfadiazine



Congenital Syphilis

- Treponema pallidum (spirochete)
- Transmitted via sexual contact
- Mother with primary or secondary syphilis.
- Typically occurs during second half of pregnancy.
- Clinical presentation:
 - ✓ Fetal: Stillbirth, Neonatal death, Hydrops fetalis
 - ✓ Early congenital(infantile): Rash and destruction of face tissue and Funisitis (Umbilical Cord Vasculitis),Osteochondritis, Periostitis (Inflammation of the periosteum), Liver and Lung fibrosis
 - ✓ Late congenital (Childhood): Frontal bossing, High palatal arch, Hutchinson teeth (Screwdriver teeth), Short maxilla, 8th nerve deafness, Saddle nose, Perioral fissures.



- ✓ RPR/VDRL: non treponemal test (RPR) Rapid plasma regain, (for prevention too)
- ✓ Confirmed if T. pallidum identified in skin lesions, placenta, umbilical cord, or at autopsy
- Treatment: Penicillin G

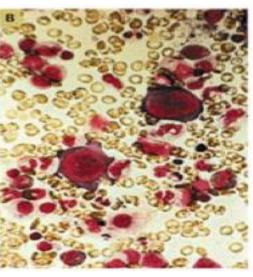




Congenital Parvovirus B19

- Causative agent of erythema infectiosum (AKA the fifth disease)
- The virus replicates in late erythroid cell precursors of the bone marrow and blood, leading to their destruction and inhibition of erythropoiesis
 → anemia.
- Spread by the respiratory route, blood and transplacental
- Risk of fetal death highest when infection occurs during the second trimester.
- Minimal risk to the fetus if infection occurred during the third trimesters.
- Known to cause fetal loss through: hydrops fetalis, severe anaemia
 → congestive heart failure, generalized oedema
 → fetal death.
- Diagnosis
 - ✓ ultrasound
 - ✓ PCR
 - ✓ Serology IgM, persistant IgG
- Treatment
 - ✓ intrauterine transfusions
 - ✓ administration of <u>digoxin</u> to the fetus.





Neonatal Varicella

- Very rare disease. 90% of pregnant women already immune
- Primary infection during pregnancy carries a greater risk of severe disease
- First 20 weeks of Pregnancy.
- Up to 3% chance of transmission to the fetus, recognized congenital varicella syndrome:
 - ✓ Scarring of skin
 - ✓ Hypoplasia of limbs
 - ✓ CNS and eye defects.
- Treatment:
 - ✓ Acyclovir at first signs of varicella pneumonia.
- Prevention:
 - ✓ Pre-exposure: live-attenuated vaccines before or after pregnancy but not during pregnancy.
 - ✓ Post exposure: Varicella-zoster immune globulin (VariZIG) to:
 - Neonates whose mothers have signs and symptoms of varicella around the time of delivery (within five days before or two days after).
 - Hospitalized premature infants born at ≥28 weeks of gestation or more.
 - Hospitalized premature infants born at <28 weeks of gestation or who weigh <1000 grams at birth regardless of maternal history of varicella or vaccination.



Congenital rubella

- Rubella typically causes a mild, self-limited illness; its major impact occurs during pregnancy, when it can have devastating effects on the developing fetus.
- Infection earlier in pregnancy has a higher probability of affected infant: First 12 weeks 70%
- Clinical Features:
 - ✓ Sensor-neural hearing loss (most common)
 - ✓ Cataracts, glaucoma.
 - ✓ "Salt and Pepper" retinopathy
 - ✓ cardiac malformations
 - ✓ Others to include growth retardation, bone disease, HSM, thrombocytopenia, purpuric skin lesions "blueberry muffin" lesions.

Diagnosis:

- ✓ Viral isolation virus from nasal secretions, throat, blood, urine, CSF.
- ✓ Serology:
 - Infant IgM → recent postnatal or congenital infection.
 - Persistence of IgG → congenital infection
- Treatment and Prevention:
 - ✓ Supportive care only with parent education.
 - ✓ Prevention by immunization.



Congenital cytomegalovirus

- Most common congenital viral infection ~40,000 infants per year.
- Mild, self limiting illness
- Transmission can occur with primary infection or reactivation of virus.
- Increased risk of transmission later in pregnancy but more severe sequalae associated with earlier acquisition
- Clinical presentation:
 - √ 90% are asymptomatic at birth
 - ✓ Microcephaly, periventricular calcifications, neurological deficits, HSM, petechiae, jaundice, chorioretinitis

√ >80% develop long term complications: Hearing loss, vision impairment, developmental delay

- Diagnosis:
 - ✓ Viral isolation from **urine** or saliva in 1st. 3 weeks of life
 - ✓ Viral load and DNA copies can be assessed by PCR
- Treatment:
 - ✓ Ganciclovir in symptomatic infants

Herpes simplex virus

- HSV1 or HSV2
- Primarily transmitted through infected maternal genital tract
- Most are asymptomatic at birth.
 - ✓ 3 patterns of equal frequency with symptoms between birth and 4th. week: Skin, eyes, mouth, CNS disease, Disseminated disease (present earliest)
 - ✓ Initial manifestations very nonspecific with skin lesions NOT necessarily present
- Diagnosis:
 - ✓ Culture of maternal lesions if present at delivery
 - ✓ Cultures in infant
- Treatment:
 - ✓ High dose of acyclovir
- Management of pregnant women with primary herpes simplex is to do C- section delivery prior to membrane rupture.

		Rate of transmission	Clinical	Diagnosis	Treatment/ Prevention
	toxoplasmosis	Infection (Transmission) rate higher with infection in 3 rd trimester Fetal death higher with infection in 1 st trimester	Chorioretinitis Hydrocephalus Intracranial calcifications	Serology "increase IgM, IgG, serial IgG → acute infection"	Spiramycin Pyrimethamine and sulfadiazine
	syphilis	Mother with primary or secondary syphilis Typically occurs during second half of pregnancy	 Fetal: Stillbirth infantile: Rash and destruction of face tissue and Funisitis Childhood: Frontal bossing, High palatal arch, Hutchinson teeth (Screwdriver teeth) 	Serology	Penicillin G
	rubella	first 12 wks 70% and 13-16 wks 20% and rare >16 wks of pregnancy	 Sensor-neural hearing loss (most common) Cataracts, glaucoma. "Salt and Pepper" retinopathy "blueberry muffin" lesions. 	Viral isolation virus Serology	
	cytomegalovirus	0.5-2% (0.2-1%) Increased risk of transmission later in pregnancy but more severe sequalae associated with earlier acquisition	90% are asymptomatic at birth	Viral isolation from urine → PCR.	Ganciclovir
	herpes simplex virus	Mostly intrapartum >90%	Most are asymptomatic at birth	Culture of maternal lesions if present at delivery	acyclovir C-section
	varicella	First 20 weeks of Pregnancy	Scarring of skin Hypoplasia of limbs CNS and eye defects.	Culture Serology PCR	live-attenuated vaccines
	Parvovirus	(1 st 20 wks of pregnancy (12%).		ultrasound PCR Serology	intrauterine transfusions administration of <u>digoxin</u> to the fetus.