

# Transplacental Infections

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دعاء قبل المذاكرة

(اللهم إني أسألك فهم النبيين و حفظ المرسلين و الملائكة المقربين، اللهم اجعل ألسنتنا عامرة بذكرك و قلوبنا بخشيتك، إنك على كل شيء قدير و حسبنا الله و نعم الوكيل)

# 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”

1. Toxoplasmosis, 2. Other (syphilis, parvovirus & VZV), 3. Rubella, 4. CMV, 5. Herpes (Hepatitis & HIV)

## Rout of transmission

Mechanisms	Timing of events	Classification
( Trans placental , Ascending infection )	(In utero)	1 -Congenital
( Contact with infected material during delivery, secretion , blood faeces )	(During labour and delivery)	2- Perinatal
( Direct contact, breastfeeding or nosocomial exposure for Blood transfusion )	(After birth)	3- Neonatal

## Risk of congenital infection

Type of maternal infection (Primary, recurrent)

Organism (Teratogenicity)

***1° Maternal infection in the first half of pregnancy have the greatest risk to the fetus***

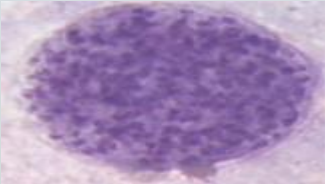
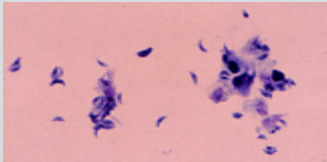

Time during pregnancy (1<sup>st</sup>, 2<sup>nd</sup>, 3<sup>rd</sup> Trimester)

### Feature of congenital infection:

Intrauterine growth retardation (IUGR) , Fever , Skin rash , jaundice , Generalized lymphadenopathy , Microcephaly , hydrocephaly , Thrombocytopenia , Hepatosplenomegaly (HSM) , IgM, Persistent IgG

# Congenital toxoplasmosis

Toxoplasma gondii is Obligat intracellular parasite and have three form :

Bradyzoites	Tachyzoites	Oocysts
<ul style="list-style-type: none"> <li>• slowly dividing forms</li> <li>• <b>CHRONIC PHASE</b></li> <li>• Immunity +</li> </ul> 	<ul style="list-style-type: none"> <li>• rapidly dividing forms</li> <li>• <b>ACUTE PHASE</b></li> <li>• Immunity -</li> </ul> 	<ul style="list-style-type: none"> <li>• Shed in cat feces</li> </ul> 

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 :**

1- Chorioretinitis 2-Hydrocephalus 3-Intracranial calcifications

Prevention	Treatment	Diagnosis
Avoid exposure to cat feces	Spiramycin	Serology : increase IgM, IgG, IgA " acute infection "
Wash hands	Pyrimethamine	Culture " rarely done "
Cook all meat thoroughly	sulfadiazine	PCR

# Syphilis

#Treponema pallidum (spirochete)

#Transmitted via sexual contact

#Mother with **primary** or **secondary** syphilis

#Typically occurs during **second half** of pregnancy

Intrauterine death in 25%  
Three classification:

	Infantile	Childhood
Late abortion or stillbirth	Rash and funisitis “umbilical cord vasculitis”	Interstitial keratitis
	Osteochondritis	Hutchinson teeth
	Periostitis	Eighth nerve deafness
	Liver and lung fibrosis	Frontal bossing, short maxilla, high palatal arch, saddle nose, perioral fissures

# Diagnosis and treatment

#RPR/VDRL: non treponemal test.

#MHA-TP/FTA-ABS: specific treponemal test.

#Confirmed if *T.pallidum* identified in skin lesions, placenta, umbilical cord or at autopsy.

#Treated by penicillin.

#Prevention: RPR/VDRL screen in ALL pregnant women early in pregnancy and at time of birth.

# Parvovirus P19

#Causative agent of Fifth disease (erythema infectiosum).

#Spread by the respiratory route, blood and transplacental .

#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 (1<sup>st</sup> 20 wks of pregnancy (12%).

#Minimal risk to the fetus if infection occurred during the third trimesters of pregnancy.

## Clinical features

#Known to cause fetal loss through hydrops fetalis; severe anaemia, congestive heart failure, generalized oedema and fetal death

#No evidence of teratogenicity

## Diagnosis

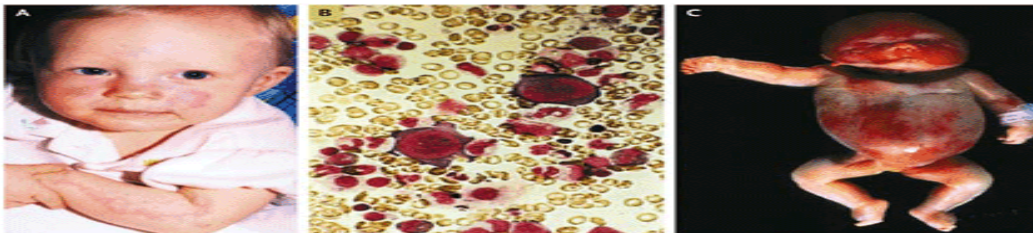
#ultrasound

#Serology IgM, persistent IgG

#PCR

## Treatment

#intrauterine transfusions and administration of digoxin to the fetus.



# Neonatal varicella zoster

#90% of pregnant women already immune (we have the vaccine)

#Primary infection during pregnancy carries a greater risk of severe disease

#Varicella( DNA herpes virus) , measles and mumps: when get the nature infection , we will get strong immunity but the problem is the re-activation of the virus.

## Information

### People at risk to get infection

#### 1. Immunocompromised

#### 2. Pregnant

#### 3. Elder people

### The diseases which are caused by Varicella zoster

- Chickenpox
- shingles ( الحزام الناري )
- congenital varicella syndrome for fetus (3%)
  - Scarring of skin
  - Hypoplasia of limbs
  - CNS and eye defects



## Diagnosis

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

## Treatment

Acyclovir	Live-attenuated vaccines	Zoster immunoglobulin
At first sign to varicella pneumonia	Pre-exposure: Before or after pregnancy but not during pregnancy	Postexposure to susceptible pregnant women and infant  Whose mother develop varicella during the last 5 days of pregnancy or the first 2 days after delivery and premature baby < 28 wks of gestation

# Rubella viruses

## INFORMATION

**R**

**REBELLA**

**RNA**

**RESPIRATORY**

❖ RNA enveloped virus, member of the togaviridae family

Spread by Respiratory droplets and transplacentally ❖

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

Sensorineural hearing loss “most common”

Neurologic “less common”

Cataract, glaucoma and “salt & pepper retinopathy”

Cardiac malformation “fetal ductal arteriosus”

## Other clinical features

- Growth retardation •
- Bone disease •
- HSM “hepatosplenomegaly” •
- Thrombocytopenia “blueberry muffin lesions” •

DIAGNOSIS	TREATMENT
<p><b>Maternal IgG is useless!</b></p> <p><b>Viral isolation virus from nasal secretions, throat, blood, urine, CSF.</b></p> <p><b>Serologic testing. IgM = recent postnatal or congenital infection.</b></p> <p><b>Rising monthly IgG titers suggest congenital infection.</b></p>	Supportive care only with parent education
	<b>Prevention by immunization</b>
	Maternal screening Vaccinate if not immune <u>(avoid pregnancy for three months)</u>

# 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 sequelae 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: Hearing loss, vision impairment, developmental delay

## Diagnosis and treatment:

- Maternal IgG shows only past infection
  - **Viral isolation from urine or saliva in 1<sup>st</sup> 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
- Treated by Ganciclovir x6wks in symptomatic infants

# 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

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

### Treated by:

High dose of acyclovir

	First trimester	Second trimester	Third trimester
Toxoplasmosis	Higher fetal death and lower rate of infection		High rate of infection with low fetal death
Virecella zoster	Primary infection have risk of severe disease		
Rubella	Infection in first 12wks have 70% risk of affected infant, Between 13-16wks 20% affect infant and rare while >16wks		
Cytomegalovirus	Primary infection has 40% risk of transmission to infant, increased transmission later but very severe if transmitted early		
HSV	First transmission through maternal genital tract, primary infection has high risk to transmission than reactivation		
Syphilis	Typically occur during the second half of pregnancy		
Parvovirus P19		Risk of fetal death highest 12%	Minimal risk to the fetus

## MCQs:

**1- which one occurs typically during the second half of pregnancy:**

- A- Toxoplasmosis
- B- cytomegalovirus
- C- Syphilis
- D- parvovirus

**2- which one is the most common congenital viral infection:**

- A- Cytomegalovirus
- B- Rubella virus
- C- P19
- D- Herpes virus

**3- a 70 years old man came to clinic with shingles, which infectious agent could he have:**

- A- cytomegalovirus
- B- parvovirus
- C- rubella virus
- D- varicella virus

## SAQs:

**1- mention the three form of toxoplasmosis:**

- A- bradyzoite
- B- tachyzoites
- C- oocytes

**2- what is the treatment of syphilis, varicella zoster and cytomegalovirus respectively:**

- A- penicillin, acyclovir and ganciclovir

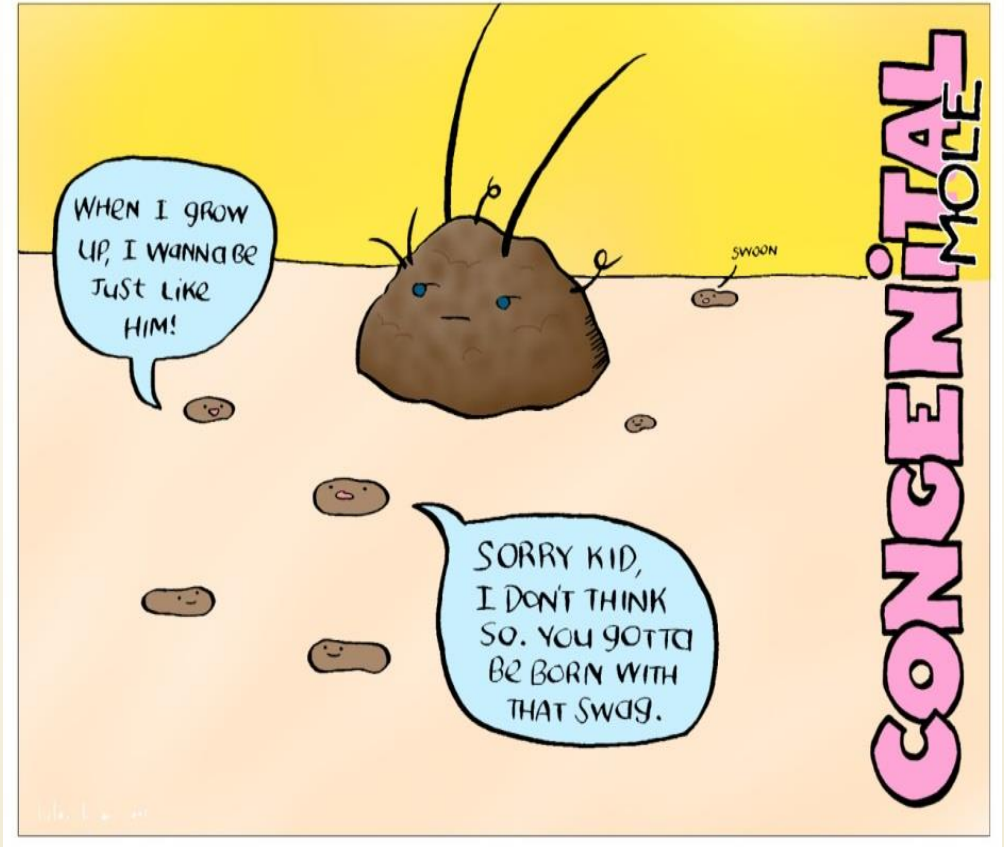
Ans: 1-C

2-A

3-D

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لي عند حاجتي إليه إنك على كل شيء قدير وحسبنا الله و نعم  
الوكيل)